Fusion of the MORF and CBP genes in acute myeloid leukemia with the t(10;16)(q22;p13)

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The CBP gene at 16p13 fuses to MOZ and MLL as a result of the t(8;16)(p11;p13) in acute (myelo)monocytic leukemias (AML M4/M5) and the t(11;16)(q23;p13) in treatment-related AML, respectively. We show here that a novel t(10;16)(q22;p13) in a childhood AML M5a leads to a MORF–CBP chimera. RT–PCR using MORF forward and CBP reverse primers amplified a MORF–CBP fusion in which nucleotide 3103 of MORF was fused in-frame with nucleotide 284 of CBP. Nested RT–PCR with CBP forward and MORF reverse primers generated a CBP–MORF transcript in which nucleotide 283 of CBP was fused in-frame with nucleotide 3104 of MORF. Genomic analyses revealed that the breaks were close to Alu elements in intron 16 of MORF and intron 2 of CBP and that duplications had occurred near the breakpoints. A database search using MORF cDNA enabled us to construct an exon–intron map of the MORF gene. The MORF–CBP protein retains the zinc fingers, two nuclear localization signals, the histone acetyltransferase (HAT) domain, a portion of the acidic domain of MORF and the CBP protein downstream of codon 29. Thus, the part of CBP encoding the RARA-binding domain, the CREB-binding domain, the three Cys/His-rich regions, the bromodomain, the HAT domain and the Glu-rich domains is present. In the reciprocal CBP–MORF, part of the acidic domain and the C-terminal Ser- and Met-rich regions of MORF are likely to be driven by the CBP promoter. Since both fusion transcripts were present, their exact role in the leukemogenic process remains to be elucidated.

INTRODUCTION

Acute myeloid leukemias (AMLs) are genetically characterized by the presence of acquired translocations and inversions that result in fusion genes of pathogenetic, diagnostic and prognostic importance (1,2). To date, ∼50 genes rearranged through different AML-associated, cytogenetically balanced, chromosomal abnormalities have been identified (2). Although some genes, e.g. ABL, BCR, ETO and PML, only have been reported to be involved in one, or a few, translocations, it has become increasingly appreciated that several genes, such as MLL and ETV6, are quite promiscuous, having numerous partners in various hematologic malignancies (2). This suggests that the pathogenetic and phenotypic impact of a chimeric gene is dependent on both genes participating in the fusion. However, some genes seem to be quite closely associated with certain morphologic and clinical features irrespective of partner, for example MOZ at 8p11. This gene is known to be fused to CBP, TIF2 and EP300 as a result of the t(8;16)(p11;p13), inv(8)(p1q13) and t(8;22)(p11;q13), respectively, in younger patients with AML characterized by (myelo)monocytic differentiation (3–9). The two known different fusion genes involving CBP at 16p13, on the other hand, are associated with quite distinct hematologic malignancies. The MOZ–CBP chimera generated by the t(8;16) occurs primarily in AML M4/M5, characterized morphologically by erythrophagocytosis and clinically by poor prognosis (10), whereas the MLL–CBP fusion, the molecular consequence of the t(11;16)(q23;p13), has been reported in AML and myelodysplastic syndromes as well as in acute lymphoblastic leukemia (ALL) occurring after previous treatment with DNA topoisomerase II inhibitors (11).

In 1999, Champagne et al. (12) described a MOZ-related gene, MORF, which displays 60% identity and 66% similarity to MOZ and which contains zinc fingers, a histone acetyltransferase (HAT) domain, an acidic region and a C-terminal Ser/Met-rich domain, as does MOZ (3). We recently identified a novel t(10;16)(q22;p13) in a childhood AML M5a. Because MORF is located at 10q22, we surmised that the t(10;16) could result in a MORF–CBP fusion gene, i.e. be a variant of the more common t(8;16)/MOZ-CBP. Further analyses, using fluorescence in situ hybridization (FISH), RT–PCR and genomic extra long (XL)-PCR, confirmed the presence of MORF–CBP and CBP–MORF chimeras, both of which were expressed.

RESULTS

G-band and FISH findings

The G-band and FISH analyses yielded the following karyotype (Fig. 1A): 47,XX,der(7) t(7;10)(p13;p11), +8, der(10)(7;10)(p13;p11)t(10;16)(q22;p13),

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der(16)t(10;16)(q22;p13)[19]/46,XX[6]. FISH signals from the cosmid contig covering the CBP gene were split between the der(10) and der(16).

