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**DNA secondary structures as regulators of the lymphoma genome**

Diffuse large B cell lymphoma (DLBCL) is a genomically heterogeneous, aggressive blood cancer that is only curable in ~60% of patients. Treatment refractory tumors exhibit a high mutation rate often associated with misappropriated DNA editing normally intended for diversifying our antibody repertoire. These tumors also frequently overexpress pro-proliferative and survival genes. Why certain cancer-enabling genes in B cells are susceptible to mutation, amplification, or translocation is still unclear. We seek to understand the role of two key DNA secondary structures, the G-quadruplex (G4) and the i-motif (iM), in facilitating DLBCL genomic instability by investigating their interaction with the DNA editing enzyme, activation induced cytidine deaminase (AID), and their regulation of oncogene expression. This presentation will describe recent findings that link G4s and iMs with AID localization and activity, as well as how these structures modulate expression of important B cell receptor signaling molecules.