Molecular Medicine

Podcast Transcript
Mol Med 13:5-6 Ep 02

Hello and thanks for downloading us! It is my pleasure to welcome you back to the "Mollie Medcast", the podcast for the biomedical journal, ***Molecular Medicine.***

This is Margot Gallowitsch-Puerta, Associate Editor, coming to you from the north shore of Long Island, New York.

It's a gorgeous day here in Manhasset, summer has definitely arrived, but that wont keep me from this weeks topics which include "High Levels Of CTGF In Idiopathic Portal Hypertension Patients", "Nano Drug Carriers Deliver Treatment" and "Arthritis – It's In Your Genes."

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.

Our journal was introduced in 1994 to serve as a forum through which scientists and researchers could communicate recent discoveries to a multi-disciplinary, international audience interested in understanding and curing disease.

Molecular Medicine is published bimonthly by the Feinstein Institute for Medical Research which is located in Manhasset, New York and this week's podcast will cover some of the manuscripts that we have in our May-June 2007 issue. So, off we go.

Our first manuscript for this week deals with:
Idiopathic Iortal Hypertension.
Idiopathic portal hypertension, or IPH, is a rare disorder with an unknown etiology (and if you remember that was our vocab word from the last episode). If you are familiar with this disease you may also have heard it referred to as Banti's syndrome. Patients with this disease have high blood pressure in the portal vein and surrounding branches. This manuscript is called, "Expression of Connective Tissue Growth Factor in the Human Liver with Idiopathic Portal Hypertension." The lead author is Dr. Morikawa from the Osaka City University Graduate School of Medicine.

Idiopathic portal hypertension is clinically associated with portal hypertension in the absence of cirrhosis. The pathogenesis of IPH remains poorly understood and Dr. Morikawa and collegues designed this study to look at the characteristics of RNA expression in liver specimens from patients with this disease. They looked at liver specimens from IPH patients and patients without liver diseases and found that several up-regulated genes were detected, including one called connective tissue growth factor or CTGF. CTGF plays an important role in matricellular regulation related to internal and external cell signaling. This up-regulated gene showed an intense positive reaction with in situ hybridization studies. Further analysis of human serum revealed that levels of CTGF in patients with IPH were significantly higher than healthy volunteers. These results enhance our current knowledge of idiopathic portal hypertension and indicate that overexpression of CTGF may be an important target in this disease.

Next up:
Nano Drug Carriers Deliver Treatment
I'm going to apologize ahead of time if I trip over the phrase liposomised etoposide. It's a tongue twister.

Anticancer drugs are generally plagued by toxic manifestations at doses necessary to control various forms of cancer. Incorporating drugs into liposomes not only reduces toxicity but also enhances their therapeutic index. Etoposide is a derivative of a toxin found in the American Mayapple and has been successfully employed as an antineoplastic agent against various forms of cancer. Earlier attempts have demonstrated usage of liposomised etoposide in delaying tumor progression, but such treatments failed to show tumor regression in animal models. Tuftsin is a tetra-peptide reported to have anti-tumor activity in experimental tumor animal models. This work is entitled, "Tuftsin Augments Antitumor Efficacy of Liposomised Etoposide Against Fibrosarcoma in Swiss Albino Mice." Dr. Arif and colleagues hypothesized that the combination of tuftsin with a potent antitumor agent could successfully suppress various forms of cancer. This work evaluated the antitumor potential of liposomal etoposide with and without tuftsin against fibrosarcoma in Swiss albino mice. Tuftsin bearing liposomes significantly reduced tumor volume, delayed tumor growth and increased upregulation of p53wt expression. The results of the present study suggest that tuftsin incorporation in drug-loaded liposomes may be a promising strategy to treat various forms of cancers including fibrosarcoma.

Arthritis – It's In Your Genes
Rheumatoid Arthritis is a chronic autoimmune disease for which there is no cure. It has a strong genetic component with a heritability of 60%. The identification of genes contributing to disease susceptibility and severity is expected to generate novel and better therapeutic targets. T cells play a central role in the pathogenesis of autoimmune arthritis, and several abnormalities in T cell homeostasis have been described in Rheumatoid Arthritis. This manuscript is entitled, "Genetic Regulation of T regulatory, CD4, and CD8 Cell Numbers by the Arthritis Severity Loci Cia5a, Cia5d and the MHC in the Rat." In it, Dr. Brenner and his colleagues hypothesized that T cell phenotypes, including frequencies of different subsets of T regulator cells, and their in vitro functional responses could be genetically determined. Brenner and his colleagues postulated further that the genetic contribution would be accounted for by one of the arthritis regulator quantitative trait loci leading to novel clues to gene mode of action in Rheumatoid Arthritis. T cells from arthritis susceptible, resistant, and quantitative trait loci congenic rats were isolated from the thymus, peripheral blood and spleen and then analyzed. Results show for the first time that arthritis quantitative trait loci regulate subsets of T regulatory cells. Furthermore, major histocompatibility complex genes are involved in this process, raising a novel potential explanation for the long-known MHC association with Rheumatoid Arthritis and other forms of autoimmune arthritis.

That's it for this week. You can find these papers and many more on our website: ***www.molmed.org*** that's w..w..w…dot m o l m e d… dot… o r g.

Also, I'd like to remind you about our "pre-submission enquiry service".
If you're thinking about submitting a paper to ***Molecular Medicine*** but you're not sure if it falls with in the scope of our journal send me an email with a summary of your novel work and its significance and I'll get back to you. My email address is: m-a-r-g-o-t@molmed.org

That's it for this week. From Long Island, New York this is margot@molmed.org thanks for listening!