**Molecular genetic findings**

RT–PCR with various combinations of MORF forward and CBP reverse primers successfully amplified DNA fragments, strongly suggesting the presence of a MORF–CBP chimeric gene. For example, the primer combinations MORF2377F and CBP1201R, MORF2843F and CBP1201R, and MORF2843F and CBP425R yielded bands of sizes 1.7, 1.2 and 0.4 kb, respectively (Fig. 2A). RT–PCR with the primers CBP96F and MORF3383R amplified very weakly a 0.5 kb fragment (data not shown), and nested PCR with CBP174F and MORF3277R generated a 0.3 kb band (Fig. 2B).

The 1.7 kb fragment detected using MORF2377F and CBP1201R was analyzed by direct sequencing, which showed that nucleotide 3103 of MORF (GenBank accession no. AF113514) was fused in-frame with nucleotide 284 of CBP (NM_004380) (Fig. 3). The sequence analysis of the nested PCR product obtained by CBP174F and MORF3277R revealed that nucleotide 283 of CBP was fused in-frame with nucleotide 3104 of MORF (Fig. 4), that the initiation ATG codon of CBP remained intact and that there was no mutation or deletion in the part of CBP included in the chimeric CBP–MORF transcript.

XL-PCR with the primer combinations MORF2913F and CBP517R and CBPINT33245F and MORF3383R amplified a 3.1 and a 4.1 kb genomic fragment, respectively (Fig. 5).

Partial sequencing of the 3.1 kb band showed that the breakpoints were located in intron 16 of MORF and intron 2 of CBP (Fig. 6). In the MORF–CBP hybrid gene, the genomic break in MORF occurred 1157 bp downstream of exon 16, ~250 bp downstream of an AluJb and 450 bp upstream of the AluSx repetitive element. The breakpoint in CBP was 1569 bp upstream of exon 3, in the beginning of an AluYb8 repeat. In the reciprocal CBP–MORF chimera, the break in CBP occurred 1520 bp upstream of exon 3, 88 bp downstream of that found in the MORF–CBP fusion (Fig. 7). The breakpoint in MORF occurred 941 bp downstream of exon 16, close to the AluJb repeat, and was 216 bp upstream of the genomic break in the MORF–CBP hybrid gene. Thus, duplications of 215 bp (intron 16, MORF) and 87 bp (intron 2, CBP) had occurred in the close vicinity of the breakpoints.

By searching the MORF cDNA (GenBank accession no. AF113514) against the sequences of GenBank + EMBL + DDBJ + PDB through the blast service of the National Center for Biotechnology Information, genomic clones containing various parts of the MORF gene were retrieved (GenBank accession nos AC063962.3, AC073371, AL360225 and AC018511.4). The MORF cDNA was then aligned with the genomic sequences in order to identify splice sites and number of exons. Subsequently, an exon–intron map was constructed containing the entire MORF gene, found to be ~112 kb and composed of 17 exons (Fig. 8). Exons 7 and 17 are the smallest (55 bp) and the largest (2557 bp), respectively. Intron sizes vary from 243 bp (intron 4) to 40 986 bp (intron 2). The
starting codon is located in exon 2. Exons 3–6 code for zinc fingers. Exon 7 has two additional splice sites, which result in the MORFα and β isoforms (GenBank accession nos AF119230 and AF119231, respectively). The HAT domain is encoded by exons 9–14. Exons 15, 16 and the first part of exon 17 code for the acidic domain of the MORF protein. Ser- and Met-rich regions are encoded by the middle and the last part of exon 17, the two nuclear localization signals by exons 6 and 16, C2HC zinc fingers by exon 10 and a putative acetyl-CoA-binding site by exon 12. The intron 16, in which the genomic breakpoint occurred, is 3238 bp and contains two AluJbs, one AluSx and a small region rich in TA-repeats.

**DISCUSSION**

In this study, FISH, RT–PCR and genomic XL-PCR were used to identify and characterize the MORF–CBP and CBP–MORF chimeras caused by an AML-associated t(10;16)(q22;p13) (Fig. 1A). Moreover, an exon–intron map of the MORF gene was constructed and the genomic breakpoints were precisely mapped in both MORF and CBP.

The present case displayed some clinical similarities with published AML with MOZ rearrangements: the patient was young and the AML was classified as AML M5a. The median age of AML patients with the t(8;16), inv(8) or t(8;22) is only 29 years and the vast majority are AML M4/M5 (2). However, there were no signs of erythrophagocytosis, which is a morphologic hallmark of t(8;16)-positive AML (10). Previously, only one AML, an AML M4, with a similar translocation, t(10;16)(q21;p13), has been reported (13), but that case also harbored an 11q23 rearrangement that could possibly affect the MLL gene. Hence, it is obviously premature to draw any firm conclusions as regards morphologic and clinical features of t(10;16)-positive AML.

