Novel Interaction between the Transcription Factor CHOP (GADD153) and the Ribosomal Protein FTE/S3a Modulates Erythropoiesis

Kunyuan Cui, Margaret Coutts, Joachim Stahl‡, and Arthur J. Sytkowski§

From the Laboratory for Cell and Molecular Biology, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, Massachusetts 02215 and the Max Delbrück Center for Molecular Medicine, Robert Rössle-Strasse 10, D-13122 Berlin-Buch, Germany

The transcription factor CHOP (GADD153) heterodimerizes with other C/EBP family members, especially C/EBPβ, thus preventing their homodimerization and binding to DNA sequences specific for the homodimers. Some CHOP-C/EBP heterodimers apparently bind to alternative DNA sequence and thereby regulate the transcription of other genes. Recently, we demonstrated that CHOP is up-regulated during certain stages of erythroid differentiation and that ectopic overexpression of CHOP enhances this process (Coutts, M., Cui, K., Davis, K. L., Keutzer, J. C., and Sytkowski, A. J. (1999) Blood 93, 3369–3378). In the present study, we report that CHOP also interacts with another non-C/EBP protein designated v-fos transformation effector (FTE) (Kho, C. J., and Zarbl, H. (1992) Proc. Natl. Acad. Sci. U. S. A. 89, 2200–2204), which is identical to ribosomal protein S3a (Metspalu, A., Rebane, A., Hoth, S., Pooga, M., Stahl, J., and Kruppa, J. (1992) Gene (Amst.) 119, 313–316). Bacterially expressed His-CHOP and in vitro translated 35S-labeled FTE/S3a-Gal4 fusion protein co-immunoprecipitated using anti-CHOP antibodies, and both anti-CHOP and anti-FTE/S3a antibodies co-immunoprecipitated CHOP and FTE/S3a from lysates of Rauscher murine erythroleukemia cells overexpressing both proteins. The in vitro interaction of CHOP and FTE/S3a was also demonstrated in cells overexpressing FTE/S3a but with endogenous expression levels of CHOP. Western blot analysis demonstrated co-localization of CHOP and FTE/S3a in both the cytosol and the nuclei of non-transfected cells. Overexpression of FTE/S3a inhibited differentiation of Rauscher cells induced either by erythropoietin or by dimethyl sulfoxide. This inhibition was reversed partially by simultaneous overexpression of CHOP or of antisense fes/S3a. FTE/S3a appears to be a bifunctional ribosomal protein that regulates CHOP and, hence, C/EBP function during erythropoiesis.

The growth of erythroid progenitor cells in the bone marrow and their differentiation into enucleate, hemoglobinized erythrocytes is regulated primarily by the glycoprotein hormone erythropoietin (Epo)1 (1). This growth factor interacts with its cognate receptor on the surface of the erythroid cell and triggers a signal transduction cascade that results in cell proliferation (anti-apoptosis) and differentiation (2–13).

A role in erythroid growth and development has been shown or suggested for several regulatory proteins including Myc, Myb, GATA-1, and NF-E2 (4, 7, 8, 14–19). Recently, we found that Epo up-regulates the expression of CHOP (gadd153) (20–24), a member of the C/EBP family of transcription factors (25). Gain-of-function studies indicated that increasing CHOP expression enhances hemoglobinization of erythroid cells, indicating a functional role for CHOP in erythroid differentiation. Additionally, we obtained evidence that CHOP protein can bind to several nuclear proteins from erythroid cells, potentially some that are not C/EBP family members.

We have now used the yeast two-hybrid system (26) to screen an erythroid cell cDNA library for proteins that interact with CHOP. We report that CHOP interacts with a non-C/EBP protein designated v-fos transformation effector (FTE) (27) which is identical to ribosomal protein S3a (28). Our results indicate that this interaction inhibits the ability of CHOP to enhance erythroid differentiation.

EXPERIMENTAL PROCEDURES

Rauscher Murine Erythroleukemia Cell cDNA Library Construction and Yeast Two-hybrid Screen—A library from Rauscher murine erythroleukemia cells (29, 30) was constructed into the pAD-Gal4 vector. Briefly, 5 μg of poly(A)+ RNA was converted to double-stranded DNA using Stratascript RNase H− reverse transcriptase (Stratagene). First strand synthesis was primed with an oligonucleotide containing poly(dT) and an XhoI site (31). Second strand synthesis was carried out using Escherichia coli DNA polymerase I and RNase H (32). The cDNA ends were filled with T4 DNA polymerase, and the internal EcoRI sites of the cDNA were methylated with EcoRI methylase. Following EcoRI linker addition and size fractionation in a 1% agarose gel, double-stranded cDNA over 800 base pairs was cleaved with EcoRI and XhoI. The fragments purified from agarose gel were ligated into the HybriZAP vector in unidirection (Stratagene). After in vitro packaging, 106 recombinant phages were plated, and the HybriZAP Lambda library was converted into the phagemid library pAD-GAL4 after in vitro mass excision.

