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The centers of premeltons signal the beginning and ends of genes

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Premeltons are examples of emergent structures (i.e., structural solitons) that arise spontaneously in DNA due to the presence of nonlinear excitations in its structure. They are of two kinds: B-B (or A-A) premeltons form at specific DNA-regions to nucleate site-specific DNA melting. These are stationary and, being globally nontopological, undergo breather motions that allow drugs and dyes to intercalate into DNA. B-A (or A-B) premeltons, on the other hand, are mobile and being globally topological, act as phase-boundaries transforming B- into A- DNA during the structural phase-transition. They are not expected to undergo breather-motions. A key feature of both types of premeltons is the presence of an intermediate structural-form in their central regions (proposed as being a transition-state intermediate in DNA-melting and in the B- to A- transition), which differs from either A- or B- DNA. Called beta-DNA, this is both metastable and hyperflexible-and contains an alternating sugar-puckering pattern along the polymer-backbone combined with the partial-unstacking (in its lower energy-forms) of every other base-pair. Beta-DNA is connected to either B- or to A- DNA on either side by boundaries possessing a gradation of nonlinear structural-change, these being called the kink and the antikink regions. The presence of premeltons in DNA leads to a unifying theory to understand much of DNA physical-chemistry and molecular-biology. In

particular, premeltons are predicted to define the 5' and 3' ends of genes in naked-DNA and DNA in active-chromatin, this having important implications for understanding physical aspects of the initiation, elongation and termination of RNA-synthesis during transcription. For these and other reasons, the model will be of broader interest to the general audience working in these areas. The model explains a wide variety of data, and carries within it a number of experimental predictions – all readily testable – as will be described in my talk.

Speaker Biography

Henry M Sobell was born in Los Angeles, California November 7, 1935, and grew up in Brooklyn, New York, where he attended Brooklyn Technical High School (1948-1952), Columbia College (1952-1956) and the University of Virginia School of Medicine (1956-1960). Instead of practicing clinical medicine, He went to the Massachusetts Institute of Technology, Cambridge, Massachusetts, to join Professor Alexander Rich in the Department of Biology (1960-1965) where, as a Helen Hay Whitney Postdoctoral Fellow, he learned the technique of single-crystal X-ray analysis. He joined the Chemistry Department at the University of Rochester, College of Arts and Sciences and was then jointly appointed to the Department of Biophysics at the University of Rochester School of Medicine and Dentistry, becoming a full professor in both departments (1965-1993). He is internationally renowned for his pioneering contributions to the understanding of how the anticancer agent, actinomycin D, binds to DNA and exerts its mechanism of action. Using the technique of X-ray crystallography, he and his research colleague, Shri C. Jain, solved the structure of a crystalline complex containing actinomycin and deoxyguanosine, and the information obtained from their study led them to propose a model to understand the general features of how actinomycin binds to DNA.

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