diagnosis of Stevens-Johnson syndrome was made and imatinib was discontinued. No other medication could be implicated. In the next 2 weeks, the rash slowly resolved without the use of steroids. The patient was very keen to restart the medication, and after 3 weeks the drug was started at a lower dose of 300 mg daily. The following day, she developed a pruritic erythematous eruption on her arms that suggested early recurrence. The drug was stopped again and oral prednisolone started at a dose of 30 mg daily. The rash quickly resolved. Two days later the imatinib was restarted at 300 mg together with the prednisolone. There was no further recurrence of the rash. In the next 5 weeks the prednisolone was slowly reduced and stopped while the dose of imatinib remained constant at 300 mg. The patient has been on the drug for a further 15 months with no further recurrence of the rash and is in complete cytogenetic remission.

Case 2 involved a 33-year-old woman with chronic-phase CML who was started on imatinib, 400 mg daily, because of interferon-α intolerance. After 2 weeks she developed an extensive pruritic maculopapular rash. She was advised to stop the imatinib and within a few days the rash had completely cleared. She did not require any steroids. No other cause for the rash could be found and she was not on any other medication. One week after the rash had cleared she was restarted on 100 mg imatinib daily. A week later the dose was increased to 200 mg with no recurrence of the rash and after 4 weeks the dose was again increased, to 300 mg. This was well tolerated, and after 6 weeks the dose was increased to 400 mg daily. By gradually increasing the dose of imatinib we were able to achieve the standard dose without recurrence of the rash. This patient has now been on treatment for 7 months and is currently 99% Philadelphia negative.

Imatinib is the most active agent for the treatment of CML, and skin rashes are quite common with this agent.[1,3] Although caution must be exercised when skin rashes occur, these cases, and others that we have observed, illustrate that it is possible to continue imatinib treatment by using concommitant short-term steroid therapy or by reintroducing imatinib with gradual dose escalation. Although we do not advocate the ongoing use of imatinib in patients who develop very severe skin reactions, careful management and perseverance can often allow patients who develop mild to moderate skin rashes to continue with the drug.

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To the editor:

Cytogenetic characterization reveals that the SAM-1 erythroid cell line is derived from K-562 cells

Recently, it was reported that the TF-1 cell line is derived from K562 cells,[1] and in this context, we cytogenetically characterized several erythroid leukemia cell lines (the SAM-1 and K562 cell lines) using spectral karyotyping (SKY) and fluorescence in situ hybridization (FISH) analysis. The SAM-1 cell line was established from a patient with chronic myelogenous leukemia (CML) in blast crisis. It was described as having the Philadelphia chromosome and a *BCR-ABL* rearrangement pattern that was identical to the patient's original cells, although the full karyotype was not reported.[2] K-562 is a CML cell line established in 1970 from a female patient with CML in blast crisis,[3] and several groups have completely characterized this cell line using FISH (with *BCR/ABL* probes), multicolor FISH, and comparative genomic hybridization.[4,5]

We received the SAM-1 cell line from the original investigators (University Medical Center, Georgetown, Washington, DC); K-562 cells were in house (Sloan Kettering Institute, New York, NY). Both cell lines were cultured in RPMI-1640 medium (Biological Industries, Beit Haemek, Israel) with 10% fetal bovine serum. Cells were harvested using standard methods. SKY was performed according to the manufacturer's protocol (Applied Spectral Imaging, Migdal Haemek, Israel). FISH was done using standard techniques with the locus-specific identifier *BCR/ABL* Dual Color, Dual Fusion Translocation Probe (Vysis, Downers Grove, IL). The karyotype description of SAM-1 based on SKY and FISH is as follows: 68-69 < 3n >, XX,-X, 2ish.add(2)(q37)*(BCR/ABL+)*, der(3)del(3)(p?)t(3;10)(q1?;q24), +der(3)del(3)(p12)t(3;5)(q1?;q?), der(5)t(5;6)(q12-13;?), +del(7)(q21), +der(7)t(7;7)(p?;q?),-9, del(9)(p13), 9ish.der(9)t(9;9)(p1?3;q22)*(ABL+;ABL+)*, der(10)t(3;10)(p21;q24), der(12)t(12;21)(p11.2;q11.2),-13, der(13)t(9;13)(?::13p11—> 13qter) *(BCR/ABL+)*,-14, der(17)t(9;17)(?;p11.2)x2, der(17)t(5;17)(?;q12-21), der(18)t(1;18)(?;q21), dic(6;20)(?;p?), del(20)(q11), der(21)t(1;21)(?;p?), der(22)t(9;22)(q34;q11.2)*(BCR/ABL+)*, + der(22)t(22pter-> 22q11.2::hsr9q34;22q11.2))*(BCR/ABL+)* (Figure 1A).

The karyotype of K562 cells, based on SKY and FISH, shows the same modal number and 14 identical marker chromosomes to SAM-1. It differs from SAM-1 by the presence of –3, dup(6)(pter-> p12::p22qter), and der(10)t(3;10;17)(?;p11.2;q22), and by the absence of der(3)del(3)(p?)t(3;10)(q1?;q24), der(3)del(3)(p12)t(3;5)(q1?;q?)x2, del(7)(q21), der(17)t(5;17)(?;q12-21), and del(20)(q11) (Figure 1B). However, several chromosomal abnormalities found in SAM-1 cells and absent from our K-562 cell line [del(7)(q21), and the del(20)(q11)] have been reported previously in K562 cells.[4] The only unique changes observed in SAM-1 (and never described in K-562 cells) are the chromosome 3 abnormalities and the der(17)(5;17).