For the expression of CHOP as a bait in the yeast two-hybrid system, two restriction enzyme cleavage sites were introduced into the two ends of CHOP cDNA by the polymerase chain reaction (PCR) using the following primers, 5′-TTCCGACGACCTAGTGCAAGCCGCA-3′ and 5′-TTAAGGATCTGGCAAGCCGCACTGATGCCTGCTTT-3′. The first primer introduced a SalI second site at the 5′ end of the cDNA, and the second primer added an EcoRI site at the 3′ end of cDNA. The 5′ end nucleotide sequence of CHOP was also adjusted by primer design so that the CHOP cDNA was in the right reading frame of the Gal-4 DNA binding domain. After cleavage, the PCR product was cloned into the SalI and

buffered saline; PAGE, polyacrylamide gel electrophoresis; DMEM, Dulbecco’s modified Eagle’s medium; FCS, fetal calf serum; eIF, eukaryotic initiation factor.

*This work was supported in part by National Research Service Awards F32 DK09201 (to K. C.) and F32 DK08866 (to M. C.) and National Institutes of Health Grant DK38841 (to A. J. S.). The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

‡ To whom correspondence should be addressed. Tel.: 617-632-9980; Fax: 617-632-0401; E-mail: asystkows@caregroup.harvard.edu.

§ This paper is available on line at http://www.jbc.org

1 The abbreviations used are: Epo, erythropoietin; FTE, v-fos transformation effector; PCR, polymerase chain reaction; PBS, phosphate-buffered saline; PAGE, polyacrylamide gel electrophoresis; DMEM, Dulbecco’s modified Eagle’s medium; FCS, fetal calf serum; eIF, eukaryotic initiation factor.
Interaction of CHOP and FTE/S3a

EcoRI sites of the pBD-Gal4 yeast vector. The library was used to screen for CHOP binding partners following the manufacturer’s protocol (Stratagene). Positive yeast clones were selected by protophory for histidine and expression of β-galactosidase. Yeast DNA was recovered and transformed into E. coli. Plasmids containing cDNA clones were identified by screening and mapping by sequencing cDNA and genomic DNA. Subsequent two-hybrid interaction was carried out with the positive and negative controls, including plasmids containing the Gal4 DNA-binding (pBD-Gal4) and Gal4 activation (pAD-Gal4) domains in Saccharomyces cerevisiae strain SPY526.

In Vitro and in Vivo Expression Constructs—For in vitro transcription and translation, fte-1 cDNA was co-translated with Saccharomyces cerevisiae strain SPY526. The PCR product was cleaved from the pAD-GAL4 vector with HindIII and SalI and subcloned into the same sites of the pSP72 vector (Promega). Sp6 polymerase was used for in vitro transcription and translation assays according to the manufacturer’s procedure. For expression of CHOP protein in E. coli, BamHI and EcoRI restriction sites were introduced at the ‘5’ and ‘3’ ends of the cDNA, respectively, by PCR using the following two primers, 5’-TTAAGGATCCGACGCTGACCTCCCGGCGGA-TATACCACTGTTGATTACC-3’. The PCR product was cleaved with BamHI and EcoRI and was cloned into the pTrcHis expression vector (Invitrogen) in the reading frame. Therefore, CHOP was expressed as a His-tagged protein. For mammalian cell expression of CHOP, an SpSbEcoRI fragment containing CHOP cDNA from the pTrcHis vector was cleaved using SalI and BamHI and sub-cloned into the Smal site of pSVK3 expression vector (Amersham Pharmacia Biotech) under the transcriptional control of SV40 early promoter. The orientation of the insert was confirmed by restriction enzyme mapping and DNA sequence analysis. In order to express fte-1 in the mammalian cells, the 5’-untranslated region of rat fte-1 and one start codon (ATG) was added to the 5’ end of mouse fte-1 cDNA by PCR using following primers, 5’-GGCCATCTAGCTTGGGCTGAGCAGGCGCCAT-AGTCCCCGCTGGAACGACAAACAAAG-3’ and Sp6 primer 5’-GA-TATACCACTGTTGATTACC-3’. The PCR product was cleaved into the pZeoSv vector (Invitrogen) for eukaryotic expression under the transcriptional control of SV40 promoter and enhancer with Zeocin resistance as a selection marker.

