Armanda Pugnaloni[3]
Fabiola Olivieri[3,4]
Attilio Olivieri[1,5]
Antonella Poloni[1,5]

[1]Hematology, Department of Clinical and Molecular Sciences, DISCLIMO, Università Politecnica delle Marche, Ancona, Italy, [2]Department of Biomolecular Sciences, University of Urbino Carlo Bo, Urbino, Italy, [3]Department of Clinical and Molecular Sciences, DISCLIMO, Università Politecnica delle Marche, Ancona, Italy, [4]Center of Clinical Pathology and Innovative Therapy, IRCCS INRCA, Ancona, Italy and [5]Hematology, AOU Ospedali Riuniti, Ancona, Italy.
E-mail: a.poloni@univpm.it

*CS and MBE contributed equally to this work

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Supporting Information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Table S1. Clinical and biological features assessed in patients.

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Blast cells in acute megakaryoblastic leukaemia with Down syndrome are characterized by low CLEC12A expression

Acute myeloid leukaemia (AML) is a genetically and clinically heterogeneous disease, characterized by expansion of undifferentiated myeloid precursor cells.[1] Despite an increased understanding of the biology and therapeutic advances, the outcomes of AML patients remain unsatisfactory. Leukaemic cells express different antigens depending on their origin and their degree of maturation.[2] Immunophenotypic assessment using flow cytometry (FCM) is essential in both diagnosis and disease monitoring.[3] Detection of minimal residual disease (MRD) determined by FCM during treatment is associated with AML prognosis[4] and the identification and characterization of cell-type-specific antigens is also essential for immunotherapy.[2] However, the similarity in immunophenotype of normal and leukaemic cells hinders the identification and characterization of leukaemic cells, and therefore, reliable antigen markers are required.

A recent paper identified significant findings related to the effectiveness of the C-type lectin domain family 12 member A (CLEC12A) (also named hMICL and CLL-1) antibody–drug conjugate (ADC) in AML.[5] According to that study, expression of CLEC12A is consistent across different types of AML, and CLEC12A-ADC can be an effective treatment. However, most AML samples examined in the study were classified as French–American–British (FAB) subtype M1, M2, M4 or M5, and information on other subtypes is lacking. Although several groups have reported that the majority