Errata

The Editor of the *2015 ASCO Annual Meeting Proceedings* has authorized correction of the following errors:

511, 590, 604, 607, 616, TPS627, TPS629, 2057, TPS1112, e17029
Notice of Correction: Conflict of interest information for José Baselga, MD, PhD, has been updated to reflect previously undisclosed relationships. The corrected disclosures of potential conflicts of interest provided by the author are available with the online abstracts on JCO.org and ASCO.org.

Abstract 10016

The abstract body published in the *ASCO Annual Meeting Proceedings* reflects an earlier version of the abstract. The abstract body now published online has been updated. ASCO apologizes for the error.

Abstract 7014

The abstract body published in the *ASCO Annual Meeting Proceedings* reflects an earlier version of the abstract. After re-analysis, the FLT3 ITD allelic burden hazard ratio predicting relapse was no longer statistically significant. The abstract body now published online has been updated.

Background: FMS-like tyrosine kinase internal tandem duplication (FLT3-ITD) mutation in normal karyotype AML (NK-AML) is a poor prognostic feature. The prognostic significance of the mutant to wild type allelic ratio and base pair insertion length (bpInsLng) of FLT3-ITD with or without Nucleophosmin (NPM1) mutation remains controversial in this population. **Methods**: The medical records of 145 patients with NK-AML were reviewed (2007-2014) including subject and disease characteristics, and treatment outcomes. Patients with any cytogenetic abnormality were excluded. Treatment failure was defined as relapsed or refractory disease after chemotherapy regardless of transplant. Death of any cause was considered an event. A FLT3-ITD mutation was detected as the presence of a migrating PCR product larger than the wild-type product (330-bp). ITD allelic burden (AB) was calculated as the ratio of ITD to wild type allele expressed as a percentage. Time to failure data were analyzed in a univariate Cox proportional hazards model. Patients who were alive with no evidence of disease at the last follow-up were treated as censored observations. The protocol was approved by the IRB. **Results:** (per re-analysis of the data) The mean age of patients was 58 years (range 22-88). FLT3 mutation status was available in 136 cases; 42 (30.9%) carried FLT3-ITD with assessment of AB and bpInsLng available in 27. The median ITD AB was 35% (range 3-433). For the time to failure analysis patients were classified as low or high allelic burden using the population median value as the cut point. The median length of the mutant fragment was 371bp (range 345-500). NPM1 mutation was detected in 38 (26%) patients. In this data set AB higher than median value (35%) did not statistically significantly predict treatment outcomes (hazard ratio = 1.39, 95% CI: 0.50-3.85, p = 0.52). ITD bpInsLng lower or higher than the population median value also did not predict treatment failure rate (p = 0.6). **Conclusions**: In patients with FLT3-ITD NK-AML, ITD allelic burden or bpInsLng did not predict outcomes in terms of complete remission or relapsed disease.

All errors have been corrected in the online versions of the abstracts on JCO.org and ASCO.org. This notice is updated on a rolling basis, as needed.