Molecular Medicine

Podcast Transcript
Episode 81

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, Managing Editor here at *Molecular Medicine* and I'll be your host for this podcast episode. In this week's podcast we'll take our first look at papers from our November-December 2010 issue: "Hyperhomocysteinaemia In Sepsis", "Telomere Dysfunction In Plasma Cell Disorders," and "IL-18 In Metabolic Syndrome".

We'll start by taking a minute to 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. Ok, let's move onto the podcast.

Our first paper in this podcast episode is:
Hyperhomocysteinaemia In Sepsis
Hyperhomocysteinaemia, or increased levels of homocysteine in the blood, is an established risk factor for coronary artery disease, which occurs when dietary supply with folate and/or vitamin B12 is inadequate. Increased homocysteine in the blood may lead to the formation of blood clots, resulting in heart attack or stroke. Homocysteine also acts as a proinflammatory mediator and may therefore play a role when the immune system is stimulated, such as in trauma or sepsis. Since no major studies have investigated homocysteine concentrations in septic patients, Dr. Martin Ploder and colleagues in Austria investigated whether hyperhomocysteinemia may develop in this cohort despite administration of enteral nutrition. The title of their paper is, "Early Increase of Plasma Homocysteine in Sepsis Patients with Poor Outcome." Results indicate increased levels of homocysteine are associated with poor patient outcomes. This may be due to the increased demand of B-vitamins associated with immunopathogenetic mechanisms. Although preliminary, this work suggests B-vitamin supplementation during inflammatory conditions should be further explored.

Next up, we have:
Telomere Dysfunction In Plasma Cell Disorders
Two of the most common plasma cell disorders are monoclonal gammopathy of undetermined significance (or MGUS for short) and multiple myeloma. Clinical manifestations of multiple myeloma include osteolytic lesions, anemia, hypercalcemia, immunodeficiency, and renal abnormalities. Telomere length and telomerase activity in cancer development have been extensively studied. Telomeres progressively shorten, lead to dysfunction and contribute to tumorigenesis. While several studies have evaluated telomere length and telomerase activity in multiple myeloma patients, there is little information regarding this data in presymptomatic MGUS patients. Dr. Julieta Panero and colleagues in Argentina examined mRNA expression in genes that encode telomere-binding proteins, in order to investigate the role of telomere dysfunction during the progression of monoclonal gammopathy of undetermined significance to multiple myeloma. The title of the paper is, "Altered mRNA Expression of Telomere-Associated Genes in Monoclonal Gammopathy of Undetermined Significance and Multiple Myeloma." Results show evidence of shortened telomeres, changes in telomere maintenance gene

expression, and evidence of modified expression in telomere-associated genes between MGUS and multiple myeloma. Understanding this process may aid in designing therapeutic approaches for patients with plasma cell disorders since many of them may be sensitive to interventions targeting telomere-damage.

Our last paper in this episode is:

IL-18 In Metabolic Syndrome

Metabolic syndrome is a disorder characterized by a group of metabolic risk factors including obesity, type 2 diabetes, and hypertension, leading to an increased risk of cardiovascular disease. Inflammation plays an important role in the pathophysiology of metabolic syndrome. While it has been suggested that elevated serum levels of the proinflammatory cytokine interleukin-18 (otherwise known as IL-18) are involved in the pathogenesis of metabolic syndrome, the relationship remains unclear. In this work, Dr. Shan-Shan Xing and colleagues in China investigated the functional consequences of IL-18 overexpression in metabolic syndrome using an experimental model. The title of the paper is, "Overexpression of IL-18 Aggravates Cardiac Fibrosis and Diastolic Dysfunction in Fructose-Fed Rats." Findings indicate overexpression of IL-18 leads to aggravated left ventricular diastolic dysfunction, suggesting an important role of IL-18 in myocardiac fibrosis in metabolic syndrome. Attenuation of the inflammatory process may represent an avenue for therapeutic strategy in treating metabolic cardiomyopathy.

And that's it for this week's episode of *Mollie Medcast*. Join us next time when we "Muscle Out Myositis with Resistance Exercise", take a look at a genome-wide association on smoking cessation and review the transition from acute to chronic arthritis. For questions or comments regarding this podcast, please feel free to send me an e-mail at: margot@molmed.org. You can also keep up with the journal by following us on Facebook at www.facebook.com/molmed and Twitter (@mol[underscore]med).

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