Silencing Mediator for Retinoid and Thyroid Hormone Receptors Interacts with Octamer Transcription Factor-1 and Acts as a Transcriptional Repressor

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Octamer transcription factor-1 (Oct-1) is a member of the POU (Pit-1, Oct-1, unc-86) family of transcription factors and is involved in the transcriptional regulation of a variety of gene expressions related to cell cycle regulation, development, and hormonal signals. It has been shown that Oct-1 acts not only as a transcriptional activator but also as a transcriptional repressor for certain genes. The mechanism of the repressive function of Oct-1 has not been well understood. Here we demonstrate by using the glutathione S-transferase pull-down assays and coimmunoprecipitation assays that the POU domain of Oct-1 directly interacts with a silencing mediator for retinoid and thyroid hormone receptors (SMRT). The interaction surfaces are located in the C-terminal region of SMRT, which are different from previously described silencing domains I and II or receptor interacting domains I and II. In transient transfection assays in COS1 cells, overexpression of SMRT attenuated the augmentation of Oct-1 transcriptional activity by OBF-1/OCA-B, activator for Oct-1. In pull-down assays, increasing amounts of SMRT could compete the binding of OCA-B to Oct-1 POU domain. The activity of Oct-1 could be determined by a regulated balance between SMRT and OCA-B. Furthermore, cotransfected unliganded thyroid hormone receptor enhanced the transactivation by Oct-1, and addition of 3,3’,5-tri-iodo-L-thyronine obliterated the stimulatory effects. Consequently, in the presence of cotransfected thyroid hormone receptor, the octamer response element acts as an element negatively regulated by 3,3’,5-tri-iodo-L-thyronine. The results suggest that the transcriptional activity of Oct-1 can be modulated by interaction through its POU domain by a silencing mediator SMRT resulting in the cross-talk between Oct-1 and nuclear receptors.

Octamer transcription factor-1 (Oct-1)§ activates the octamer motif containing gene promoters that are ubiquitously as well as tissue-specifically expressed genes such as histone H2B, the small nuclear RNA, and Ig (1–3). Oct-1 is a member of a family of transcription factors characterized by the presence of a bipartite DNA-binding domain (POU domain). The POU domain consists of two conserved regions, a POU-specific domain and a POU homeodomain (4, 5). The both subdomains have a helix-turn-helix motif, acting not only as a DNA-binding domain but also as a protein-protein interaction domain. A number of transcription factors have been identified to interact with the POU domains of Oct-1 such as TBP, TFIIB, HMG2, and Oct-binding factor 1 (OBF-1) also referred to as Oct-1-associated coactivator (OCA-B) (6–11). It has been shown that Oct-1 interacts with nuclear hormone receptors such as retinoid X receptor, thyroid hormone receptor (TR), and glucocorticoid receptor and influences their transcriptional activity (12–14).

Oct-1 possesses not only transactivation function but also repression function; von Willebrand factor promoter (15), pro-lactin gene promoter (16), or rGH promoter (14) was shown to be down-regulated by Oct-1. However, the mechanism of the bifunctional transcriptional activity of Oct-1 was not fully understood. OCAB/OBF-1 has been shown to be involved in the transcriptional activation by Oct-1, whereas the factor mediating the repressor function has not been isolated (15, 16).

Silencing mediator for retinoid and thyroid hormone receptors (SMRT) and the nuclear hormone receptor corepressor (N-CoR) were identified as interacting proteins for unliganded nuclear receptors that repress the basal transcriptional activity of target genes (17, 18). These corepressors associate with hinge domain of nuclear receptors. It has been demonstrated that SMRT and N-CoR directly interact with mSin3A and recruit the histone deacetylases (HDAC1/Rpd3) to form a multisubunit complex to modify the chromatin template of target genes (19). SMRT/N-CoR functions as an adapter to link unliganded receptor heterodimers with mSin3A and HDAC1 to create a hormone-sensitive multimeric repressor complex that lead the chromatin to be transcriptionally inactive (20). Interestingly, it has been recently shown that SMRT/N-CoR interacts with various nonreceptor transcription factors, such as oncprotein PLZF (21, 22), acute myeloid leukemia-associated protein ETO (23–25), activating protein 1 (AP-1), nuclear factor-aB (26), and TRL1 (27).

