Molecular Medicine

Podcast Transcript
Episode 68

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 your host for this podcast episode. In this week's podcast we'll continue with the papers from our March-April 2010 issue, including: "Homing In On Cellular Mechanisms Of Proliferation In CLL", "Spotting A Protein Linked With Aggressive Liver Cancer" and a review, "Ghrelin, A Potential Therapy For RCI".

We'll start by taking a minute to remind you about what our goal here is at *Molecular Medicine*. 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 let's get started with this podcast. Now, we're going to start with the first paper which is:

Homing In On The Cellular Mechanisms Of Proliferation In CLL
Chronic lymphocytic leukemia (or CLL for short) is the most common type of leukemia, and usually affects the adult population. CLL affects the blood and bone marrow and gets the name chronic because the disease advances more slowly than other leukemias.1 According to the National Cancer Institute2, it was estimated that more than 15,000 people in North America were diagnosed with CLL in 2009. Like all cancers, an understanding of the specific mechanisms of cell proliferation is crucial for the development of effective CLL treatments. Human CD38, which is a glycoprotein involved in the catabolism of extracellular nucleotides, is a negative prognostic marker for CLL patients. In this work, Dr. Fabio Malavasi and his colleagues in Italy characterize the long-term interactions between CD38-positive CLL cells and CD31-positive cells, confirming the role of this protein complex in proliferation, cell migration and homing. The title of their paper is, "CD38/CD31 Interactions Activate Genetic Pathways Leading to Proliferation and Migration in Chronic Lymphocytic Leukemia Cells." Their results suggest that the CD31/CD38 axis is part of a network of accessory signals that modify the cellular microenvironment, favoring localization of leukemic cells to growth-permissive sites. Improved understanding of cell–cell interactions could lead to new diagnoses and treatments for CLL.

The North Shore-LIJ Health System currently has 13 CLL patient studies going on right now. If you would like more information on this, please contact Ruth Morgan at 516-562-4874 or visit the North Shore-LIJ Web site at: http://www.northshorelij.com

Next up:

Spotting A Protein Linked With Aggressive Liver Cancer
Hepatocellular carcinoma (or HCC) is the fifth most common malignancy worldwide and ranks third among causes of cancer-related deaths. The five-year survival rate is less than 5%. HCC is a major clinical challenge – first because the disease is so severe, but also because there are a lack of good diagnostic markers and treatment strategies. To garner a better understanding of HCC, Dr. Daniela Spano and colleagues, also from Italy, performed a systematic functional genomic analysis on human HCC cell lines: HuH-7 and JHH-6. They also followed this up

with data from HCC patients. The title of their paper is, "Galectin-1 and Its Involvement in Hepatocellular Carcinoma Aggressiveness." The group identified a genetic signature consisting of 11 genes. Of these genes, LGALS1 was selected and characterized in the context of HCC patient samples. (A little background here, LGALS1 codes for Galectin-1, a protein involved in various aspects of tumorigenesis.) The authors show that overexpression of Galectin-1 leads to increased carcinoma cell migration and invasion in vitro. They also show a positive association between increased expression of Galectin-1 and the presence of metastasis, which is an indicator of cancer aggression. This study clears the way for future investigations to identify markers for the prediction, diagnosis and prognosis of HCC.

And last but not least for this episode, we have a review paper:
Ghrelin, A Potential Therapy For RCI
There is growing concern worldwide about the threat of nuclear terrorism and subsequently, exposure to radiation. Whole- or partial-body exposure to a high dose of radiation results in acute radiation syndrome. Little is known about therapeutic approaches for acute radiation syndrome, which is often accompanied by trauma, burn, infection and sepsis – and when these are present we have radiation combined injury (or RCI). Dr. Asha Jacob and colleagues from the Feinstein Institute for Medical Research in New York review radiation combined therapy in their paper. It's titled, "Ghrelin as a Novel Therapy for Radiation Combined Injury." Because of its proven antiinflammatory effects, the authors suggest ghrelin as a possible therapy for radiation combined injury.

And that's it for this week's episode of *Mollie Medcast*. Join us next time when we talk about chronic nonhealing wounds, cystic fibrosis and thrombosis.

If you've enjoyed this podcast please consider posting a reviewer rating for us up in iTunes, the more the merrier! If you have questions or comments regarding this podcast, or topics and suggestions you would like to see covered, 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.org. You can also keep up with the journal by following us on Twitter (@mol_med).

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From New York, this is margot@molmed.org, thanks for listening!

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