An additional common feature between these 2 cell lines is the presence of extensive amplification of *BCR/ABL* fusion genes, clustered on marker chromosomes (Figure 1C). This has been previously described for K-562 cells.[5,6]

In the light of these cytogenetic data, we reviewed our prior polymerase chain reaction–microsatellite analysis of 43 microsatellite loci on chromosome 20 in the K562 and SAM-1 cell lines.[7] An identical pattern was observed in these 2 cell lines at 39 loci (Figure 1D).

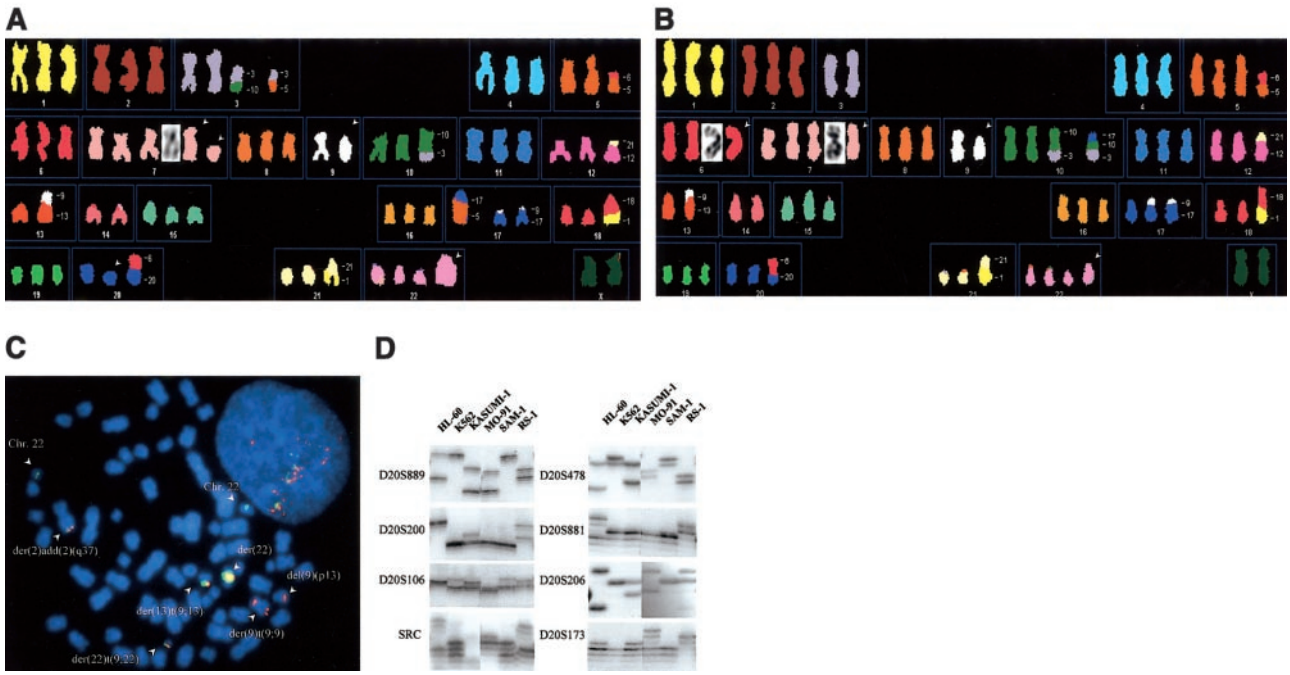

Figure 1. Cytogenetic and molecular characterization of the SAM-1 and K-562 cell lines. SKY analysis of SAM-1 (A) and K-562 (B) cells. Translocations are identified with numbers, and structural abnormalities (observed by G-banding) are indicated with arrows. See text for complete description. (C) Using SAM-1 cells, dual-color FISH probe for *BCR*, (green signal), was seen on 22q11 on 2 normal chromosomes 22, *ABL*, (red signal), was seen on the 9q34 band on the del(9)(p13) and on both ends of der(9)t(9;9)(p1?3;q22). Simple fusion signals were observed in Ph chromosome and in add(2). Multiple fusion signals were seen in the der(13) and in the der(22). (D) PCR-based analysis of 8 dinucleotide repeat loci in 6 myeloid leukemia cell lines. The D20S889 marker is on p-arm, whereas all others are on the q-arm of chromosome 20. SAM-1 and K562 cells exhibit an identical pattern of PCR amplification.

The presence of many identical and very complex structural aberrations, the presence of a unique cell clone, and the microsatellite pattern on chromosome 20 allowed us to conclude that SAM-1 is a derivative of K-562. Drexler and colleagues[8] have estimated that 18% of human tumor cell lines have intraspecies cross contamination, and several cases of cross contamination with the K562 cell line have been reported.[1,8,9] With this report we wish to further emphasize the need for tight control of the identities of the cell lines used in research settings.

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Supported by grant SAF2001-0056 from the Spanish Ministry of Science and Technology.

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To the editor:

Adult acute myeloid leukemia cells do not express nonfunctional Ikaros isoforms

We read with great interest the article by Yagi et al[1] on the expression of nonfunctional Ikaros isoform 6 (Ik6) in childhood acute myeloid leukemia (AML). The authors reported that Ik6 was expressed in 7 of 10 cases of M4 and M5 AML (French-American-British [FAB] classification), but in none with M2 and M7 AML. Their M4 and M5 cases with Ik6 expression had karyotype