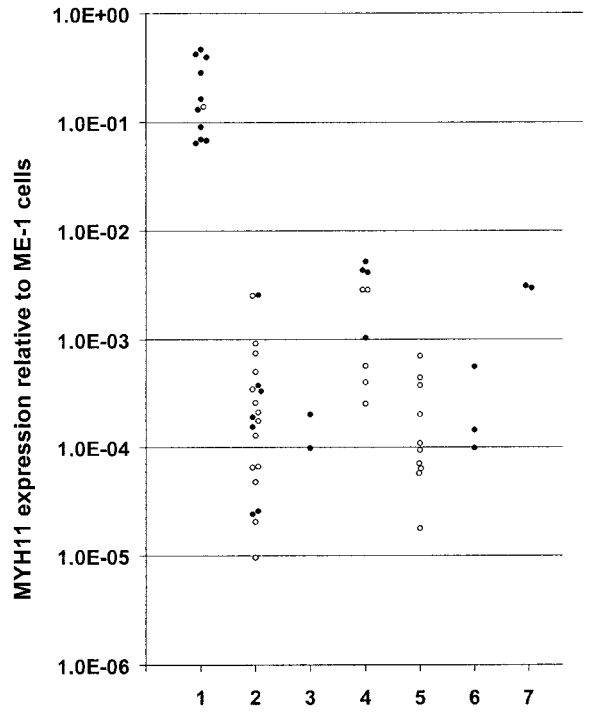

Figure 1. *MYH11* overexpression in *CBFB-MYH11*– positive cells. The expression in *CBFB-MYH11*–positive ME-1 cells was set at 1.0. From all cases informed consent was obtained in accordance with the Declaration of Helsinki. The numbers indicate the following: (1) newly diagnosed *CBFB-MYH11*–positive AML (n = 11); (2) newly diagnosed *CBFB-MYH11*–negative AML (n = 21); (3) *CBFB-MYH11*–positive cases in complete hematologic remission (n = 2); (4) de novo chronic myeloid leukemia (n = 9); (5) history of chronic myeloid leukemia (n = 10); (6) de novo acute lymphocytic leukemia (n = 3); and (7) healthy volunteers (n = 2). ○ and ● indicate blood and bone marrow samples, respectively. Primers and probes were developed downstream of all known *MYH11* fusion points using Primer Express version 1.5 (Applied Biosystems, Foster City, CA). Sequences (5′-3′) of *MYH11* forward and reverse primers and probe are respectively, AGTAGCCTGTCGGGAAGGAAC, GCCTGCTGTGTGGCTTTG, CACTCCAGGACGAGAAGCGCCG. The cDNA synthesis and input, cycling conditions, and *PBGD* expression measured for normalization were as described.[9] For quantification, serial log dilutions of cDNA in H_2O derived from the *CBFB-MYH11*–positive cell line ME-1 were used. Linear amplification extended down to a 4 log dilution.

compared with *CBFB-MYH11*–positive AML cases (Figure 1). We conclude that up-regulation of *MYH11* expression because of the fusion to *CBFB* can be used to rapidly identify *CBFB-MYH11*–positive cases in newly diagnosed AML.

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To the editor:

No exon 4 polymorphism of cytochrome P450 CYP2C9 in Taiwanese

A recent article by Leung et al[1] analyzed the relationship between the genetic polymorphism in exon 4 of cytochrome P450 CYP2C9 and warfarin sensitivity in Chinese patients. They used polymerase chain reaction (PCR) and direct sequencing methods to analyze the genetic changes of the *CYP2C9* gene in 89 patients, and their results showed that 4 polymorphisms in exon 4 were found: heterozygosities for 608TTG>GTG (Leu208Val), 561CAG>CCG (Gln192Pro), 537CAT>CCT (His184Pro), and 527ATT>CTT (Ile181Leu) existed at frequencies 0.75, 0.20, 0.10, and 0.09, respectively. We used a similar approach to analyze the genetic polymorphisms in exon 4 of the *CYP2C9* gene in Taiwanese patients. The primers used to amplify the exon 4 were 5′-AATACAGTGTTTTATATCTAAAG-3′ (GenBank accession number L16879, nucleotide [nt] 1-23) and 5′-TAAGTGGTTTCTCAGGAAGC-3′ (nt 256 to 237) as forward and reverse primers, respectively. We were unable to find these 4 polymorphisms or other new polymorphisms in the exon area in 50 healthy people (Figure 1). In

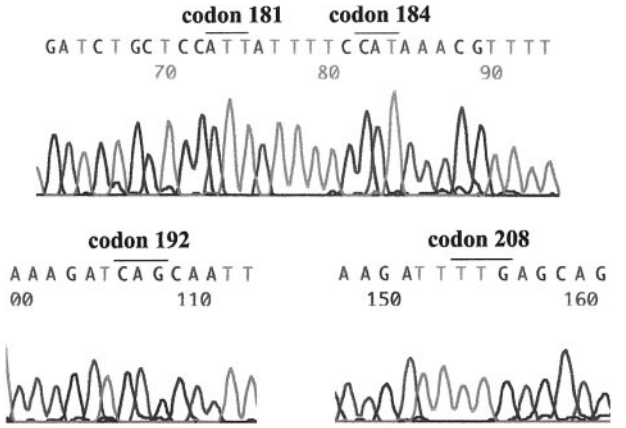

Figure 1. The representative case of direct sequencing analysis for exon 4 polymorphisms of *CYP2C9* gene. No polymorphism was found in exon 4 of 50 cases after sequencing analysis.