Furthermore, previous studies demonstrated that several homeodomain proteins such as Pit-1 and Pbx1 functionally interact with N-CoR/SMRT resulted in a formation of the factor 1; OCA-B, Oct-1-associated coactivator; PBS, phosphate-buffered saline; PAGE, polyacrylamide gel electrophoresis; PVDF, polyvinylidene difluoride; RID, receptor interacting domain; SD, silencing domain.
repressor complex (28, 29). These results suggested that NCore/SMRT possesses transcriptional repression activity for multiple transcriptional factors.

In this study, we have shown that SMRT physically interacts with POU domain of Oct-1 and is involved in transcriptional repression by Oct-1. Our results illustrate a novel mechanism by which Oct-1 regulates genes both negatively and positively. These results indicate that SMRT and N-CoR are involved in a wide array of biological processes and signaling pathways.

EXPERIMENTAL PROCEDURES

Plasmid Constructions—The Oct-1 expression vector pCDNA3HA-Oct-1 was a gift from Dr. H. Singh (30). The in vitro transcription and translation vector for Oct-1 pHis Oct-1 and OCA-B expression vector pRC OCA-B were from Dr. R. G. Roeder (11, 31). The OBF-1 expression vector pCDNA OBF-1 was a gift from Dr. P. Matthias (10). The pBS Oct-1 was a gift from Dr. W. Herr (2). The eukaryotic GST expression vector for Oct-1 p6His Oct-1 and OCA-B expression vector pCDM TR was a gift from Dr. R. M. Evans (17). To construct the bacterial expression vector for GST fusion proteins, polymerase chain reaction amplified fragments were cloned into EcoRI and XhoI restriction sites of pGEX-2T-1 vector (Amersham Pharmacia Biotech). Following oligonucleotides were used to amplify the full-length SMRT: forward primer, ‘gatg ATGAGGGC ATGGAGCC-3′; reverse primer, 5′-ctgag CTCG GTCGCGGAGG-3′; SMRT I: forward primer, 5′-gatg ATGAGG AGCTGGAGCC-3′; reverse primer, 5′-ctgag GTCGTAGCAGG-3′; SMRT II: forward primer, 5′-gaattc GAGAGCCTGAAGA-3′; reverse primer, 5′-ctgag GTCGCGGCTGCTGAAAGG-3′; SMRT III: forward primer, 5′-gaattc GCCCTTTCCGAGCC-3′; reverse primer, 5′-ctgag CGAGGGCTGGCTCTCAG-3′; and SMRT IV: forward primer, 5′-gatcCGTGTCGGAGAGTGT-3′. T R

Preparation of Nuclear Extracts, Coimmunoprecipitation, and Western Blotting—Nuclear extracts were prepared from COS1 cells grown in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal calf serum, 100 units/ml penicillin, and 0.25 mg/ml streptomycin at 37°C in 5% CO2. Transfection was done in COS1 cells using the standard calcium phosphate procedure. Briefly, cells were plated in 24-well plates 6 h prior to transfection. Luciferase reporter plasmids containing reporter plasmids 8xOcta-Luc and 8xOcta/mut-Luc were gifts from Dr. P. Matthias (34). The pGL3-IgH promoter luciferase reporter plasmid was a gift from Dr. R. G. Roeder (35).

Cell Culture and Transient Transfection and Reporter Assays—COS1 cells were grown in Dulbecco’s modified Eagle’s medium supplemented with 10% fetal calf serum, 100 units/ml penicillin, and 0.25 mg/ml streptomycin at 37°C in 5% CO2. Transfection was done in COS1 cells using the standard calcium phosphate procedure. Briefly, cells were plated in 24-well plates 6 h prior to transfection. Luciferase reporter (0.25 μg) was cotransfected with 1 μg of the indicated expression vectors. After 12 h of incubation, the medium on the cells was replaced with fresh medium. 10−7 M of TSA (see Fig. 4) or 10−7 M of T3 (see Fig. 5) was added to the medium when indicated. Cells were harvested after 24 h for reporter assays. Luciferase activity was determined by the PicaGene Luciferase Assay System (Toyo Inki, Tokyo) using Lumat L9501 (Berthold Japan K.K., Tokyo, Japan) and expressed as relative light units normalized to the amounts of protein. Each transfection was conducted in triplicate, and data represent the means ± S.D. of more than three individual experiments.