We recently reported that both the MOZ–CBP and the CBP–MOZ chimeric genes are expressed in t(8;16)-positive AML (9). Two types of MOZ-CBP transcript, type I and II, were detected. In type I, the part of MOZ upstream of the large exon encoding the acidic domain of the protein was fused in-frame with exon 3 of CBP, whereas, in type II, the same part of MOZ was fused out-of-frame with the fourth exon of CBP. In the reciprocal CBP–MOZ transcript, exon 2 of CBP was fused in-frame with the exon coding for the acidic domain of MOZ. Considering the high homology between MORF and MOZ–MORF transcripts would be similar to MOZ–CBP and CBP–MOZ. In addition, the present FISH analysis revealed that the break in CBP occurred in the segment covered by the cosmid RT166 (Fig. 1B), a probe frequently rearranged in the t(8;16) (14). Thus, we used the same CBP reverse primers as in our
previous study (9) to detect a possible MORF–CBP fusion transcript, and we placed the MORF forward primers upstream of the part of the gene encoding the acidic domain. For the reciprocal CBP–MORF transcript, the CBP forward primers were used to detect CBP–MOZ (9) and the reverse primers of MORF were positioned in the region of MORF coding for the acidic domain. One-step PCR with various MORF forward and CBP reverse primers amplified an in-frame MORF–CBP cDNA fragment (Figs 2A and 3). As in the case of CBP–MOZ (9), nested PCR was required for the detection of an in-frame CBP–MORF transcript (Figs 2B and 4), indicating that it is less expressed than MORF–CBP.

For the characterization of the genomic breakpoint in MORF, an exon–intron map was constructed (Fig. 8), which made it possible to localize the breaks to intron 16, upstream of exon 17 which encodes the acidic domain and the Ser- and Met-rich regions of the MORF protein. This location seems to be similar to previously mapped breakpoints in the MOZ gene, although no detailed sequence data have been reported (4–9,15). In all published myeloid malignancies with MOZ rearrangements, i.e. t(8;16)/MOZ-CBP, inv(8)/MOZ-TIF2 and t(8;22)/MOZ-EP300, the breakpoints have occurred in the intron just upstream of the 4 kb-long exon starting from position 3746 and ending at position 7747 of the MOZ cDNA (4–9,15). The fusion points of MORF–CBP and CBP–MORF chimeric transcripts and the exon–intron structure of the CBP gene suggested that the genomic breakpoint was located in intron 2 of CBP, as in the t(8;16) (9,14,16). This was verified by XL-PCR and direct sequencing (Figs 5–7).

Figure 4. The CBP–MORF chimeric transcript. (A) Partial sequence chromatogram showing the junction of the CBP and MORF genes (arrow). (B) Nucleotide sequence of the amplified fragment determined after direct sequencing. The arrow shows the junction (GA) of the CBP and MORF genes. The nucleotide 283 of CBP is fused in-frame with nucleotide 3104 of MORF. The primers CBP174F and MORF3277R (Table 1) are underlined.

