



Sudipta Maiti

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Sudipta pursued his undergraduate studies at IIT Kanpur, and then obtained a Ph.D. in Biophysics in 1994 from the University of Pennsylvania. He subsequently joined Cornell University to work in the laboratory of Prof. Watt Webb, who was at that time developing some revolutionary new microscopy techniques. Sudipta joined the Tata Institute of Fundamental Research (TIFR) in 1998, where he is currently a Professor of Chemical Sciences. He uses spectroscopy and microscopy as the primary tools of his research. His current research interest spans several different fields, namely, protein aggregation (which is related to diseases such as Alzheimer's and Parkinson's), chemical neurotransmission (which is related to the molecular basis of drug addiction), and development of new optical tools for studying biology. When he is not busy in his lab, he loves to promote scientific exchange between fellow scientists and students. He does this through several initiatives: The Fluorescence Society (he is the Chairman and co-Founder), the National Photonics Fellowship program, the Indian Biophysical Society (of which he is the Secretary) and a science club of Western India called "Biophysics Pashchim".

ABSTRACT

Optical Techniques for Understanding Intrinsically Disordered Protein

Aggregation of intrinsically disordered proteins cause a variety of incurable human diseases. Understanding these unconventional proteins present both a challenge and an opportunity for optical techniques. We show that membrane encased plasmonic nanoparticles can be a powerful tool for probing the mechanisms of toxicity. They can act as Raman enhancers as well as fluorescence quenchers to provide complementary information through Raman and Fluorescence Lifetime spectroscopy. They show that the secondary structure of membrane attacking amyloid oligomers can be very different from the less toxic fibrils. This, combined with results obtained from single molecule photobleaching studies, have begun to yield a consistent picture of a putative mechanism of toxicity in Alzheimer's disease.