In Vitro Transcription and Translation—Coupled transcription and translation of Oct-1, SMRT were carried out using a T7 TnT translation/translation kit (Promega) according to the manufacturer’s instructions.

Expression of Recombinant Proteins—Overnight cultures of Escherichia coli BL21 carrying the recombinant GST fusion or GST control plasmid was diluted 100-fold, cultured for 5–6 h, and then induced with 0.1 mM isopropyl β-D-thiogalactopyranoside. After 3 h of induction, bacteria were collected and washed with PBS. Pellets were suspended in PBS containing 1% (v/v) Triton X-100 and sonicated. Debris was removed by centrifugation. The fusion protein or the GST control protein was bound to glutathione-Sepharose (Amersham Pharmacia Biotech) and extensively washed with PBS containing 1% (v/v) Triton X-100. Matrix-bound proteins were used for interaction experiments.

Interaction Experiments—In vitro translated 35S-labelled proteins (1–2 μl) were incubated for 20 min at room temperature with glutathione-Sepharose (10 μl) beads containing 2–5 μg of GST recombinant proteins in 250 μl of binding buffer (20 mM Tris-Cl, pH 7.8, 0.1% Triton X-100, 100 mM NaCl, 10% glycerol, 1 mM dithiothreitol, 1 mM EDTA, 1 mM phenylmethylsulfonyl fluoride, 1 mM leupeptin, 1 mM pepstatin, 2 mg/ml aprotinin). Ethidium bromide (50 μg/ml) was included in the binding reaction. After extensive washing with binding buffer, bound proteins were eluted in 25 μl of Laemmli sample buffer and resolved by SDS-polyacrylamide gel electrophoresis (PAGE 10%). The results of the in vitro reactions and the amount of 35S-labelled proteins bound by GST fusions were visualized and quantified using a Phosphor Imager (Fuji BAS 1500).

Preparation of Nuclear Extracts, Coimmunoprecipitation, and Western Blotting—COS1 cells were plated in 10-cm plates 6 h prior to transfection at a density of 2 × 105/plate. The amount of transfected DNA was kept constant (20 μg) by addition of appropriate amounts of the parental empty expression vector. Cells were transfected with 10 μg DNA.
to direct the interaction. The interacting surface of SMRT shown in Fig. 1, the third domain of SMRT (SMRT III) was required for the interaction. The COS1 cells were transfected with 10 $\mu$g of expression vectors for SMRT along with Oct-1 or control vector using standard calcium-phosphate method. Nuclear cell extracts (50 $\mu$g) were immunoprecipitated (IP) with monoclonal antibody (Ab) directed against Oct-1 protein, separated by SDS-PAGE, transferred to a PVDF filter, and probed with polyclonal antibody directed against SMRT proteins.

