

Method for Predicting the Risk and Onset Time of Acute Myeloid Leukemia

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Background & Unmet Need

- Acute myeloid leukemia (AML) pathogenesis is characterized by the acquisition and accumulation of somatic mutations in certain AML-associated genes
- However, it is not known when these mutations arise, how they evolve, and the relative disease risk conferred by each one
- **Unmet Need:** Method to identify and stratify mutations correlated to increased AML risk to enable long-term patient monitoring and early therapeutic intervention

Technology Overview

- **The Technology:** A panel of mutations correlated to increased risk of developing AML for screening and early therapeutic intervention
- Using comprehensive longitudinal genomic studies in AML patients, the inventors identified a panel of high-risk premalignant somatic mutations in certain AML-associated genes
- The detection of these mutations accurately predicts the risk and onset time of AML even years before the emergence of symptoms
- The assay only requires a peripheral blood sample from the individual
- May be used for preventative screening as well as regular monitoring of AML progression in diagnosed patients

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Patents:

[PCT Application Filed](#)

Publications:

[Desai et al. Nat Med. 2018.](#)

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Technology Applications

- Assessing the risk of AML in symptom-free individuals for preventative screening purposes
- Predicting AML onset time in symptom-free individuals
- Monitoring AML progression in patients
- Providing guidance to the interventional options for AML patients and highly susceptible individuals

Technology Advantages

- Robust and reliable
- Predicts the risk and onset time of AML years before symptom emergence
- Convenient: only requires a peripheral blood/serum/plasma sample

Supporting Data / Figures

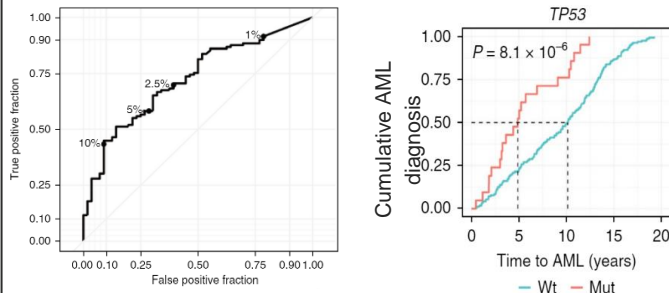


Figure 1: (Left) performance of the method is indicated by the receiver operating characteristic (ROC) curves showing the true-positive rates vs. the false-positive rates when predicting AML cases. (Right) Premalignant somatic mutations in certain AML-associated genes greatly influence the AML onset time. Patients with premalignant somatic mutations in TP53 developed AML significantly faster than those with wild-type TP53.

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