Molecular Medicine

Podcast Transcript
Episode 76

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. This week we'll be going over some of our upcoming papers, which include: "Personalized Smoking Cessation", "Expression Of Novel Mutations Causing Niemann-Pick Disease", and "Severe Burn Injury".

Let's quickly review our goal here at *Molecular Medicine*. Since 1994 our mission has been 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. I'd also like to share some great news with everyone, the most recent impact factors released by Journal Citation Reports show that our impact factor has increased by 47% to 5.02.[1] This is the highest impact factor in *Molecular Medicine* history and we would like to thank all our authors and reviewers for helping us make this happen. Now on to the podcast.

The first paper in this podcast episode is:
Personalized Smoking Cessation
Cigarette smoking is a significant cause of disease and premature death. While successful quitting reduces these risks to smokers, success rates following attempts to quit smoking remain modest with long-term abstinence rates below 25%. More effective smoking cessation might result from personalizing existing treatments based on characteristics of individual smokers. Here, Dr. Jed Rose and colleagues from Duke University and NIH-IRP, NIDA, hypothesized that highly dependent smokers may require higher doses of nicotine replacement therapy before and after quit dates. The paper title is, "Personalized Smoking Cessation: Interactions between Nicotine Dose, Dependence and Quit-Success Genotype Score." In this paper, two nicotine replacement therapy doses were compared in precessation smokers, along with quit success, genotype and end-expired air carbon monoxide. Results provide support for a personalized and adaptive approach to smoking cessation treatment, which tailors the dose of nicotine replacement therapy to phenotypic and genotypic characteristics of the individual smoker.

Next up in this podcast:
Expression Of Novel Mutations Causing NPD
Types A and B Niemann-Pick disease (NPD) are autosomal recessive sphingolipidoses caused by mutations in the sphingomyelin phosphodiesterase 1 (or SMPD1) gene. We remember that autosomal recessive means two copies of an abnormal gene must be present for the disease to develop.[2] The type A Niemann-Pick disease results in infantile neurodegeneration and hepatosplenomegaly, with death occurring in the first few years of life. The type B disease also includes hepatosplenomegaly, but replaces the infantile neurodegeneration with pulmonary disease and patients usually survive into adolescence or adulthood. While mutations have been reported in types A and B phenotypes, expression studies are the most accurate way to predict which mutations have residual enzyme activity and may be neuroprotective. Here, Jonathan Desnick and his colleagues from the Mount

Sinai School of Medicine present clinical and molecular findings for six unrelated type A and type B patients. The title of the paper is, "Identification and Characterization of Eight Novel SMPD1 Mutations Causing Types A and B Niemann-Pick Disease." The results provide additional information for predicting clinical phenotypes in newly diagnosed infants and children with Niemann-Pick disease.

Last up for this podcast episode is:

Severe Burn Injury

Patients with severe burns are susceptible to sepsis, which is a systemic inflammatory condition that may lead to multiple organ failure and shock, which are common causes of morbidity and mortality. Full-thickness burns that exceed 25% of the total body surface area produce a profound systemic inflammatory reaction characterized by leukocyte activation and plasma leakage in the microvasculature of tissues and organs remote from the wound. Endogenous hydrogen sulfide [H2S] is naturally synthesized in many types of mammalian cells and plays a proinflammatory role in various experimental models. In this work, Dr. Jing Zhang and colleagues from Singapore and New Zealand investigate the effect of local burn injury on hydrogen sulfide release in distant organs. The paper title is, "Role of Hydrogen Sulfide in Severe Burn Injury-Induced Inflammation in the Mouse." Burn injury significantly increased plasma and liver hydrogen sulfide levels. Prophylactic, as well as therapeutic, administration of an inhibitor of hydrogen sulfide synthesis significantly reduced systemic inflammation. These findings highlight the role of hydrogen sulfide in contributing to inflammatory damage after burn injury and contribute to our understanding of this process.

And that's it for this week's episode of the *Mollie Medcast*. Join us next time when we take a look at: homocysteine in sepsis, MIF in asthma, and imaging stem cells in brain tumors. If you have a suggestion for a future podcast topic, please send me an e-mail at: margot@molmed.org, that's m-a-r-g-o-t-(at)-m-o-l-m-e-d-(dot)-o-r-g. You can also keep up with the journal by following us on Facebook and Twitter (@mol_med).

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Music: Opuzz.com
Photos: iStock or BigStock

Citations:

1. 2009 Journal Citation Reports® Science Edition (Thomson Reuters, 2010)
2. www.nlm.nih.gov/medlineplus/ency/article/002052.htm Accessed 9. July.2010