Molecular Medicine

Podcast Transcript
Episode 79

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 a look at two primary research papers and one review paper from our upcoming September-October 2010 issue. The primary research papers are: "Genetic Risk Factors For Coronary Heart Disease" and "Polymorphisms And *p16* Hypermethylation In Colorectal Cancer." The review paper looks at the protein ABCA1 and its possible link between inflammation and reverse cholesterol transport.

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, so now onto the podcast.

Genetic Risk Factors For CHD

While the role of common Apolipoprotein E (*APOE*) variants on Coronary Heart Disease [CHD)] is well known, the influence of other nearby apolipoprotein genes is unclear. Dr. Gie Ken-Dror and colleagues examined the association between gene cluster variations of apolipoproteins in the genotypes of over 2700 middle-aged men, and included 275 coronary heart disease events, over a 15-year follow-up period. The title of the paper is, "APOE/C1/C4/C2 Gene Cluster Genotypes, Haplotypes and Lipid levels in Prospective Coronary Heart Disease Risk among UK Healthy Men." Results indicate carriers of the *APOE* ε2 and APOC2 single nucleotide polymorphisms (or SNPs) had a significantly lower risk of coronary heart disease when compared with non-carriers, while carriers of the APOC1 SNP had higher risk of coronary heart disease. The common *APOE* polymorphism may explain the majority of the locus genetic determinants of plasma lipid levels; however, additional SNPs in the *APOC1/C2* region may contribute to coronary heart disease risk. This work exposes the potential of non-*APOE* SNPs as potential risk factors for coronary heart disease.

Next up:

Polymorphisms And *p16* Hypermethylation In Colorectal Cancer

Hypermethylation of *p16* is associated with reduced survival in patients with colorectal cancer. In this work, Dr. Yvonne Wettergren and her colleagues from Sweden's University of Gothenburg, investigated whether polymorphisms in folate- and methyl-associated genes were linked with *p16* hypermethylation in colorectal cancer patients. The title of the paper is, "MTHFR, MTR, and MTRR Polymorphisms in Relation to *p16INK4A* Hypermethylation in Mucosa of Patients with Colorectal Cancer." Results indicate patients with MTRR gene variants exhibit significantly worse cancer-specific survival when the mucosa is positive for *p16* hypermethylation. The authors hypothesize this may be due to oxidative stress associated with this genotype as well as with *p16*. These data begin to unravel a mechanism which may be responsible for increased risk of recurrent disease as well as poor cancer-specific survival.

Next up is our review paper:
ABCA1: A Possible Link Between Inflammation and Reverse Cholesterol Transport
Atherosclerosis, a chronic inflammatory disease of the artery wall, involves numerous cellular and molecular inflammatory components. ATP-binding membrane cassette transporter A1, abbreviated ABCA1, is a crucial protein involved in cellular cholesterol efflux and reverse cholesterol transport. Recent studies suggest this protein may be the molecular basis for the interaction between inflammation and reverse cholesterol transport. In this paper, Dr. Kai Yin and colleagues from the University of South China, review recent findings on the role of inflammatory cytokines, inflammatory proteins, inflammatory lipids and endotoxin-mediated inflammatory processes in expression of ABCA1. Additionally, the authors cover this protein's function in modulating immunity and inflammation through direct and indirect antiinflammatory mechanisms.

And that's it for this week's episode of the *Mollie Medcast*. Join us next time when we take a look at: regulation of HMGB1 release, diabetic angiopathy, and sepsis patients with poor outcomes. For questions or comments regarding this podcast, 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.o-r-g. You can also keep up with the journal by following us on Facebook and Twitter (@mol_med).

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