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Manhasset, NY, USA

Mollie Medcast

Episode 19 Transcript: Hematopoiesis, Osteoarthritis, and Catecholamines

Hello everyone and thanks for downloading us! Welcome back to “Mollie Medcast,” the podcast for the bio-medical journal, *Molecular Medicine*. My name is Margot Puerta. I’m the Associate Editor here at *Molecular Medicine* and your host for this podcast episode. My office is freezing cold right now and I have a sore throat so forgive me if I’m a little bit on the raspy side. I hope everyone enjoyed their holidays this past weekend, Easter and Purim and that you’re ready for some more podcast summaries.

In this week’s podcast: “Neuronal NO Synthase In Hematopoiesis,” “A Joint Cause,” and “Foolishly Unlocking Pandora’s Box?”

Before we get on with that, let me remind you about what our goal here at Molecular Medicine is. Our mission is to publish novel work 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 and click on the author center link.

Alright, so let’s get started with the papers for this podcast. The first paper in this “Mollie Medcast” episode is:

Neuronal NO Synthase In Hematopoiesis

Nitric oxide, is abbreviated NO, is an important signaling molecule and it can act in an autocrine and paracrine mode. NO is a crucial regulator of vasodilation, immunity and neurotransmission. It’s also involved in regulating the balance between proliferation and differentiation in several developmental and differentiation settings. In the hematopoietic system, NO contributes to the regulation of hematopoietic stem and progenitor cells in the bone marrow. While the action of NO in the hematopoietic system has been demonstrated, the contribution of individual NO synthase (NOS) isoforms and their mode of action are not understood. In this paper, which appears in our March-April issue, Dr. Peter Krasnov and his colleagues investigated these mechanisms. The title of their paper is, “Neuronal Nitric Oxide Synthase Contributes to the Regulation of Hematopoiesis.” Their results suggest that nNOS-produced NO acts as a paracrine regulator of hematopoietic stem cells and that nNOS-selective inhibitors may have therapeutic potential for hematopoiesis-related disorders.

The second paper in this podcast episode has the summary title:

A Joint Cause

Osteoarthritis is a very common joint disease and it severely restricts patient mobility. The cartilage destruction that takes place is not only associated with an imbalance of anabolic and catabolic processes, but also with poorly understood alterations in the cytoskeletal organization of chondrocytes. Dr. Helga Joos and her colleagues from Germany investigated the effects of IL-1 β on components of the chondrocyte cytoskeleton on different expression levels. The title of their paper is, “IL-1 β Regulates FHL2 and other Cytoskeleton Related Genes in Human Chondrocytes.” Results show that IL-1 β is involved in the regulation of various cytoskeletal components in human chondrocytes and may be relevant in the pathogenesis of osteoarthritis. A deeper knowledge of these molecular processes may set the pathogenetic mechanisms of degenerative joint diseases in a novel context.

And lastly we have a review paper:

Foolishly Unlocking Pandora's Box?

Catecholamines derive from the adrenal medulla and presynaptic neurons and regulate immune and inflammatory responses. Recent studies reveal that T cells also can synthesize and release catecholamines, which then can regulate T cell function. Macrophages and neutrophils, when stimulated, can generate and release catecholamines de novo which, in an autocrine/paracrine manner, regulate mediator release using adrenergic receptors. Here, Dr. Michael Flierl and his colleagues review the roles of catecholamines and their receptors in immunity and inflammation. The title of their review article is, "Catecholamines – Crafty Weapons In The Inflammatory Arsenal Of Immune/Inflammatory Cells Or Opening Pandora's Box?" I love that title.

That's it for this week's episode of "Mollie Medcast." You can find these papers and many more 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.

I am super excited this week because on Thursday, my Managing Editor and I are heading down to Florida for the Molecular Medicine: Applying Current and Emerging Technologies Symposium. It's going to be at the Buena Vista Palace in the Walt Disney World Resort and the talks are going to cover an overview of the current and emerging technologies that are changing the patterns of medical practice today and moving us towards personalized medicine. After the symposium is over, we're going to work with its director, Shala Masood, Dr. Shala Masood and we're are going to produce a special focused issue featuring short reviews written by some of the meeting attendees and speakers. So be sure to check that out and to look for that in the coming months.

If you're taking a coffee break and have a second, 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. If you have a moment, help us expand our community by adding your pin to the map. And if you're not shy you can even include your picture.

This podcast is available on molmed.org and 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 Gallowitsch-Puerta

Associate Editor, *Molecular Medicine*

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