Cancer Genomics: Chapter 17. Acute Myeloid Leukemia

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On first consideration, acute myeloid leukemia (AML) represents a nearly insurmountable challenge in terms of understanding it at the molecular level in large part because of its immense heterogeneity as well as its variability across different age groups. In addition, while significant progress has been made in the overall survival of subsets of patients with AML, many continue to show little progress in terms of positive treatment outcomes. Cytogenetic and initial molecular studies have resulted in the ability to stratify patients into specific risk categories that predict favorable-, intermediate- and poor-risk outcomes. However, these categories are limited in their ability to predict accurately how individual patients will respond to therapy and have not resulted in the ability to treat effectively patients with specific treatments. They have, however, resulted in excluding hematopoietic stem cell transplantation for patients with favorable-risk disease. Genome-wide analysis promises to improve both treatment and outcomes. The initial studies using whole-exon or whole-genome sequencing identified mutations in several novel genes that surprisingly were involved in regulating DNA methylation and chromatin structure. Subsequently, mutations were found in genes encoding transcription factors, signaling pathway modulators and genes involved in RNA splicing. Further analyses have identified mutations in key elements of miRNAs. Genome-wide methylation studies have highlighted key patterns that track with specific cytogenetic and gene mutations. Such epigenetic studies have led to the use of treatments directed to altering chromatin structure and DNA methylation. These treatments remain targeted specifically at specific enzymatic components of chromatin structure and function, but their key molecular consequences remain unclear and clinical responses unpredictable. RNA sequencing has led to the identification of both novel pathways of leukemia cell survival and unexpected fusion transcripts, which may ultimately be therapeutically targeted.

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