Acute myeloid leukemia (AML) is a genetically complex group of cancers wherein patients can be divided into those with chromosomal translocations and patients that are cytogenetically normal (CN-AML). In general, the outcomes for AML patients are poor and treatment options are restricted mainly to chemotherapy, thus approaches identifying novel therapeutic candidates are greatly needed. My research aims to better understand the mechanisms exploited by AML cells that are critical for supporting the initiation, growth, and survival of the tumor. Many factors commonly deregulated in AML have the functional capacity to control vast gene networks, factors such as epigenetic regulators and non-coding RNA. To this end, I will present findings from two different projects. The first is the generation of a novel rapid spontaneous murine model of AML, which closely parallels human CN-AML. Second, is an unbiased functional screening discovery of a global miRNA-target regulatory network, which has shed light on a new therapeutic opportunity in AML.

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