Beginning Tuesday, January 21, 2014, LSUHSC is excited to announce that we will assume operations for all dining services including the Tiger Den Café in the MEB, the Dental School Café, the Atrium Coffee Kiosk, and Campus Catering. You will start to notice many positive transformations as we ramp up services with quality, customer service, healthier options, variety and value at the forefront.

The new operating hours for Monday through Friday are:
Tiger Den Café: 7:00am – 2:00pm
Dental Café: 7:30am – 1:30pm
Atrium Coffee Kiosk: 7:00am – 4:00pm

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LSUHSC research reveals structure of master regulator & new drug target for autism, cervical cancer

A team of LSUHSC scientists has discovered the structure of the active form of E6-associated protein (E6AP), an enzyme that acts as a master regulator in the body. They report, for the first time, that the active form of E6AP is composed of three distinct protein molecules. E6AP controls functions as diverse as the ability of nerve cells to “rewire” themselves in response to external stimuli and the mechanism by which certain viruses, like human papillomaviruses or HPV, hijack normal cellular processes in order to replicate – a process that can ultimately lead to cancer. The research is published today in the Journal of Biological Chemistry.

The research team included lead investigator Dr. Virginia Ronchi, a postdoctoral fellow, and her colleagues, Jennifer Klein and Dr. Daniel Edwards, all of whom work in the laboratory of Dr. Arthur Haas, the Roland Coulson Professor and Chairman of Biochemistry and Molecular Biology.

LSUHSC in the thick of the Battle in Seattle

As evidenced by the number of LSUHSC faculty, staff, and students sporting their black and gold today, along with the rest of the Who Dat Nation, excitement about the Saints is rising to a fever pitch. Housekeeping’s pre-game meal today was Popeyes and green Gatorade. One member of the LSUHSC family will have an up close view of the action in tomorrow’s divisional playoff game. Dr. John Amoss (in the gray jacket above), Chief of our Section of Hospital Medicine, will take his usual place on the sidelines as the Saints’ Team Internist. Here’s hoping he will stay and that the Saints’ victory will be injury free.
Because the assembly of cells is like an elaborate tinker toy set in which the parts can be used in different combinations to serve various roles, E6AP normally functions in nerve cells to direct brain development and in a functionally related process termed neuronal plasticity, which allows nerve cells to alter their patterns of communication with neighboring cells during learning.

Inherited loss of E6AP function in the brain results in the mild to severe neurodevelopmental defects of Angelman Syndrome, which occur in 1 in 10,000-20,000 births. Angelman Syndrome is a developmental condition characterized by severe mental retardation in children because the brain is unable to “learn” by adapting its nerve connections to outside stimuli. In contrast, other types of mutations that lead to increases in brain E6AP activity are thought to cause certain forms of inherited Autism Spectrum Disorder (ASD), suggesting that a carefully orchestrated balance of E6AP function is necessary for normal brain development.

Computer analysis identified a region of the molecule critical for forming the three-part structure, allowing the investigators to create a drug to block the assembly and activity of the enzyme. The computer analysis also demonstrated that several mutations associated with Angelman Syndrome result from defects in assembly of the three protein molecules.

In other studies reported in the paper, Dr. Ronchi and her colleagues show that the replication strategy of two forms of HPV associated with cervical cancer hijacks normal cells, directing them to make a viral protein called E6 that binds to E6AP at the point of assembly and that this feature of the viral protein’s function could be used to reverse the molecular assembly defects of the Angelman Syndrome mutations. While not a cure for Angelman Syndrome, this work emphasizes the feasibility of future drug design to promote E6AP assembly as a potential therapy for some forms of the disease. It also provides a target for vaccine and drug development to prevent or treat cervical cancers caused by HPV by derailing cell transformation at the step of E6-E6AP binding.

The findings of this National Institutes of Health supported research represent a major advance in our understanding of the mechanism of E6AP function and potential strategies for drug design to combat cervical cancer and familial ASD. Since E6AP is but one member of a larger superfamily of 29 related human enzymes, the current findings with E6AP have important implications for the other regulatory pathways within cells.