Antibodies to Ribosomal Proteins—Polyclonal antisera recognizing FTE/S3a or ribosomal protein S2B were raised in rabbits or goats by immunization with the corresponding ribosomal proteins purified from rat liver ribosomes by a combination of carboxymethylcellulose chromatography, reversed phase liquid chromatography, and polyacrylamide gel electrophoresis. From the sera, monospecific antibodies were prepared by immunosorption to the purified ribosomal proteins immobilized on CNBr-Sepharose 4B (Amersham Pharmacia Biotech) as described (33). Anti-S Labeling and Immunoprecipitation—Rabbit polyclonal antisera recognizing His-CHOP was prepared by Organon Teknika using anti-CHOP was affinity purified using His-CHOP as the ligand covalently linked to Affi-Gel 15 (Bio-Rad).

S-Labeled proteins were generated with TNT SP6 polymerase-coupled reticulocyte lysate system according to the manufacturer’s protocol (Promega). Five μl of the 50-μl translation mixture was used for immunoprecipitation. In vitro translation products were combined with 250 μl of binding buffer (20 mM Tris-HCl, pH 7.5, 500 mM NaCl, 1 mM EDTA, 1 mM phenylmethylsulfonyl fluoride, 1 μg/ml leupeptin, and 0.25 mM dithiothreitol) and preclarified by addition of 10 μl of preimmune rabbit serum. After the mixture containing preimmune serum was incubated for 1 h at 4 °C, 10 μl of immunoprecipitin (Life Technologies, Inc.) were added, and the mixture was incubated for another hour. Nonspecific immune complexes were pelleted by slow speed centrifugation and discarded. The supernatant was mixed with 3 μl of bacterially expressed CHOP (6.0 μg), anti-CHOP antibody, and immunoprecipitin. The mixture was incubated at 4 °C for 2 h. The beads were collected by brief centrifugation and washed four times with washing buffer (same as binding buffer except containing 200 mM NaCl). The pellet was resuspended in 50 μl of binding buffer, and the slurry was heated to 100 °C for 5 min in a water bath. After centrifugation in a microcentrifuge for 2 min, the supernatant was carefully removed, and 200 μl of binding buffer was added. Another round of immunoprecipitation was carried out as described above. After extensive washing, the precipitate was resuspended on SDS-polyacrylamide gels. The gel was dried and exposed to x-ray film.

Western Blot Analysis of FTE/S3a and CHOP—Rauscher cells were harvested by centrifugation and were washed twice with ice-cold phosphate-buffered saline. A 5-fold volume excess packed-cell pellet of Dignam buffer A (34) was added to the pellets that were gently resuspended by low speed vortexing. The mixture was incubated at 0 °C for 10 min and was centrifuged at low speed in a microcentrifuge followed by vigorous vortexing. The nuclei were pellets; the supernatant was removed, and four original cell pellet volumes of Dignam buffer C were added. The nuclei were lysed by 20 strokes of a Dounce homogenizer (type B pestle). The lysate was shaken in a microcentrifuge tube on a tuberculin syringe and then resuspended in 100 μl of SDS buffer containing CHOP and FTE/S3a on each membrane half, respectively.

RESULTS

To carry out the two-hybrid screen, CHOP was fused to the Gal-4 DNA binding domain in a pBD-Gal4 vector. This bait plasmid was co-transformed into cells with a Rauscher murine erythroleukemia cell cDNA library fused with the Gal-4 activation domain in a pAD-Gal4 vector. Ten positive clones were obtained from 3 x 10⁶ transformations screened.

To determine the specificity of the interaction, plasmids containing activation domain fusion proteins from the putative positive clones were co-transformed into yeast with Gal-4 binding domain CHOP and control heterologous baits. Nine clones were found to interact with the Gal-4 DNA domain protein containing CHOP but not with the DNA vector alone or with the heterologous bait. DNA sequence analysis revealed that...
two of the nine clones encoded the mouse homologue of rat v-fos transformation effector-1 gene (fte-1) (27), also known as ribosomal protein S3a (28). Within the coding region, the mouse and rat nucleotide sequences are 97% identical. The deduced amino acid sequences are identical. Data base searches did not identify sequences with significant similarity to the other positive clones.