Figure 5. XL-PCR for the detection of MORF–CBP and the reciprocal CBP–MORF hybrid genomic DNA. (A) Lane 1, XL-PCR using the MORF2913F and CBP517R primers amplified a 3.1 kb genomic fragment; lane 2, negative control (XL-PCR performed on genomic DNA from a healthy donor); lane 3, blank, no DNA in the PCR; lane M1, λDNA/HindIII DNA ladder; lane M2, 1 kb DNA ladder. (B) Lane 1, XL-PCR using the CBPIXNT33245F and MORF3383R primers amplified a 4.1 kb genomic fragment; lane 2, negative control (XL-PCR performed on genomic DNA from a healthy donor); lane 3, blank, no DNA in the PCR; lane M1, λDNA/HindIII DNA ladder; lane M2, 1 kb DNA ladder.
The present genomic data show that the breakpoints were very close to Alu repetitive elements in intron 16 of MORF as well as in intron 2 of CBP. Although there was no evidence for recombination of Alu repeats, the involvement of these sequences in the genesis of the t(10;16) cannot be ruled out. It is noteworthy in this context that the observed breaks were quite complex in the sense that no end-to-end reciprocal translocation had occurred; instead, intronic duplications were present in the breakpoint regions of both MORF and CBP (Figs 6 and 7). Several neoplasia-associated translocations have been investigated at the genomic level, for example the t(4;11)/MLL-AF4 in ALL, t(9;22)/BCR-ABL in chronic myeloid leukemia, t(11;22)/EWS-FLI1 in Ewing sarcoma, t(12;16)/FUS-CHOP in myxoid liposarcoma, t(12:21)/ETV6-CBFA2 in ALL and t(15;17)/PML-RARA in acute promyelocytic leukemia (17–24). These studies have revealed that cytogenetically balanced, reciprocal translocations are rarely true end-to-end fusions. In contrast, they are often accompanied by additional genomic aberrations, such as deletions, insertions, inversions and duplications. However, there are only a few examples of genomic characterization of both derivative chromosomes, e.g. ETV6-CBFA2 in ALL (22), MLL-AF4 in ALL (23,24), MLL-AF9 in AML (25) and EWS-FLI1 in Ewing sarcoma (21). As regards the present finding of intronic duplications, this is reminiscent of what has been reported in a subgroup of Ewing sarcomas (21) and in ALL with MLL-AF4 (23,24). Thus, the genomic fusion regions in MORF–CBP and the reciprocal CBP–MORF hybrids share characteristic features of the damage repair model described by Reichel et al. (23) and Gillert et al. (24).

The putative MORF–CBP chimeric protein retains the part of MORF that encodes the zinc fingers, two nuclear localization signals, the HAT domain and a portion of the acidic domain and the CBP protein downstream of codon 29, which also contains a HAT domain (Fig. 9). Thus, as in MOZ–CBP transcript type I, the part of CBP coding for the domain that binds to the nuclear receptor RARA was present in addition to the CREB-binding domain, the three Cys/His-rich regions, the bromodomain and the Glu-rich domains (9). In the reciprocal CBP–MORF fusion, similar to CBP–MOZ, part of the acidic domain of MORF, the Ser-rich region and the highly Met-rich C-terminal part are likely to be driven by the CBP promoter. Because both reciprocal fusion transcripts were detected, it is presently unknown which of them is leukemogenic.

This study shows, for the first time, the involvement of MORF in a neoplasia-associated chromosomal translocation. The CBP gene and its homolog EP300 at 2q13, on the other hand, have previously been reported to be fused to MOZ and MLL (3,7,9,11,26,27). In addition, the TIF2 gene, at 8q13, has also been described as a partner to MOZ (4,5,8,15). Considering
these findings, it is tempting to suggest that MORF-EP300 and MORF-TIF2 chimeras will be detected in hematologic malignancies with the t(10;22)(q22;q13) and t(8;10)(q13;q22), respectively. However, to date no such translocations have been reported (2).

MATERIALS AND METHODS

Patient

A previously healthy 4-year-old girl was admitted to hospital because of fatigue and easy bruising. The clinical examination revealed petechiae, subcutaneous hematomas and lymphadenopathy, but no hepato- or splenomegaly. The peripheral blood count showed 71 g/l hemoglobin, 31 × 10^9/l white blood cells, with 50% blasts and 24 × 10^9/l platelets. The bone marrow aspirate displayed 94% blasts that were medium-sized, with a medium to high nuclear:cytoplasmic ratio and a basophilic cytoplasm. The nuclei were rounded, with a finely dispersed chromatin and prominent nucleoli. Approximately 50% of the blasts stained positively for unspecific esterase, whereas 3% were positive for myeloperoxidase. No Auer rods were detected, and there was no erythrophagocytosis. A diagnosis of AML M5a was made. It was difficult to establish a distinct immunophenotype of the leukemic cells. The population was reported to be positive for CD4, CD7 and CD15 and negative for CD3, CD8, CD19, CD20, CD22, CD13, CD33, CD65, CD14, CD117 and CD56. The patient is presently being treated according to NOPHO-93 [chemotherapeutic agents and doses as in NOPHO-88 (28)].

Chromosome banding and FISH analyses

Bone marrow cells were cytogenetically investigated by standard methods, and the description of karyotypes and the clonality criteria followed the recommendations of ISCN (29). For FISH, performed as described previously (30), the following probes were used: whole chromosome painting.
(wcp) probes for chromosomes 7, 8, 10 and 16 (Vysis) and a cosmid contig covering the CBP gene (telomere to centromere: RT100, RT102, RT191, RT203 and RT166; generously supplied by Dr M.H. Breuning, Leiden, The Netherlands) (14).

