The promoter of the major histocompatibility class II gene DRA contains an octamer element (ATTTGCAT) that is required for efficient DRA expression in B cells. Several DNA-binding proteins are known to bind this sequence. The best characterized are the B cell-specific OTF-2 and the ubiquitous OTF-1. This report directly demonstrates that OTF-2 but not OTF-1 regulates the DRA gene. In vitro transcription analysis using protein fractions enriched for the octamer-binding protein OTF-2 demonstrate a positive functional role for OTF-2 in DRA gene transcription. In contrast, OTF-1-enriched protein fractions did not affect DRA gene transcription although it functionally enhanced the transcription of another gene. Recombinant OTF-2 protein produced by in vitro transcription/translation could also enhance DRA gene transcription in vitro. In vivo transient transfection studies utilizing an OTF-2 expression vector resulted in similar findings: that OTF-2 protein enhanced DRA gene transcription, and that this effect requires an intact octamer element. Together these results constitute the first direct evidence of a positive role for the lymphoid-specific octamer-binding factor in DRA gene transcription.

Major histocompatibility (MHC) class II proteins control the level of the immune response by functioning as ligands for the activation of antigen-specific T lymphocytes (1, 2). In addition, class II proteins transmit signals which activate B lymphocytes (3). MHC class II protein expression is limited to a few cell types, most notably on B cells, and is subject to transcriptional gene regulation. The transcriptional regulation of class II expression is mainly mediated by elements in their promoters, presumably via proteins that bind to these elements. Several cis-acting elements critical for MHC class II expression have been defined in B lymphocytes and other cell types including the W, X, and Y elements (4). Furthermore, the class II DRA promoter contains an octamer element, ATTTGCAT, that is required for its efficient expression in B cells (5, 6) (see Fig. 1A). This octamer element is identical to the octamer elements important for the expression of a variety of other genes including the immunoglobulin (Ig) light and heavy chains (7, 8), the human histone H2B (9), and the U1, U2, and U4 snRNA genes (10). The octamer element has therefore been determined to be a functional element for genes expressed in restricted cell types as well as for genes expressed ubiquitously.

Nuclear proteins have been defined that bind specifically to the octamer element. These include both the ubiquitous protein OTF-1 (also referred to as oct-1, NF-A1, OBPI00, NFIII, and oct-B3) (11–17) and OTF-2, classically referred to as OTF-1's ability to activate transcription from these two promoters has been suggested to be due to the restricted cell type expression of the Ig gene (16, 18, 19) and the MHC class II gene, DRA (5). In fact, it has been shown that purified OTF-2 can activate transcription of Ig heavy and light chain promoters in vitro (19, 28, 29). It was also demonstrated that OTF-1 could activate transcription of these same Ig promoters in vitro, but less efficiently (28, 29). The difference in OTF-2's versus OTF-1's ability to activate transcription from these two promoters has been suggested to be due to the requirement for an additional B-cell-specific component (29, 30) or due to quantitative differences in the amount of octamer-binding protein present (28). The context of the octamer element within the promoter may also be important (28). It has been hypothesized that the B cell-specific octamer-binding protein, OTF-2, is required for MHC class II DRA promoter function in B cells, but this has yet to be shown directly.

In this report, we demonstrate a functional role for the octamer-binding protein OTF-2 in MHC class II gene expression using both in vitro transcription and in vivo transient
transfection systems. This is similar to the positive functional role for OTF-2 observed in Ig gene transcription (19, 28, 29).

In contrast to the Ig system, however, OTF-1 appears not to play a role in transcription from the MHC class II DNA promoter in a B cell system, whereas it can activate transcription of the testis H2A histone (TH2A) gene in our system. These findings underscore that even though there are several similarities between the Ig and the DNA genes, including their expression in B lymphocytes, there are significant differences in their transcriptional regulation. The differences observed may be due to the context of the octamer element in the promoters of these genes. The octamer element upstream of a TATA box seems sufficient to confer B cell-specific promoter activity of Ig genes (31, 32), whereas MHC class II genes require additional promoter elements for expression (33, 34).

**MATERIALS AND METHODS**

**Plasmids, Probes, and Competitors—**The construction of plasmids pWToct and pMUToct (Fig. 1A) have been described in detail (5). They contain wild type DNA X and Y elements and either a wild type or mutant octamer element placed upstream of the bacterial chloramphenicol acetyltransferase (CAT) gene.