We demonstrated the interaction of the CHOP and FTE/S3a proteins in vitro. The fte/S3a Gal-4 DNA binding domain clones were subcloned into a pSPT2 vector containing the SP6 promoter. 35S-Labeled protein prepared by in vitro transcription and translation was tested for association with bacterially expressed recombinant CHOP protein. Immunoprecipitation with anti-CHOP antibodies showed that CHOP bound to the GAL4-FTE/S3a fusion protein (Fig. 1).

Next, we confirmed the in vivo interaction of FTE/S3a and CHOP in erythrocyte cells. Rauscher cells were co-transfected transiently with fte/S3a and CHOP cDNAs and incubated with [35S]methionine. Lysates were prepared and were incubated either with anti-FTE/S3a or with anti-CHOP antibodies followed by precipitation with protein A-agarose, SDS-PAGE, and autoradiography. Incubation with either antibody resulted in co-immunoprecipitation of both CHOP and FTE/S3a as a closely spaced doublet (Fig. 2).

We also confirmed the identities of the co-immunoprecipitated proteins and demonstrated both cytosolic and nuclear co-localization of FTE/S3a and CHOP. Non-transfected Rauscher cells were lysed, and cytosolic and nuclear protein fractions were prepared and subjected to SDS-PAGE and Western blot analysis (Fig. 3). CHOP was detected in both the cytosolic and, in a significantly higher amount, in the nuclear fraction. It migrated as a single, well resolved species aligned precisely with the 29-kDa molecular mass standard. A trace amount of a higher molecular weight cross-reacting protein was also seen. FTE/S3a was also detected in both the cytosolic and the nuclear fractions but in approximately equal amounts. It also migrated as a single, well resolved species that, in contrast to CHOP, was slightly (~2 mm) ahead of the 29-kDa molecular mass standard, thus identifying CHOP and FTE/S3a as the upper and lower members, respectively, of the closely spaced doublet seen in Fig. 2.

In a second experiment we established a Rauscher cell line stably overexpressing fte/S3a. Cells were lysed and the cytosolic fraction was incubated with anti-CHOP antibody followed by immunoprecipitation, SDS-PAGE, and Western blot analysis with anti-FTE/S3a antibody (Fig. 4). Cytosolic fractions were also subjected to SDS-PAGE and Western blot. Anti-FTE/S3a antibodies identified FTE/S3a in the cytosolic fractions and in the immunoprecipitate obtained with the anti-CHOP antibodies again running slightly ahead of the 29-kDa standard.

The evidence for co-localization of CHOP and FTE/S3a in both the nucleus and the cytosol prompted us to ask whether the interaction might also occur within the ribosome itself. Immunoelectron microscopy studies have mapped FTE/S3a to the surface of the small subunit and the 80 S ribosome (33).
Therefore, we reasoned that if CHOP binds to FTE/S3a on the surface of the ribosome, immunoprecipitation with anti-CHOP antibody should result in the appearance of other ribosomal proteins in the immunoprecipitate, including proteins such as RPS26, which is located within the ribosomal particle rather than at its surface (33, 35) and is not known to interact with FTE/S3a. We prepared cytosolic and nuclear extracts of Rauscher cells and carried out an immunoprecipitation with anti-CHOP antibody (see “Experimental Procedures”). Then the immunoprecipitates, along with purified ribosomal total protein (containing rpS26) and Rauscher cell cytosolic lysate, were subjected to SDS-PAGE, electrophoretic transfer to nitrocellulose, and probing with anti-S26 antibody. As seen in Fig. 5, S26 protein was detected readily in the control purified ribosomal protein fraction (Fig. 5, lane 1) and in the Rauscher cell cytosolic lysate (Fig. 5, lane 2). However, S26 was not detected in the anti-CHOP cytosolic immunoprecipitate (Fig. 5, lane 3) nor in the nuclear immunoprecipitate (Fig. 5, lane 4). Similar results were obtained in several repeat experiments. Occasionally, an extremely faint band was detected in the cytosolic immunoprecipitate. However, this was not seen regularly and may have represented a small amount of ribosomal contamination of the centrifuged immunoprecipitate pellet.

Northern blot analysis showed that fte/S3a transcript is approximately 0.9 kilobase pairs, close to the size of the cDNA clones. The mRNA level was not induced by Epo or dimethyl sulfoxide (Me2SO) (not shown).