of expression vectors for SMRT along with Oct-1 or control vector using standard calcium-phosphate method. 12 hours later, cells were washed with PBS and refed with Dulbecco’s modified Eagle’s medium containing 10% fetal calf serum and incubated for 24 h. Nuclear extracts were prepared according to the method previously described (36). Briefly, after washing two times with cold phosphate-buffered saline, cells were lysed by adding 5 ml of ice-cold SMRT buffer (0.32 m sucrose, 1 mM MgCl$_2$, 1% Triton X-100, 10 mM Tris-CI, pH 7.5, 1 mM phenylmethylsulfonyl fluoride, 1 $\mu$g/ml leupeptin, 1 $\mu$g/ml aprotinin) to the plates. After nuclear fractions were recovered by centrifugation at 4,000 rpm for 10 min, pellets were resuspended in 40 $\mu$l of buffer A (20 mM Tris-CI, pH 7.5, 1 mM MgCl$_2$, 400 mM NaCl, 1 mM EDTA, 1% phenylmethylsulfonyl fluoride, 1 $\mu$g/ml leupeptin, 1 $\mu$g/ml aprotinin, 0.5% Triton X-100), followed by a 30-min incubation at 4 °C. Nuclear extracts were spun down at 12,000 rpm for 5 min. Immunoprecipitations were then performed with the anti-Oct-1 antibody for 2 h at 4 °C on a rotator, followed by the addition of 40 $\mu$l of protein A-Sepharose for 1 h at 4 °C. Immunoprecipitates were washed three times with buffer A. The bound proteins were solubilized in SDS-PAGE sample buffer, resolved by SDS-polyacrylamide gel electrophoresis, and transferred to PVDF (Millipore Immobilon-P) at 100 mA for 1 h at 4 °C with a Mini-Transblot system (Bio-Rad). Sheets of PVDF containing transferred protein from entire gels were incubated first in TBS-T (10 mM Tris, pH 8.0, 150 mM NaCl, 0.05% Tween 20) containing 5% nonfat dry milk for 1–3 h to block nonspecific binding of antibody, followed by 2 h of incubation in primary antibody (anti-SMRT antibody). The sheets were then washed in TBS-T and incubated for 1 h with an appropriate horseradish peroxidase-conjugated secondary antibody diluted 1:2000 in TBS-T. After washing two times in TBS-T, bound antibody was detected with the ECLTM system (Amersham Pharmacia Biotech) according to the manufacturer’s protocols. PVDF blots were reprobed with protein from entire gels were incubated first in TBS-T (10 mM Tris, pH 8.0, 150 mM NaCl, 0.05% Tween 20) containing 5% nonfat dry milk for 1–3 h to block nonspecific binding of antibody, followed by 2 h of incubation in primary antibody (anti-SMRT antibody). The sheets were then washed in TBS-T and incubated for 1 h with an appropriate horseradish peroxidase-conjugated secondary antibody diluted 1:2000 in TBS-T. After washing two times in TBS-T, bound antibody was detected with the ECLTM system (Amersham Pharmacia Biotech) according to the manufacturer’s protocols. PVDF blots were reprobed with

Results

POU Domain of Oct-1 Interacts with SMRT—To examine the interaction between Oct-1 and SMRT, we used the matrix-bound fusion protein of glutathione-S-transferase with Oct-1 (GST-Oct-1) for in vitro pull-down assays. As shown in Fig. 1A, [35S]methionine-labeled in vitro translated SMRT interacted with GST-Oct-1 POU domain (lane 3) but not GST alone (lane 2), and GST-Oct-1 did not retain any of the in vitro-translated control luciferase protein (lane 7–10). The matrix-bound GST-POU-specific domain (lane 4) and GST-POU homeodomain (lane 5) retained considerable amounts of [35S]methionine-labeled SMRT. Because the binding mixture included ethidium bromide to destroy the nonspecific DNA-protein interaction, it was concluded that the association of SMRT to matrix-bound Oct-1 was not due to the presence of contaminating DNA. Reciprocal pull-down experiment was performed to confirm the interaction between Oct-1 and SMRT. We examined the specific domains in SMRT that interact with Oct-1. Series of deletion mutants of GST fusion proteins representing overlapping portions of SMRT (Fig. 1B) were expressed in bacteria, purified, and used to bind 35S-labeled full-length Oct-1. As shown in Fig. 1C, the third domain of SMRT (SMRT III) was required for the interaction. The interacting surface of SMRT with Oct-1 is distinct from the nuclear receptor interacting domains (RID) or silencing domains (SD-1 and SD-2), which are known to interact with mSin3. As control experiments, the nuclear receptor interactions with SMRT were also determined. [35S]Methionine-labeled TR was incubated with matrix-bound GST fusions of SMRT (lanes 8–12). In agreements with previous reports (17), significant associations of TR were detected with GST-SMRT-IV and GST-SMRT-III+IV, whereas no association with GST-SMRT-III was observed. To further confirm the interaction between Oct-1 and SMRT within the cells, coimmunoprecipitation experiments were performed using COS1 cells cotransfected with SMRT and/or Oct-1 expression vector. As shown in Fig. 2, SMRT was coimmunoprecipitated by anti-Oct-1-specific antibody in the presence of transfected Oct-1, suggesting the physical interaction between Oct-1 and

