Molecular Medicine

Podcast Transcript
Episode 51

Hello *Mollie Medcast* listeners and welcome back to the podcast. *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 I'll be your host for this podcast episode. In this week's podcast we're going to talk about research articles related to smoking. We'll start with: "Kicking The Habit – It's In The Genes", "Smoking Out Genes Responsive To Oxidative Stress", and end with "Heavy Metal Consequences: Cadmium Affects Sperm Concentration and Motility".

Before we get to those let's just take a minute to review our goal here at *Molecular Medicine*. 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 have a manuscript you'd like to submit, please visit our Web site for information, www.molmed.org. Okay, so let's get started with this podcast.

There are an estimated 29 million men and women smoking in the United States.[1] So, w e all know smoking is bad, but here is a sobering statistic, cigarette smoking kills 1 in 10 adults worldwide. In our July-August 2009 issue, authors study different aspects of smoking, including cessation and the link to other complications.

So, we'll start of with the cover story from our July/August issue:

Kicking The Habit – It's In The Genes

Smokers and non-smokers alike can appreciate that some people have an easier time quitting than others. By examining twin studies, we've seen that there is a heritability component to this difference. Dr. Tomas Drgon and authors at the NIH, Duke University Medical Center, and Vector Tobacco Incorporated decided to look deeper at these apparent genetic differences. The authors have recently used genome–wide association studies to show that genetics can determine quitting success rates in individuals who used either nicotine replacement, bupropion, or community support quitting methods. Their current study is entitled "Genome-Wide Association for Smoking Cessation Success: Participants in a Trial with Adjunctive Denicotinized Cigarettes". So, this study expands on their prior work by demonstrating that many of the genetic patterns found in prior studies also underlie a patient's ability to quit when using de-nicontinized cigarettes. Not only do these findings support their previous observations, but they also suggest a new method of smoking cessation that mitigates potential side effects of nicotine or other similar compounds. These studies also identify specific genes associated with addiction and could help inform future efforts to curb or prevent substance dependence.

Next is:

Smoking Out Genes Responsive To Oxidative Stress

Tobacco smoke exposure leads to the significantly increased likelihood that cancer, cardiopulmonary diseases, and other morbidities may develop. The small airway epithelium is one of the first lines of defense against the oxidative stress associated with cigarette smoke. Several antioxidant mechanisms work to eliminate these toxins. One of these includes those in the nuclear factor erythroid 2-related factor transcription factor pathway. That's a long one. Let's call the nuclear factor erythroid 2-related factor- Nrf2. Dr. Ralf-Harto Hübner and colleagues at Weill Cornell Medical College and Cornell University examined the effects of cigarette smoke on the expression of Nrf2 and on Nrf2-linked genes in the airway epithelium of humans and mice. The title of their manuscript is "Coordinate Control of Expression of Nrf2-Modulated Genes in the Human Small Airway Epithelium Is Highly Responsive to Cigarette Smoking." The authors found that Nrf2 is upregulated in human small airway epithelium. The expression can be localized to the nucleus, highlighting its role as a transcription factor responsive to specific oxidative stress. This study not only provides further understanding of the pathways leading to cigarette smoke-induced damage in humans but also underscores similarities between human and murine responses to oxidative stress.

For those local New Yorkers looking to quit, the North Shore LIJ Health System has created the Center for Tobacco Control. It's open to the public and it's helped thousands of people quit smoking. In fact this center has one of the highest success rates in the whole United States and, it's free of charge. If you would like more information on the Center for Tobacco Control you can call the following number: 516-466-1980. Again that's 516-466-1980.

I'll also put the address and the Web site in the podcast transcript notes so you can check it out. [Center for Tobacco Control (of the North Shore-LIJ Health System), 225 Community Drive, South Entrance, Great Neck, NY 11021 website: http://www.northshorelij.com/body.cfm?id=558]

Now for the last paper for this podcast episode:

Heavy (Metal) Consequences: Cadmium Effects Sperm Concentration and Motility

While this paper isn't centered on smoking, active or passive cigarette smoke is one way humans are readily exposed to the heavy metal cadmium. Other sources may include food and drinking water. Since this metal tends to accumulate in reproductive tissues such as the testes, Dr. Susan Benoff and her colleagues hypothesized that cadmium exposures contribute to worldwide reports of declining sperm counts and decreased male fecundability. The title of this paper is "Cadmium Concentrations in Blood and Seminal Plasma: Correlations with Sperm Number and Motility in Three Male Populations (Infertility Patients, Artificial Insemination Donors, and Unselected Volunteers)". The authors demonstrate that there is increased cadmium in the seminal plasma of infertile patients as contrasted with the two healthy populations. Additionally, sperm motility and number are lower with increased cadmium concentration, and these endpoints did not correlate with confounders. Experiments in rats recapitulated the human observations. They showed that cadmium exposures alone were sufficient, and indicated that animal model systems can serve as a platform for the further study of cadmium toxicity. These findings illuminate a connection between environmental exposures and observed decreases in fertility.

That's it for this week's episode of Mollie Medcast. Join us next time for, when we "Tamp Down TNF-Induced Inflammation via Cholinergic Agonist" and get some "Molecular Therapy of Dysimmune Neuropathy".

If you have any questions or comments about this podcast, please feel free to send me an e-mail. My e-mail address is margot@molmed.org, that's m-a-r-g-o-t at m-o-l-m-e-d.org.

If you're taking a break from your experiments and have a moment, check out our podcast Web page molmed.org/podcast. You can play around with our frappr map, view other *Molecular Medicine* users from around the globe, and see our latest twitter updates. To follow us on twitter, search for the user name "MollieMedcast" – all one word.

This podcast is available on molmed.org and is up in iTunes, just put "Mollie Medcast" in the search bar. *Molecular Medicine* is published bimonthly by The Feinstein Institute for Medical Research.

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

Produced by Margot Puerta
Managing Editor, *Molecular Medicine*

Written by Robert L Pinsonneault
Associate Editor, *Molecular Medicine*

Edited by Veronica J Davis
Communications Editor, *Molecular Medicine*

Music: Opuzz.com
Photos: iStock, StockXpert

References:

1. American Heart Association. http://www.americanheart.org/presenter.jhtml?identifier=4559. Accessed June 24, 2009.