Introducing our AUTHORS

Khandaker A.Z. Siddiquee

Current position: University of Central Florida, College of Medicine, Burnett School of Biomedical Sciences, Biomolecular Science Center, Postdoctoral Research Fellow with Prof. James Turkson
Education: Dhaka University, Bangladesh, B.S. and M.S in microbiology, 1999 and 2002; Kyushu Institute of Technology, Japan, Ph.D. in metabolic engineering, 2005
Nonscientific interests: Fishing, music, spending time with my wife

The initiation of this scientific study began from the simple observation about the disruption of activated Stat3 dimer by a small molecule. I am interested in drug–protein interactions from the biochemical, structural, and molecular biology viewpoints. In this study, we present a novel small-molecule Stat3 dimerization inhibitor, S3I-M2001, and describe the dynamics of the processing of activated Stat3 in malignant cells within the context of the biochemical and biological effects of the Stat3 chemical probe inhibitor. S3I-M2001 induces an early perinuclear aggresome formation of Stat3, which was hitherto unknown, a late-phase proteasome-mediated Stat3 degradation, and antitumor cell effects. (Read Siddiquee's article on p 787.)

Olga A. Timofeeva

Current position: Georgetown University Medical Center, Department of Oncology, Washington, DC, Research Fellow with Prof. A. Dritschilo
Education: Novosibirsk State University, Russia, M.S. in molecular biology, 1995; Novosibirsk Institute of Bioorganic Chemistry, Russia, Ph.D. in biochemistry with Dr. M. Filipenko, 2000
Postdoctoral work: National Cancer Institute, Laboratory of Comparative Carcinogenesis, Frederick, MD, with Dr. A. Perantoni, 2001–2006
Nonscientific interests: Swimming, hiking, music, art history

My long-term goal is to develop new cancer therapies based on disruption of signal transduction pathways abnormally activated in breast and prostate cancers, with the special emphasis on STAT transcription factors. Such studies offer opportunities for discovering novel basic science in the quest to contribute to improving cancer treatment. I became interested in molecular mechanisms underlying activation of STAT proteins in tumorigenesis and their role in different types of cancers during my postgraduate training. I was fascinated by the diversity of signaling pathways regulated by STATs through interactions with other proteins. I believe that disruption of STAT3 protein–protein interactions in cancer cells is the key to targeting these molecules for cancer therapy. (Read Timofeeva's article on p 799.)

Delphine Pouchain

Current position: University of Edinburgh, Department of Chemistry, Ph.D. candidate with Prof. Mark Bradley
Education: University of Montpellier II, France, M.S. in biomolecular chemistry, 2004
Nonscientific interests: Traveling, swimming, watching movies

Because protein kinases represent one of the largest families of enzymes and play crucial roles in many cellular processes, it is particularly interesting to study their activities. Our article describes the use of a peptide nucleic acid encoded library to determine the substrate specificity of three different protein tyrosine kinases. The entire library was arrayed onto a DNA array, and an anti-phosphotyrosine antibody and a fluorescently labeled secondary antibody were used to detect the extent of phosphorylation. This method allowed for the identification of known and new target proteins for each kinase. (Read Pouchain's article on p 810.)

Sandro F. Ataide

Current position: University of California, Berkeley, Howard Hughes Medical Institute, Department of Cell and Molecular Biology, Postdoctoral Fellow with Prof. Jennifer A. Doudna
Education: University of Campinas, Brazil, B.S. in chemistry, 1999; University of São Paulo, Brazil, M.S. in biochemistry with Prof. Shaker Chuck Farah, 2001; The Ohio State University, Ph.D. in microbiology with Prof. Michael Ibba, 2006
Nonscientific interests: Triathlon, seafood, movies, art

During my Ph.D. studies, I wanted to understand how the aminoacyl-tRNA synthetases evolved efficient substrate selection to exploit it as a potential route to develop useful inhibitors of microbial protein synthesis. The structural and functional diversity among the aminoacyl-tRNA synthetases prevents infiltration of the genetic code by noncognate amino acids. In our article we investigate the mechanism of the bacterial growth inhibition by *S*-(2-aminoethyl)-L-cysteine (AEC). Using an *in vivo* model, we demonstrate that active site variants of lysyl-tRNA synthetase restore growth in the presence of AEC, an indication that the primary target of this compound is translation and that the L box riboswitch is a secondary cellular target. (Read Ataide's article on p 819.)

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