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Hello *Mollie Medcast* listeners and welcome back! *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'll take a look at three research papers. The first two come from our May/June 2010 issue: "Antitumorigenic Activity Of hGBP-1," and "Digesting Celiac Disease," The third comes from our July-August 2010 issue: "Towards Better Stroke Diagnosis and Prognosis."

Let's take a moment to review our goal here at *Molecular Medicine*. Established in 1994, 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're interested in submitting a manuscript to the journal, please visit our Web site for information, www.molmed.org. Alright, so now we'll be starting up with papers in this podcast.

The first paper in this podcast episode is:

Antitumorigenic Activity Of hGBP-1

Cancer can develop when the immune system doesn't properly identify and target abnormal cells for destruction. Recently, interferon gamma and its effectors, such as guanylate binding proteins (or GBPs), have been implicated in cancer immunosurveillance. Dr. Karoline Lipnik and her colleagues in Austria, Germany, and Italy, therefore investigated whether one well-known human guanylate binding protein, hGBP-1, could contribute to interferon gamma-mediated tumor defense. The title of the paper is, "The Interferon Gamma-Induced Human Guanylate Binding Protein-1 Inhibits Mammary Tumor Growth in Mice." The authors found that animals induced to express greater quantities of hGBP-1 exhibited reduced tumor growth, lower levels of tumor tissue hemoglobin contents, and attenuated tumor cell proliferation. These results also correlated to reduced amounts of VEGF-A both in vitro and in vivo. The authors conclude that these observations implicate hGBP-1 in interferon gamma-mediated antitumorigenic activities through inhibition of tumor cell angiogenesis. An increased understanding of how the immune system recognizes and disposes of neoplastic cells could yield more innovative treatments for a variety of malignant cancers.

Okay, number two for this podcast episode:

Digesting Celiac Disease

Celiac disease (or CD) is an immune-mediated disorder, and it's triggered by ingestion of wheat gliadin and other related proteins in those individuals who are genetically predisposed to it. The result – damage to the small intestine and improper absorption of food.¹ Currently, a permanent gluten-free diet is the only accepted therapy for celiac disease. This disease can be difficult to diagnose because of a lack of concordance with serological and histological findings. In this work, Dr. Maria Paola Simula and her colleagues in Italy, sought to develop a diagnostic celiac disease signature and to gain a better understanding of the pathogenic mechanisms associated with this disease. They analyzed intestinal mucosa proteome alterations of celiac disease patients with varying degrees of histological abnormalities. Results indicate that downregulation of proteins involved in peroxisome proliferator-activated receptor signaling and the modulation of several cancer-related proteins, are associated with the highest celiac disease histological scores. The data suggest peroxisome proliferator-activated receptor

may be a therapeutic target for modulation of inflammation in celiac disease and warrants additional research.

And, the last paper in this podcast episode is:

Towards Better Stroke Diagnosis and Prognosis

Stroke accounts for about 1 out of every 17 deaths in the United States. Many of the surviving patients must then contend with severe disabilities. Acetylcholinesterase [AChE] activity and the systemic inflammatory response are both implicated in stroke, and carriers of polymorphisms that alter cholinergic activity could be more susceptible to inflammatory damage. Since there is currently no reliable diagnostic biomarker for mild stroke, Dr. Einor Assayag and colleagues examined acetylcholinesterase activity and cholinergic status (so, the total capacity for acetylcholine hydrolysis) in suspected stroke patients. The authors found that acetylcholinesterase activities were lower and butyrylcholinesterase activities higher in stroke patients when compared with controls. These values correlated with multiple inflammatory biomarkers, including fibrinogen, interleukin-6, and C-reactive protein. These findings suggest that circulation cholinesterase measurements could be useful as early diagnostic tools for the occurrence of stroke, which may in turn open new venues for earlier stroke diagnosis and treatment.

And that's it for this week's episode of the *Mollie Medcast*. Join us next time when we take a look at manuscripts involving trauma-hemorrhage, ischemia/reperfusion injury, and ST segment elevation in chest pains. For questions or comments regarding this podcast, or to request a certain topic for a podcast, please send me an e-mail at: margot@molmed.org. You can also keep up with the journal by becoming our fan on Facebook or following our tweets on Twitter (@mol_med).

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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*

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