FIG. 2. In vivo association between SMRT and Oct-1 proteins. COS1 cells were transfected with 10 $\mu$g of expression vectors for SMRT along with Oct-1 or control vector using standard calcium-phosphate method. Nuclear cell extracts (50 $\mu$g) were immunoprecipitated (IP) with monoclonal antibody (Ab) directed against Oct-1 protein, separated by SDS-PAGE, transferred to a PVDF filter, and probed with polyclonal antibody directed against SMRT proteins.

FIG. 3. Effect of SMRT on transcriptional activity of Oct-1. Control vector or Oct-1 (0.1 $\mu$g) expression vector was cotransfected into COS1 cells with 0.25 $\mu$g of 8xOcta or 8xOcta-mutant luciferase reporter (A) or BCL1 IgH promoter or BCL1 luciferase reporter (B) and 0.1 $\mu$g of SMRT or OBF-1/OCA-B expression vector. Relative luciferase activities are presented after being normalized by the amounts of protein. Each transfection was conducted in triplicate, and data represent the means ± S.D. of more than three individual experiments.
SMRT within the cells. In the absence of transfected Oct-1, because of the limiting amounts of endogenous Oct-1, interaction with SMRT could not be detected in this assay.

**SMRT Represses the Transcriptional Activity of Oct-1**—To test the possible role of SMRT in Oct-1 transcriptional activity, we performed transient transfection experiments in COS1 cells. Full-length Oct-1 expression vectors or empty expression vectors were cotransfected with luciferase reporter plasmids containing eight copies of the Oct-1 response element (8xOcta) into COS1 cells. As shown in Fig. 3A, cotransfection of SMRT repressed the Oct-1-activated reporter activity by 50%. When OCA-B/OBF-1 was cotransfected, significant stimulation of the Oct-1-dependent transcription was observed in agreement with previous reports (10, 11). Even in the presence of cotransfected OBF-1/OCA-B, SMRT still repressed the transcriptional activation by Oct-1. Next we examined the effects of SMRT on a naturally occurring Oct-1 response element using Ig heavy-chain (BCL1 IgH) promoter in COS1 cells. As shown in Fig. 3B, coexpression of Oct-1 and OBF-1/OCA-B stimulated the BCL1 IgH promoter. The expression of SMRT inhibited the Oct-1 transcriptional activity on IgH promoter in either the presence or absence of OBF-1/OCA-B but had no effect on the control reporter. These results strongly suggested that SMRT could function as a corepressor for Oct-1-dependent transcription, indicating physiological relevance of the interaction of SMRT with Oct-1.

TSA Releases the Transcriptional Repression by Oct-1 through SMRT—SMRT has been shown to recruit histone deacetylases (HDAC1 and HDAC2) through direct interaction with mSin3. These histone deacetylases are thought to be involved in the trans-repression function of unliganded nuclear receptors. To clarify whether HDACs mediated the trans-repression by Oct-1 through SMRT, we next examined the effects of TSA, a specific HDAC inhibitor, on the transcriptional activity of Oct-1 (37). As shown in Fig. 4, treatment of COS1 cells with 10^{-7} M TSA abolished the trans-repression by cotransfected Oct-1 and SMRT. Because of the presence of endogenous Oct-1 and SMRT, TSA increased the promoter activity in some degree even in the absence of cotransfected Oct-1 and SMRT. In contrast, TSA had no effect on the control promoter. These results suggested that histone deacetylase system would be involved in the Oct-1-mediated transcriptional repression.