**PCR and sequence analyses**

The primers used for PCR amplification and sequence analyses are listed in Table 1. RT–PCR was carried out for the detection of MORF–CBP and the reciprocal CBP–MORF chimeric transcripts using total RNA, which was extracted from the bone marrow cells using the Trizol reagent according to the manufacturer’s instructions (Gibco BRL). Five micrograms of total RNA was reverse-transcribed in a 20 µl reaction volume containing 50 mM Tris–HCl pH 8.3 (at 25°C), 75 mM KCl, 3 mM MgCl₂, 10 mM DTT, 1 mM of each dNTP, 37 U of RNA guard (Pharmacia), 10 pmol random hexamers, 1 µg of oligo(dT)₁₀ and 400 U of M-MLV reverse transcriptase (Gibco BRL). The reaction was carried out at 37°C for 60 min, heated for 10 min at 65°C and then kept at 4°C.

A one-step PCR was performed for amplification of the MORF–CBP fusion transcript. The 50 µl reaction volume contained 20 mM Tris–HCl pH 8.4 (at 25°C), 50 mM KCl, 1.25 mM MgCl₂, 0.2 mM of each dNTP, 1 U of Platinum Taq DNA polymerase (Gibco BRL), 0.5 µM of each of the MORF forward (MORF2377F, MORF2843F) and CBP reverse (CBP1201R, CBP425R) primers (Table 1) and 2 µl of the cDNA. A nested PCR was performed for the detection of the CBP–MORF transcript. The 50 µl reaction volume in both the first- and second-round PCR had the same composition as above except that CBP forward and MOZ reverse primers were used. In the first-round PCR, 2 µl of the cDNA was amplified using the primers CBP96F and MORF3383R (Table 1) and 2 µl of the cDNA. A nested PCR was performed for the detection of the CBP–MORF transcript. The 50 µl reaction volume in both the first- and second-round PCR had the same composition as above except that CBP forward (MORF2377F, MORF2843F) and CBP reverse (CBP1201R, CBP425R) primers (Table 1) and 2 µl of the cDNA. A nested PCR was performed for the detection of the CBP–MORF transcript. The 50 µl reaction volume in both the first- and second-round PCR had the same composition as above except that CBP forward and MOZ reverse primers were used. In the first-round PCR, 2 µl of the cDNA was amplified using the primers CBP96F and MORF3383R (Table 1). Two microlitres of the first PCR product was then reamplified in a second PCR with the inner primers CBP174F and MORF3277R (Table 1). After an initial denaturation at 94°C for 5 min, 30 cycles of 1 min at 94°C, 1 min at 60°C and 1 min at 72°C were run using a PCT-200 DNA Engine (MJ Research), followed by a final extension for 10 min at 74°C.
For the detection of genomic MORF–CBP and CBP–MORF hybrids, XL-PCR was carried out using the XL-PCR kit (Perkin Elmer). XL-PCR was performed in 100 µl of 1.3 diluted 3.3× XL buffer, 1.1 mM Mg(OAc)₂, 0.2 mM of each dNTP, 1 U of rTth DNA polymerase XL, 0.4 µmol of each of the forward and reverse primers [MORF2913F and CBP517R for MORF–CBP and CBPINT33245F and MORF3383R for CBP–MORF (Table 1)] and 1.5 µg of DNA extracted from bone marrow cells according to standard methods (31). After an initial denaturation for 1 min at 94°C, 32 cycles of 15 s at 94°C and 10 min at 68°C were run using a PCT-200 DNA Engine (MJ Research), followed by a final extension for 10 min at 72°C.

Fifteen microlitres of the PCR products was analyzed by electrophoresis through 1.5% agarose gels, stained with ethidium bromide and photographed.

For sequence analyses, the RT–PCR- and XL-PCR-amplified MORF–CBP and CBP–MORF fragments were run on 1.0% agarose gels, purified using a Qiagen gel extraction kit and directly sequenced using the dideoxy procedure with an ABI Prism BigDye terminator cycle sequencing ready reaction kit (PE Applied Biosystems) with various primers (Table 1) on the Applied Biosystems model 373A DNA sequencing system. The BLAST software (http://www.ncbi.nlm.nih.gov/BLAST/) was used for computer analysis of sequence data, and screening for repetitive elements was performed using the RepeatMasker web server (http://ftp.genome.washington.edu/cgi-bin/RepeatMasker).

ACKNOWLEDGEMENTS

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REFERENCES

Table 1. Primers for RT–PCR and sequencing

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