::: CSHL in the News

Newsday
Put Long Island Brain Power to Work - Editorial
March 8, 2013

GenomeWeb
Trove of Treasures
March 5, 2013

Scientific American
What's Wrong with the Brain Activity Map Proposal
March 5, 2012

::: "In Quotes"

Assistant Professor Michael Schatz quoted in **Wired**
Selling Your Most Personal Item: You
March 27, 2013

Assistant Professor Gholson Lyon quoted in **Nature**
Patients should learn about secondary genetic risk factors, say recommendations
March 21, 2013

Assistant Professor Gholson Lyon quoted by **New York Genome Center**
Sequester puts "entire generation of scientists at risk," Collins says
March 14, 2013

Professor Thomas Gingeras quoted by **The Scientist**
Sequencing the Underdogs
March 8, 2013

President Bruce Stillman quoted in **Science**
Ready for More 10,000 Cancer Genomes Projects?
March 6, 2013

::: Milestones

James Watson honored as one of American Association for Cancer Research first class of Fellows

Bo Li promoted to Associate Professor

CSHL wins top performance awards for our electronic newsletter

::: Upcoming Events

Walking Tours
Saturday April 6 & Sunday April 21

DNA Day Scavenger Hunt on Main Street
Saturday April 20

Watson School of Biological Sciences Commencement Convocation
Sunday April 28

2013 Cultural Series
Concerts and Lectures Posted

2013 DNALC Summer Camps
Registration Now Open

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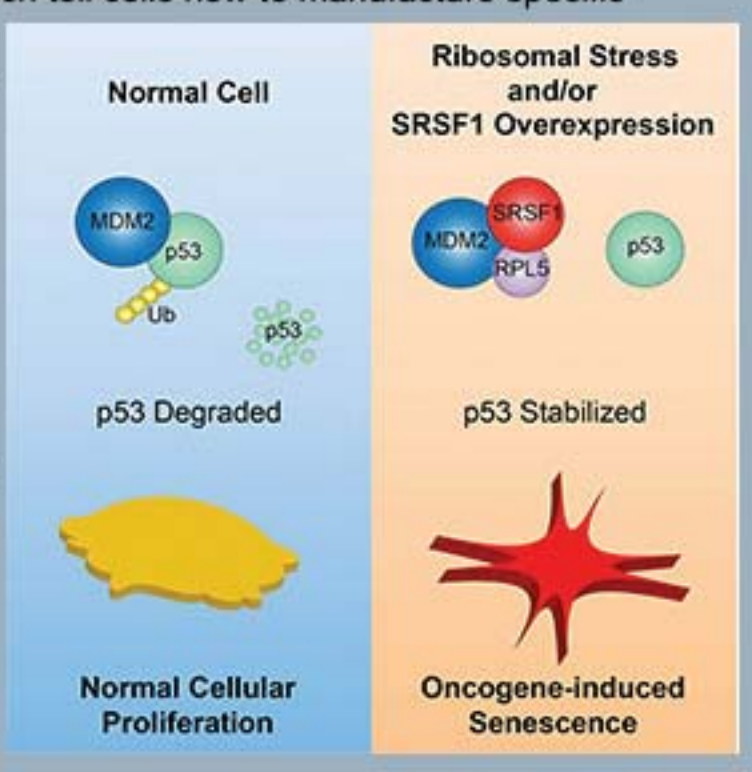
A protein that keeps check its own cancer-causing pontential

When they first discovered the protein SRSF1, Professor **Adrian Krainer's** lab group understood that it was important in splicing - a "cutting and pasting" process that edits and assembles the RNA messenger molecules which tell cells how to manufacture specific proteins. The same protein was later found to be cancer-promoting, or oncogenic, under certain conditions. In a **paper** published in *Molecular Cell*, Krainer's team now shows that SRSF1 can also paradoxically **halt cell growth**. It does this through interacting with a protein called RPL5 found in ribosomes, the cell's protein "factories." SRSF1 and RPL5 interact as part of a complex that stabilizes the powerful tumor-suppressor protein p53. This interaction results in the activation of p53 and halts cell growth. "In the mechanism we've identified, SRSF1 keeps a check on its own aberrant activity," explains graduate student and lead author of the new paper, Shipra Das. "The study enhances our understanding of how tumors arise and the pathways to cancerous transformation."



Open House draws hundreds to CSHL

On Saturday, March 23, Cold Spring Harbor Laboratory welcomed over 400 members of the community to campus for its first Open House.

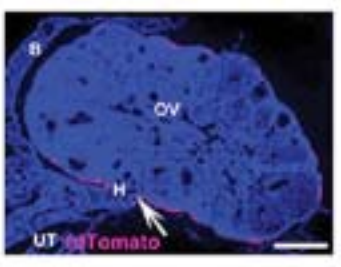
Visitors included many who had never been to CSHL before, who plainly enjoyed learning about the spectrum of CSHL research and education programs, as well as ways in which they can be involved -- as students, friends and neighbors. During a continuously running series of *5-Minute Science* talks, CSHL postdoctoral researchers engaged audiences throughout the afternoon on topics from "Why haven't we cured cancer?" to "Molecular Photography." Free mini-tours of campus led by Watson School Ph.D. students and lab postdocs proved to be extremely popular. Kids and adults alike crammed a booth where DNA Learning Center teachers led hands-on biology demonstrations, featuring DNA extraction.

Visit CSHL's Facebook page to see a **photo album** from this wonderful day, and please take a moment to give us some feedback in this **survey** about what you would like to see next time.

How does a stem cell become a leaf cell?

The development process in living things is like a grand symphony: the whole is the product of many events involving the contributions of many "players," which must occur in almost perfectly coordinated temporal and spatial sequence. In simple animals like the fruit fly and more recently in plants and animals, scientists have been able to identify the principal players. Now for the first time in plants, a team led by Professor **Marja Timmermans** has explained how the action of key players "is very precisely coordinated in time and space." In their **paper**, published in *Genes & Development*, they explain how a complex of gene-repressing proteins knows exactly where to attach in the genome in order to enable a stem cell to "commit" to developing as, for instance, a leaf cell. Read more about it **here.**

Cells in ovarian niche prone to become cancerous



Professor **Grigori Enikolopov** and colleagues have made an important discovery about a possible source of ovarian cancers, a subject that has long perplexed scientists. In research **published** in *Nature*, they show that when two important tumor-suppressor genes are inactivated, a previously unknown type of stem-like epithelial cell in the ovaries undergoes cancerous transformation. The idea that cells with stem-like properties can spontaneously mutate to become seeds for cancer has been gaining momentum. Enikolopov and colleagues report that a part of the mouse ovary called the hilium is a niche for stem cells, supplying new cells to repair the surface of the ovary after rupture due to ovulation. Cells in this region **are prone** to spontaneous cancerous transformation, they suggest.

Watson op-ed

Dr. James Watson has published a new **opinion piece** in London-based *The New Scientist* further detailing his unconventional thinking about the relation between antioxidants, oxidants, and cancer. In it he considers the possibility that antioxidants may not only have a role in promoting cancer under some circumstances, but type 2 diabetes as well.