**T3-dependent Repression of Oct-1-responsive Element Containing Promoter**—Because SMRT interacts with TR and acts as a nuclear receptor corepressor, we assume that coexpression of TR may enhance the transcriptional activity of Oct-1 by sequestration of the limiting amount of SMRT from Oct-1. If this is the case, ligand-induced dissociation of SMRT from TR increases the amounts of SMRT that can be recruited to Oct-1, resulting in the transcriptional repression of Oct-1 element.
Therefore, we next examined the effects of overexpression of TR and addition of T3 on Oct-1 function. As shown in Fig. 5A, as we assumed, unliganded TR enhanced the transcriptional activation of 8xOcta by Oct-1 and addition of T3 obliterated the enhancement, indicating that the promoter regulated by Oct-1 might act as a negative T3-responsive promoter through the mechanism that involves SMRT. To further confirm the mechanism that unligand TR can sequester SMRT from Oct-1, we next performed the pull-down assay. As shown in Fig. 5B, unliganded TR decreased the amount of SMRT that interacted with Oct-1 POU domain. Addition of T3, which releases the SMRT from TR and increases the number of SMRT available for the interaction with Oct-1, restored the interaction between SMRT and Oct-1.

**SMRT Influences the Interaction of Oct-1 with OCA-B/OBF-1**—SMRT and OCA-B interacts with the same POU domain; therefore the balance of the amounts of SMRT and OCA-B may regulate the bifunctional transcriptional activity of Oct-1. To determine whether SMRT and OCA-B/OBF-1 bound to Oct-1 competitively, the effect of SMRT on the interaction between Oct-1 and OCA-B were analyzed using pull-down assays. As shown in Fig. 6, addition of increasing amounts of SMRT results in diminished binding of OCA-B to Oct-1. These data suggested that SMRT and OCA-B/OBF-1 actually compete for binding to Oct-1 POU domain.

