Molecular Medicine

Podcast Transcript
Episode 27

Hello everyone and welcome back to "Mollie Medcast," the podcast for the biomedical journal, *Molecular Medicine*. My name is Margot Puerta. I'm the Associate Editor here at *Molecular Medicine* and your host for this podcast episode. In this week's podcast: "If You Give A Rat A Cookie", "PPARγ Ligands in Prostate Cancer", and "ALI Pathogenesis", a review paper dealing with acute lung injury. Before we move on to our summaries, I'd like to wish the Mollie Medcast a happy first birthday. July marks a year of podcasts for us here at Mollie Medcast and to celebrate we'll be giving away an ipod shuffle. To become eligible, visit www.molmed.org/podcast and put your pin up on our frapprmap. The drawing will be at the end of July. Let's review Molecular Medicine's mission. We strive 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 the journal, please visit our website for information, www.molmed.org. Alright, so let's get started with the papers in this week's podcast. The first paper in this "Mollie Medcast" episode is:

If You Give A Rat A Cookie

High dietary intake of the simple sugar fructose has rapidly become an important causative factor in the development of metabolic syndrome. There are several factors involved in metabolic sydrome and some of these include: having extra fat around your abdomen, high blood pressure, high cholesterol, and insulin resistance.[1] Metabolic syndrome is also associated with accelerated macrovascular and microvascular coronary disease, cardiomyopathy and elevated inflammatory status. IL-18 is an inflammatory cytokine that's associated with increased adiposity and insulin resistance. Dr. Shan-Shan Xing and colleagues from the Shandong University in China fed rats fructose to test whether metabolic syndrome-associated elevation of IL-18 could be pharmacologically attenuated. Their results show the calcium channel blocker felodipine could attenuate serum levels of IL-18, cardiac IL-18 mRNA, and coronary perivascular fibrosis suggesting that IL-18 may contribute to the pathological sequelae of this disease. The title of their paper is, "Felodipine Reduces Cardiac Expression of IL-18 and Perivascular Fibrosis in Fructose-fed Rats." In case any of you were wondering about the summary title, "What happens if you give a rat a cookie?" – he'll want a glass of milk.

PPARγ Ligands in Prostate Cancer

Prostate cancer is the most common malignancy in men over 60. Over 186,000 men per year are diagnosed with prostate cancer in the United States, and if it's found and treated at an early stage the recovery rate is high.[2] However, in its advanced stage prostate cancer is associated with significant cancer-related mortality. PPARγ, a member of the peroxisome proliferator-activated receptor family, is overexpressed in prostate cancer. The PPARγ ligands promote cell cycle arrest and apoptosis in prostate cancer cells. Dr. Papageorgiou and colleagues from the National and Kapodstrian University of Athens in Greece analyzed the ability of two PPARγ ligands to increase cytotoxicity and suppress survival in PC-3 prostate cancer cells. The title of their paper is, "Rosiglitazone Attenuates Insulin-Like Growth Factor 1 Receptor Survival Signaling In PC-3 Cells." While both ligands induced cytostasis, only the synthetic ligand inhibited the survival factor action of IGF-1 on chemotherapy-induced apoptosis of PC-3 cells. This occurred via a non-genomic action. This attenuation of IGF-1-dependent signaling in PC-3 cells could have clinical implications for the management of androgen ablation-refractory and chemotherapy-resistant advanced prostate cancer patients with bone metastasis.

And last up, our "Review and Assess" article for this week's podcast:

ALI Pathogenesis
Acute Lung Injury or ALI is associated with high morbidity and mortality. ALI is a co-morbid event associated with a diverse family of diseases and the lack of therapeutic options for ALI may result from distinct pathological processes. Activated neutrophil induced tissue injury and epithelial cell apoptosis-mediated lung damage represent two potentially important candidate pathomechanisms in ALI. In this review, Dr. Mario Perl and his colleagues focus on these pathogenic mechanisms as well as the role of small interfering RNA as a potential therapeutic for Acute Lung Injury. The title of the paper is, "Epithelial Cell Apoptosis and Neutrophil Recruitment in Acute Lung Injury - A Unifying Hypothesis? What we have learned from small interfering RNAs."

Thanks for joining us. That's it for this week's episode of "Mollie Medcast". Join us next time when we 'target LPA in sepsis' and 'alarm the host of danger.' 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.

If you happen to be taking a coffee break and have a moment, 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. Help us expand our community by adding your own pin to the map. If you're not shy you can even include your picture.

At the end of July we'll do a drawing, and the winner will receive a free iPod shuffle – courtesy of *Molecular Medicine* to celebrate Mollie Medcast's one year birthday.
This podcast is available on molmed.org 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!

1 http://www.americanheart.org/presenter.jhtml?identifier=4756 Accessed July 9, 2008.
2 http://www.prostatecancerfoundation.org/site/c.itIWK2OSG/b.46631/k.8E90/Prostate_Cancer_Information.html Accessed July 9, 2008.

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