Since we had discovered previously that up-regulation of CHOP enhanced hemoglobinization in response to Epo or Me2SO (25), and since CHOP and FTE/S3a interact, we hypothesized that FTE/S3a might participate in regulating erythroid differentiation. To test this, Rauscher cells were transfected with fte/S3a. Transfection efficiency was monitored by transfection efficiency of 10 and 20% Hb S3a, respectively (Fig. 6, lane 1 and 2, Rauscher cell cytosolic lysate; lane 3, anti-CHOP cytosolic immunoprecipitate; lane 4, anti-CHOP nuclear immunoprecipitate. See “Experimental Procedures” and “Results.”

The results of transiently overexpressing CHOP in the fte/S3a-transfected cells (Fig. 6, FTE/CHOP). Such CHOP overexpression also resulted in partial reversal of the FTE/S3a inhibition of hemoglobinization (18 and 22% Hb S3a, respectively), similar to the results obtained with antisense fte/S3a. As observed by us previously (24), transient transfection with CHOP alone without fte/S3a overexpression enhanced hemoglobinization significantly to 53 and 62% Hb S3a, respectively (Fig. 6, +/CHOP). Transfection with antisense fte/S3a alone (Fig. 6, +/AS FTE) had no significant effect.

**DISCUSSION**

Our results demonstrate a novel interaction between the transcription factor CHOP, a C/EBP family member, and FTE/S3a, a ribosomal protein. This interaction appears to play a role in modulating erythroid differentiation. We speculate that the interaction of CHOP with one or more other proteins (especially C/EBPβ, its principle binding partner among the C/EBP family members) plays a positive role in enhancing erythroid differentiation and that its interaction with FTE/S3a interferes with this. However, it is also possible that FTE/S3a not bound to CHOP inhibits erythroid differentiation by some other mechanism, and that the increased CHOP expression seen with differentiation binds to FTE/S3a and blocks this action.

**FIG. 6.** FTE/S3a and CHOP have opposing effects on erythroid differentiation. Cells were cultured at a density of 1 × 10⁶ cells in DMEM, 10% FCS and 5% CO₂ overnight. The culture medium was changed to α-MEM/10% FCS. Cells were transfected to the 24-well plates (0.5 ml/well) at the same cell density and induced by addition of 30 units of rhEpo/ml (white bars) or 0.8% Me2SO (black bars). Following 48 h incubation at 37 °C and 5% CO₂, hemoglobinized cells were quantified by benzidine staining. Values are the mean ± S.D. of six or more determinations.

**FIG. 5.** Absence of ribosomal protein S26 in anti-CHOP cytosolic or nuclear immunoprecipitate. SDS-PAGE and Western blot using anti-S26 antibody. Lane 1, purified total ribosomal protein; lane 2, Rauscher cell cytosolic lysate; lane 3, anti-CHOP cytosolic immunoprecipitate; lane 4, anti-CHOP nuclear immunoprecipitate. See “Experimental Procedures” and “Results.”
Interaction of CHOP and FTE/S3a

3OH end of 18 S RNA (48) and shown to be directly involved in formation of functional ribosomal sites using cross-linking, affinity labeling, and immunoelectron microscopy. FTE/S3a has been characterized as a ribosomal protein involved in interactions of the 40 S subunit with initiation factors eIF-2α and eIF-2γ (49, 50), initiator-tRNA (51), initiation factor eIF-3 (52–54), mRNA (52, 55–57), and of the 80 S ribosome with elongation factor EF-2 (58, 59), each of which has regulatory implications. Whether CHOP may take part in this process and influence ribosome maturation remains an unanswered question. The results presented here favor the interaction of CHOP with FTE/S3a as an isolated protein, either in the nucleus or the cytoplasm, and not with FTE/S3a as an integral part of preribosomal or ribosomal particles. However, it is still possible that CHOP also binds to 40 S or 80 S ribosomes. Additional experiments will be necessary to analyze possible binding sites and/or direct influences on ribosome activity.

In the adipocytic lineage, CHOP is related to inhibition of differentiation. The inhibition of adipocytic differentiation appears to depend on the ability of CHOP to antagonize the activities of C/EBP proteins at several levels (60). In our yeast two-hybrid screen we did not detect any C/EBP family members. However, this does not preclude the possibility of association of CHOP with C/EBP proteins in erythroid cells. Indeed, our previous work showed low levels of C/EBPα, -β, and -δ mRNA in Rauscher cells, with C/EBPβ expression increasing along with that of CHOP (25). Initially, it may seem paradoxical that CHOP enhances differentiation in erythroid cells and inhibits it in adipocytes. However, we have now shown that CHOP has a novel alternative non-C/EBP binding partner, FTE/S3a. Thus, the effects of CHOP on cell differentiation may also depend upon these alternative interactions and may be quite lineage-specific.