**REFERENCES**

1. Fletcher, C., Heintz, N., and Roeder, R. G. (1987) *Cell* 51, 773–781
2. Sturm, R. A., Das, G., and Herr, W. (1988) *Genes Dev.* 2, 1582–1599
3. Murphy, S., Yoon, J. B., Gerster, T., and Roeder, R. G. (1992) *Mol. Cell. Biol.* 12, 3247–3261
4. Herr, W., Sturm, R. A., Clerc, R. G., Cororran, L. M., Baltimore, D., Sharp, P. A., Ingraham, H. A., Rosenfeld, M. G., Finney, M., and Ruvkun, G. (1988) *Genes Dev.* 2, 1513–1528
5. Sturm, R. A., and Herr, W. (1988) *Nature* 336, 601–604
6. Zwillinger, S., Annweiler, A., and Wirth, T. (1994) *Nucleic Acids Res.* 22, 1655–1662
7. Zwillinger, S., Konig, H., and Wirth, T. (1995) *EMBO J.* 14, 1198–1208
8. Nakehati, H., Nakehati, P., and Currie, R. A. (1995) *J. Biol. Chem.* 270, 19613–19623
9. Gostaiger, M., Georgiev, O., van Leeuwen, H., van der Vliet, P., and Schaffner, W. (1996) *EMBO J.* 15, 2781–2789
10. Strubin, M., Newell, J. W., and Matthias, P. (1995) *Cell* 80, 497–506
11. Luo, Y., and Roeder, R. G. (1995) *Mol. Cell. Biol.* 15, 4115–4124
12. Krotsh, E., Stremseth, P. E., and Poellinger, L. (1992) *Mol. Cell. Biol.* 12, 4960–4969
13. Chandran, U. R., Warren, B. S., Baumann, C. T., Hager, G. L., and DeFranco, D. B. (1999) *J. Biol. Chem.* 274, 3727–3738
14. Kikaiwa, T., Miyamoto, T., Ichikawa, K., Kaneko, A., Suzuki, H., Harayama, M., Nagaaiwa, T., Takada, T., Mori, J., Kamagai, M., and Hashizume, K. (1999) *J. Biol. Chem.* 274, 19103–19108
15. Schachtgen, J. L., Remac, J. E., Janel, N., Brys, R., Huybrechts, D., Meyer, M., and Kerbiriou-Nabias, D. (1998) *Blood* 92, 1247–1258
16. Subramaniam, N., Cairns, W., and Okret, S. (1998) *J. Biol. Chem.* 273, 25967–25974
17. Chen, J. D., and Evans, R. M. (1995) *Nature* 377, 454–457
18. Horlein, A. J., Naar, A. M., Heintzel, T., Torchia, J., Goss, B., Kurokawa, R., Yuan, A., Kamei, Y., Soderstrom, M., Glass, C. K., and Rosenfeld, M. G. (1995) *Nature* 377, 397–404
19. Heintzel, T., Lavinsky, R. M., Muller, T. M., Soderstrom, M., Lamberth, C. D., Torchia, J., Yang, W. M., Brard, G., Ngo, S. D., Davie, J. R., Seto, E., Eisenman, R. N., Rose, D. W., Glass, C. K., and Rosenfeld, M. G. (1997) *Nature* 387, 43–48
20. Nagy, L., Kao, H. Y., Chakravarti, D., Lin, R. J., Hassig, C. A., Ayer, D. E., Schreiber, S. L., and Evans, R. M. (1997) *Cell* 80, 373–380
21. Hong, S. H., David, G., Wong, C. W., Dejean, A., and Privalsky, M. L. (1997) Proc. Natl. Acad. Sci. U. S. A. 94, 9028–9033
22. Lin, R. J., Nagy, L., Inoue, S., Shao, W., Miller, W. H., Jr., and Evans, R. M. (1998) Nature 391, 811–814
23. Wang, J., Hoshino, T., Redner, R. L., Kajigaya, S., and Liu, J. M. (1998) Proc. Natl. Acad. Sci. U. S. A. 95, 10860–10865
24. Gelmetti, V., Zhang, J., Fanelli, M., Misucci, S., Pelicci, P. G., and Lazar, M. A. (1998) Mol. Cell. Biol. 18, 7185–7191
25. Lutterbach, B., Westendorf, J. J., Inggi, B., Patten, A., Moniwa, M., Davie, J. R., Huynh, K. D., Bardwell, V. J., Lavinsky, R. M., Rosenfeld, M. G., Glass, C., Seto, E., and Hiebert, S. W. (1998) Mol. Cell. Biol. 18, 7176–7184
26. Lee, S. K., Kim, J. H., Lee, Y. C., Cheong, J., and Lee, J. W. (2000) J. Biol. Chem. 275, 12470–12474
27. Guenther, M. G., Lane, W. S., Fischle, W., Verdin, E., Lazar, M. A., and Shiekhattar, R. (2000) Genes Dev. 14, 1048–1057
28. Xu, L., Lavinsky, R. M., Dasen, J. S., Flynn, S. E., Melnerney, E. M., Mullen, T. M., Heizel, T., Seto, D., Kersh, E., Kurokawa, R., Aggarwal, A. K., Rose, D. W., Glass, C. K., and Rosenfeld, M. G. (1998) Nature 395, 301–306
29. Asahara, H., Dutta, S., Kao, H. Y., Evans, R. M., and Montminy, M. (1999) Mol. Cell. Biol. 19, 8219–8225
30. Shah, P. C., Bertolino, E., and Singh, H. (1997) EMBO J. 16, 7105–7117
31. Inamoto, S., Segi, N., Pan, Z. Q., Kimura, M., and Roeder, R. G. (1997) J. Biol. Chem. 272, 29852–29858
32. Coenjaerts, F. E., van Oosterhout, J. A., and van der Vliet, P. C. (1994) EMBO J. 13, 5401–5409
33. Nakai, A., Sakurai, A., Bell, G. I., and DeGroot, L. J. (1988) Mol. Endocrinol. 2, 1087–1092
34. Sauter, P., and Matthias, P. (1998) Mol. Cell. Biol. 18, 7397–7409
35. Luo, Y., Ge, H., Stevens, S., Xiao, H., and Roeder, R. G. (1998) Mol. Cell. Biol. 18, 3803–3810
36. Miyamoto, T., Ichikawa, K., Hashizume, K., Nishii, Y., Takeda, T., Kobayashi, M., Suzuki, S., and Yamada, T. (1991) Endocrinology 129, 907–914
37. Yoshida, M., Kijima, M., Akita, M., and Beppu, T. (1990) J. Biol. Chem. 265, 17174–17179
38. Prefontaine, G. G., Walthier, R., Giffin, W., Lemieux, M. E., Pope, L., and Hache, R. J. (1999) J. Biol. Chem. 274, 26713–26719
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