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Hematology in China

In the last decade Chinese hematologists have made fundamental contributions to experimental and clinical hematology. The best example undoubtably concerns acute promyelocytic leukemia (APL). In 1988 Huang *et al.*[1] showed that all-trans retinoic acid (ATRA) is an effective inducer for attaining complete remission in APL. Subsequent elegant molecular studies contributed to clarifying the molecular pathogenesis of APL and the mechanisms by which ATRA induces remission of this condition.[2,3] An animal model was established in 1996.[4] More recently the efficacy of arsenic in relapsed patients has been shown.[5-7]

Haematologica is highly interested in interacting with hematologists in developing countries. Stimulating information on this subject can be found in a recent note by Dr. Harry S. Jacob.[8] We decided to set up a formal collaboration with the *Chinese Journal of Hematology*. The Editor-in-Chief, Jia-Zeng Li, has agreed to publish selected papers from Haematologica with an extended summary in Chinese in the *Chinese Journal of Hematology*. In exchange, Haematologica will have a section called *Hematology in China* in which selected papers from the *Chinese Journal of Hematology* will be summarized. Professor Zhong Chao Han will be the guest editor of this new section together with other colleagues. At the same time, Professor Zhen-Yi Wang has joined our Publication Policy Committee, while Zhu Chen has joined our Editorial Board. We hope that the presently small number of Haematologica's papers originating from China will increase sharply.[9,10]

We believe that this newly-generated interaction will grow and be fruitful. Our expectation is that it will benefit both Chinese and European hematologists. Time will tell us if we are right.

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Hematology in China
Acute myeloid leukemia M2b

In the late 1950s Yang and Yan *et al.*[1-3] identified a unique form of acute myelogenous leukemia (AML) on the basis of morphologic and clinical features. The predominant leukemia cells in the bone marrow were characterized by asynchronous development between nucleus and cytoplasm. The cells were large and somewhat irregular with a relatively lower N/C ratio as compared to usual AML cells. The cytoplasm was abundant and basophilic and filled with numerous fine pinkish granules and there was often a homogenous salmon coloration over the invagination of the nucleus. The nucleus was usually eccentric with spongy chromatin and prominent nucleoli. Clinically, the main manifestations were anemia related symptoms and signs, with minimum infection or bleeding complications, but a tendency to extramedullary granuloma formation. With simply supportive therapy and blood transfusion the disease usually ran a protracted course. Yang and Yan considered these leukemic cells to be abnormal early myelocytes and thus named this form of leukemia as subacute leukemia (SAL). SAL became increas-

ingly accepted in China, and more information about the disease was obtained further indicating it as a separate entity. Cytochemically, the leukemia cells were strongly positive for SB, POX and CE staining, but negative for NaF inhibition test of NSE.[4] Electron microscopy showed the cytoplasm of the leukemia cells to be usually packed with abundant granules over the invagination of the nucleus.[5] Cytogenetic study found that the t(8;21) translocation was the exclusive chromosomal aberration of the leukemic cells in about 90% of the patients[6,7] and it was verified that all the SAL patients, whether or not they had the t(8;21) translocation, were positive for AML1 gene rearrangement, ETO gene rearrangement, and/or AML1-ETO fusion transcript.[8-12]

After the publication of the FAB proposal for the classification of acute myeloid leukemias,[13] we considered that SAL was within the M2 category at a more mature stage. We thus divided the AML-M2 subtype into two subcategories: M2a and M2b. When there are more than 30% blasts in the bone marrow and more than 20% of the blasts show the morphology of SAL cells, we define this leukemia as AML-M2b, otherwise as M2a.[14] The subdivision of AML-M2 was adopted by the National Symposium on the Classification of Acute Leukemias in China in 1986.[15]

AML-M2b is basically the same thing as AML t(8;21).[16-18] The point is that this form of leukemia can be definitely diagnosed simply on a morphologic basis without cytogenetic examinations. Therefore, AML-M2b is naturally a distinct subtype in the morphologic classification of acute myeloid leukemia.

Like t(9;22) translocation and bcr-abl fusion gene for chronic granulocytic leukemia (CGL), AML-M2b is characterized by t(8;21) translocation and the AML1-ETO fusion gene, and consequently there are t(8;21)+AML1-ETO-M2b and t(8;21)-AML-ETO+ AML-M2b.[11] According to the data from the nation wide epidemiologic survey of leukemias conducted during 1986-1988 in China, the annual incidence of AML-M2b was 0.205 per 10^5 population, accounting for 10.4% of all AML.[19]

In our experience, with intensive induction chemotherapy using HA (hemoharringtonine + Ara-C) or HAD (hemoharringtonine + Ara-C + daunorubicin) regimens, patients with AML-M2b tend to achieve a higher complete remission rate and longer disease-free survival than patients with other subtypes of AML.

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