fus? Third, the effects of STX11- or MUNC18B-deficiency on lysosome secretion are much more modest than those on α or dense granule release, implying distinct controls for secretion from lysosomes relative to dense and α granules. Either of 2 models might explain this phenomenon: either the effects on lysosomal secretion are indirect (eg, a consequence of reduced signaling because of the lack of ADP release from dense granules) and a distinct syntaxin isoform, such as the less abundant STX7, participates in lysosomal secretion, or lysosomal secretion is mediated by a set of redundant syntaxins. Finally, if release of dense and α granules (and to at least some extent, lysosomes) is under similar molecular control, then how does one account for the different kinetics of their release after platelet stimulation? Surely the answers to these questions will be unlocked in the years ahead.

Conflict-of-interest disclosure: The author declares no competing financial interests.

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and non–HCT settings. A comparison with other prognostic schemes for predicting outcome would be of significant interest.\(^1\) It is worth noting that in a preliminary communication for the Eastern Cooperative Oncology Group, Strickland and coworkers found that aberrations of chromosomes 5 and 7 aberrations failed to add prognostic significance to monosomal cytogenetics.\(^6\) Do these differences in prognostic significance vary as a result of the treatment modality, or are they a consequence of age, as the patient population Strickland et al reported included older adults while the Germans enrolled mostly younger patients, under 60 years of age? Further, the German group does not provide the description of the breakpoints in the 5q deletion; a distinct tumor suppressor gene is proposed to be located at the 5q13.3 locus.

Given these data, how should a clinician now approach such AML patients? The German authors question the role of allogeneic HCT and the great financial and potentially high morbidity and mortality costs and recommend alternative strategies in these high-risk patients. But are they correct? Certainly their observation provides an additional stratification of the breakpoints in the 5q deletion; a distinct tumor suppressor gene is proposed.

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AML cytogenetics: the complex just got simpler

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