Acknowledgment—We thank Rosemary Panza for editorial expertise.

REFERENCES
1. Krantz, S. B. (1991) Blood 77, 419–434
2. Choi, H. S., Wojcikowski, D. M., and Sytkowski, A. J. (1987) J. Biol. Chem. 262, 2933–2936
3. Choi, H. S., Bailey, S. C., and Sytkowski, A. J. (1990) J. Biol. Chem. 265, 14121–14125
4. Papathanasiou, M., Fargnoli, J., and Holbrook, N. J. (1989) Proc. Natl. Acad. Sci. U. S. A. 86, 5583–5587
5. Bailey, S. C., Spangler, R., and Sytkowski, A. J. (1991) J. Biol. Chem. 266, 681–684
6. Bailey, S. C., Spangler, R., and Sytkowski, A. J. (1991) J. Biol. Chem. 266, 24121–24125
7. Spangler, R., and Sytkowski, A. J. (1992) Blood 79, 52–57
8. Patel, H. R., Choi, H. S., and Sytkowski, A. J. (1992) J. Biol. Chem. 267, 21300–21302
9. Wittthuhn, B. A., Quelle, F. W., Silvennoinen, O., Yi, T., Tang, B., Miura, O., and Igle, J. N. (1993) Cell 74, 227–236
10. Mayeux, P., Dusans-Fourt, I., Muller, O., Mauduit, P., Sabath, M., Druker, B., Vainchenker, W., Fischer, S., Lacombe, C., and Gisselbrecht, S. (1993) Eur. J. Biochem. 216, 821–828
11. Damen, J. E., Mui, A. L., Puil, L., Pawson, T., and Krystal, G. (1993) Blood 81, 3204–3210
12. Miura, O., Nakamura, N., Igle, J. N., and Aoki, N. (1994) J. Biol. Chem. 269, 614–620
13. Li, Y., Davis, K. L., and Sytkowski, A. J. (1996) J. Biol. Chem. 271, 27025–27030
14. Todokoro, K., Watson, R. J., Higo, H., Amanuma, H., Kuramochi, S., Yanagisawa, H., and Ikawa, Y. (1988) Proc. Natl. Acad. Sci. U. S. A. 85, 8900–8904
15. Tsai, S. F., Martin, D. I., Zen, L. I., D’Arenda, A. D., Wang, G. G., and Orkin, S. H. (1989) Nature 339, 446–451
16. Chiba, T., Ikawa, Y., and Todokoro, K. (1991) Nucleic Acids Res. 19, 3843–3848
17. Chen, Y. J., O’Hara, C., and Sytkowski, A. J. (1991) Blood 78, 991–996
18. Chen, Y. J., Spangler, R., Choi, H. S., and Sytkowski, A. J. (1991) J. Biol. Chem. 266, 2009–2012
19. Andrews, N. C., Erdjument-Bromage, H., Davidson, M. B., Tempst, P., and Orkin, S. H. (1993) Nature 362, 722–726
20. Fornace, A. J., Jr., Alam, I. Jr., and Holland, M. C. (1988) Proc. Natl. Acad. Sci. U. S. A. 85, 8800–8804
21. Fornace, A. J., Jr., Nebert, D. W., Hollander, M. C., Luethy, J. D., Papathanasiou, M., Fargnoli, J., and Holbrook, N. J. (1988) Mol. Cell. Biol. 9, 4196–4203
22. Luethy, J. D., Fargnoli, J., Park, J. S., Fornace, A. J., Jr., and Holbrook, N. J. (1990) J. Biol. Chem. 265, 16521–16526
Novel Interaction between the Transcription Factor CHOP (GADD153) and the Ribosomal Protein FTE/S3a Modulates Erythropoiesis
Kunyuan Cui, Margaret Coutts, Joachim Stahl and Arthur J. Sytkowski

J. Biol. Chem. 2000, 275:7591-7596.
doi: 10.1074/jbc.275.11.7591

Access the most updated version of this article at http://www.jbc.org/content/275/11/7591

Alerts:
- When this article is cited
- When a correction for this article is posted

Click here to choose from all of JBC’s e-mail alerts

This article cites 56 references, 28 of which can be accessed free at http://www.jbc.org/content/275/11/7591.full.html#ref-list-1