Acute myeloid leukaemia genomics

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Author(s) Medinger, Michael; Passweg, Jakob R.
Author(s) at UniBasel Medinger, Michael ;
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Acute myeloid leukaemia (AML) is a biologically complex, molecularly and clinically heterogeneous disease. Despite major advances in understanding the genetic landscape of AML and its impact on the pathophysiology and biology of the disease, standard treatment options have not significantly changed during the past three decades. AML is characterized by multiple somatically acquired mutations that affect genes of different functional categories. Mutations in genes encoding epigenetic modifiers, such as DNMT3A, ASXL1, TET2, IDH1, and IDH2, are commonly acquired early and are present in the founding clone. By contrast, mutations involving NPM1 or signalling molecules (e.g., FLT3, RAS gene family) are typically secondary events that occur later during leukaemogenesis. This review aims to provide an overview of advances in new prognostic markers, including targetable mutations that will probably guide the development and use of novel molecularly targeted therapies.

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