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ogy, 1993; M.S University of Baroda, India, M.S. in biochemistry, 1996; University of Mumbai, Ph.D. in applied biology with Dr. Nishigandha Naik, 2002 Postdoctoral work: University of New Mexico with Prof. Eric Prossnitz, 2002-2006; Georgetown University Medical Center with Prof. Robert Dickson, 2006 Nonscientific interests: Traveling, photography, cooking

Current position: UniversitätsSpital Zürich, Insti-

tute of Neuropathology, Department of Pathology,

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During my graduate studies, I became interested in the trafficking of G-proteincoupled receptors (GPCRs). My postdoctoral work gave me additional opportunities to further explore interactions of GPCRs and various signaling molecules. While we were investigating the functions of GPR30, its intracellular localization was intriguing to me. The novel approaches used in our paper will be helpful in exploring functions of newly identified intracellular GPCRs as well as in elucidating the functional significance of intracellularly expressed GPCRs. (Read Revankar's article on p 536.)

My research focuses on developing new tools for studying protein misfolding diseases, such as Alzheimer's disease. These tools, luminescent conjugated polymers, have a flexible thiophene backbone with alternative single- and double-carbon bonds that gives these molecules interesting optical properties. When the polymers bind to aggregated proteins, the relative orientation of the thiophene rings within the polymer backbone and between polymer chains is altered, and this affects the fluorescence being emitted. Hence, optical fingerprints for specific protein conformations can be obtained. These molecules can prove helpful for the understanding of the pathogenic information encoded in multiple conformations and morphologies of the protein aggregates. (Read Nilsson's article on p 553 and Point of View on p 525.)

I am interested in learning the molecular mechanisms underlying drug action. In my current research, I'm trying to understand the mechanisms by which antibiotics affect the ribosome. Knowing the structural and mechanistic details of how antibiotics target ribosomes can aid in improving the existing antibiotics and designing new drugs in order to overcome emerging bacterial drug resistance. Antibiotics interfere with certain steps of translation and thus can also be used as a tool to study the ribosome function. In the current paper, we discuss the mechanism by which the clinically used antibiotic spectinomycin inhibits translocation of messenger RNA and transfer RNAs on the bacterial ribosome. (Read Borovinskaya's article on p 545.)

My graduate research involves biochemical approaches to determine how the ribosome works. The ribosome is an RNA-based machine that translates messenger RNA (mRNA) into proteins by using aminoacyl-transfer RNA (tRNA) as substrates. Our understanding of the mechanisms of translation has improved thanks to recent crystallographic advances. A primary challenge that remains is to uncover the dynamic aspects of translation, such as translocation, the movement of tRNA-mRNA in the ribosome. We describe the structural and biochemical effects of spectinomycin on ribosomal translocation, providing evidence that the movement of the "head" domain of the small ribosomal subunit is important for the movement of tRNA-mRNA. These findings will help transform our current static pictures of this megadalton machine into dynamic movies. (Read Shoji's article on p 545.)

Modulation of specific protein-protein or protein-DNA interactions with small molecules, designed or identified through a screen, offers organic chemists an opportunity to perturb protein interaction networks and gene expression programs in a logical manner. Dervan-type polyamides represent a moleculardesign approach to interfering with protein–DNA interactions that can be used to effect programs of gene expression. This report shows how a polyamide designed to bind a six-base-pair DNA sequence disrupts a subset of the target genes activated by the hypoxia-inducible factor 1 (HIF-1) and offers a measure of specificity of this polyamide in this system compared with other methods of HIF-1 antagonism. (Read Nickols' article on p 561.)