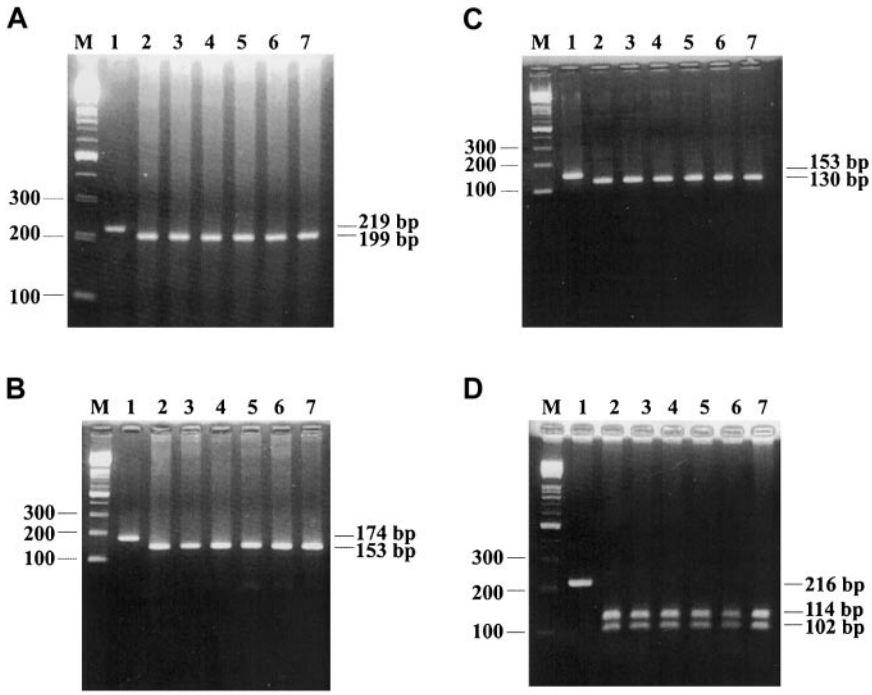

Figure 2. The results of PCR-RFLP analysis for 4 polymorphisms in exon 4 of *CYP2C9* gene. (A) The results of detection of 608TTG>GTG are shown, the wild type (TTG) is completely digested to 199-bp fragment (lanes 2-7) by *Taq*I. (B) The results of detection of 561CAG>CCG are shown, the wild type (CAG) is completely digested to 153-bp fragment by *Alu*I (lanes 2-7). (C) The results of detection of 537CAT>CCT are shown, the wild type (CAT) is completely digested to 130-bp fragment by *Nla*III (lanes 2-7). (D) The results of detection of 527ATT>CTT are shown, the wild type (ATT) is completely digested to 114-bp and 102-bp fragments by *Nla*IV (lanes 2-7). M indicates 100-bp ladder marker; and lane 1, uncut control.

order to double-check our results, we developed a PCR–restriction fragment length polymorphism (RFLP) method to analyze these 4 polymorphisms. For the detection of 608TTG>GTG, we used primer 5′-AATACAGTGTTTTATATCTAAAG-3′ (forward: nt 1 to 23) and 5′-CTGGATCCAGGGGCTGCTCG-3′ (reverse: nt 219 to 190). A mismatched base (underlined) was introduced at the 3′-end base of the reverse primer to create a *Taq*I restriction enzyme cutting site in combination with the base 608T of wild type after PCR and no *Taq*I site for the 608GTG polymorphism. For the detection of 561CAG>CCG, we used the same forward primer, and 5′-TTCCATTAAGTTAAGAAATAGC-3′ (reverse: nt 174 to 153). A mismatched base (underlined) was introduced at position 3 from the 3′ end of the reverse primer to create an *Alu*I site in combination with the base 561A of wild type after PCR and no *Alu*I site for the 537CCT polymorphism. For the detection of 537CAT>CCT, we used the same forward primer and 5′-CTGATCTTTATAATCAAAACGTTCA-3′ (reverse: nt 153 to 129). A mismatched base (underlined) was introduced at position 2 from the 3′ end of reverse primer to create an *Nla*III site in combination with the base 536C and base 537A of wild type after PCR reaction, and no *Nla*III site for the 537CCT polymorphism. For the detection of 527ATT>CTT, we used primer 5′-CCCTGCAATGTGATCTGGTC-3′ (forward: nt 97 to 116) and 5′-GCAATTCAGAGCTTGATCCATG-3′ (reverse: nt 312 to 291). A mismatched base was introduced at position 3 from 5′ end of forward primer to create an *Nla*IV site in combination with the base 527C of wild type after PCR, and no *Nla*IV site for the 527CTT polymorphism. The PCR condition was performed as described,[2,3] except the annealing temperature was done at 55°C, 57°C, 56°C, and 55°C for 608, 561, 537, and 527, respectively. All the above mentioned primer sequences can be found in GenBank accession number L16879. They are specific for the *CYP2C9* gene.

In total, 300 cases were analyzed with these 4 PCR-RFLP methods (Figure 2), and the polymorphisms found by Leung et al were not found in our analysis.

In conclusion, we demonstrated that no genetic polymorphism in exon 4 of cytochrome CYP2C9 is found in Taiwanese, which is different from the results reported by Leung et al. We suggest that further study of the Chinese population is necessary.

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To the editor:

Lack of alteration in GATA-1 expression in CD34$^+$ hematopoietic progenitors from patients with idiopathic myelofibrosis

Idiopathic myelofibrosis (IM), also known as myelofibrosis with myeloid metaplasia, is a myeloproliferative disorder of clonal origin characterized by extramedullary hematopoiesis with a leukoerythroblastic blood picture, tear-drop erythrocytes, and progressive splenomegaly associated with bone marrow fibrosis.[1] A number of data suggested that alterations of megakaryocytopoiesis,