Human papillomaviruses (HPV) represent a family of 150+ double-stranded DNA viruses, some of which constitute major risk factors for development of a variety of cancer.

Various HPV types have been classified as low-risk or high-risk for cancer based on their frequency as a driver of cervical cancer as determined from epidemiological data. Despite this, many HPV types are difficult to classify correctly due to lack of sufficient patient data. The crucial mechanistic differences between these two types of HPVs remain unknown, except that they differ in the frequency and extent of DNA replication in addition to disease prognosis. The HPV-encoded E2 replication initiator protein and its four 12 bp DNA binding sites in the origin of HPV genome play a pivotal role in viral DNA replication, as well as in determining the possibility of oncogenesis of the HPV-infected cells. Mutations in the E2 binding sites lead to attenuation in DNA replication. These mutations also correlate with the oncogenicity of HPV types. All known low-risk HPV types have consensus E2 binding sites, whereas, all high-risk HPV types appear to have one or more point mutation(s) in the E2 binding sites. These sequence data provide new metrics for risk-assessment of all HPV types and further analysis of global distribution of various HPV types, particularly in under-developed regions.

BIOGRAPHY

Dr. Biswas is applying recent advances in molecular biology and genetics to analyze and understand human diseases with goals to develop novel therapeutics. Current efforts are focused in two areas: (1) delineating the molecular mechanisms of Human Papillomavirus family (HPV) induced oncogenesis that are known to lead to head & neck cancers and cervical cancer; and (2) mechanism of DNA replication in pathogenic bacteria primarily E.coli and Bacillus anthracis. His laboratory is also collaborating with Dr. Esther Biswas-Fiss to investigate pathogenesis of inherited diseases involving ABC transporters, ABCA4 and ABCA7 proteins.