The human OTF-2 cDNA was isolated by polymerase chain reaction-based cloning from a human B lymphoblastoid cell line, Raji, using the DNA sequences described above (26). Firststrand DNA cDNA was obtained by polyadenylated RNA, size fractioned by avian myeloblastosis virus reverse transcriptase (Life Sciences, St. Petersburg, FL) from 2 μl of oligo(dT) (Boehringer Mannheim Biochemicals, Indianapolis, IN; 10-mer) primed amplification by polymerase chain reaction in a DNA thermal cycler.

mRNA isolated by batch elution (35) from log phase Raji cells. Reverse transcription and PCR amplification (Perkin-Elmer Cetus Corp., Emeryville, CA) using 50 PM each kinased phosphorylated SmaI-digested pGEM3Z (Promega, Madison, WI). For transcription, 20 μg of plasmid linearized with either BamHI or EcoRI (3 pg), 50 units of SP6 or T7 polymerase, and Hz0 was allowed to proceed for 30 min at room temperature in a 20-p1 reaction mixture. DNA binding reactions were performed essentially as described (15). DNA binding reactions were performed essentially as described (15). DNA binding was allowed to proceed for 30 min at room temperature in 2 μl volume containing either 3 μg of nuclear extract or 2 μl of in vitro transcription/translated protein, 20,000-50,000 cpm of radiolabeled probe, 1 μg of poly(dI-dC)-poly(dI-dC) (Pharmacia LKB, Piscataway, NJ) in a buffer containing 20 mM NaCl, 0.1 mM EDTA, pH 7.5.

DNA binding reactions were performed essentially as described (15). DNA binding was allowed to proceed for 30 min at room temperature in 2 μl volume containing either 3 μg of nuclear extract or 2 μl of in vitro transcribed/translated protein, 20,000-50,000 cpm of radiolabeled probe, 1 μg of poly(dI-dC)-poly(dI-dC) (Pharmacia LKB, Piscataway, NJ) in a buffer containing 20 mM NaCl, 0.1 mM EDTA, pH 7.5.

DNA binding reactions were performed essentially as described (15). DNA binding was allowed to proceed for 30 min at room temperature in 2 μl volume containing either 3 μg of nuclear extract or 2 μl of in vitro transcribed/translated protein, 20,000-50,000 cpm of radiolabeled probe, 1 μg of poly(dI-dC)-poly(dI-dC) (Pharmacia LKB, Piscataway, NJ) in a buffer containing 20 mM NaCl, 0.1 mM EDTA, pH 7.5.
OTF-2 Regulates DRA Gene Transcription

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**RESULTS**

**OBPs from B Cells but Not from HeLa Cells Can Partially Reconstitute DRA Gene Transcription in Vitro**—To assess whether OBPs from B cells which contain both OTF-1 and OTF-2 or OBPs from HeLa cells which only contain OTF-1 could reconstitute OTF-depleted DRA gene transcription in vitro, we compared the effects of OBPs enriched from these two cell lines on DRA transcription in an in vitro run-off transcription assay. A plasmid containing the basal DRA promoter upstream of the bacterial chloramphenicol acetyltransferase gene was used as a template for transcription (Fig. 1A). A schematic depiction of the strategy used for the transcription experiments is shown in Fig. 2A. OBPs were enriched by eluting HeLa or B cell octamer-binding proteins from an octamer element DNA affinity column. In Fig. 2B, a gel mobility shift assay (15) was used to test for the presence and specificity of octamer-binding factors in the material eluted from the column. Shifted complexes corresponding to OTF-1 and OTF-2 were identified based on published profiles from our and other laboratories (6, 19, 28, 33). *Lanes 1* and *2* are whole nuclear extract from Namalwa B cells and HeLa cells, respectively. Both cells contain OTF-1 protein (upper arrow), but only the B cell line contains OTF-2 protein (lower arrow). Namalwa B cell OBPs eluted off the octamer element affinity matrix contain both OTF-1 and OTF-2 (lane 3) which are specifically competed with an excess of octamer competitor (lane 4), but not with the same amount of a mutant octamer competitor (lane 5). HeLa cell OBPs eluted off the octamer element affinity matrix contain OTF-1 (lane 6) and another complex, which can be specifically competed with an excess of octamer element competitor (lane 7), but not with mutant octamer competitor (lane 8). These OBP-enriched extracts were used as a source of OBP proteins to reconstitute DRA gene transcription in a B cell nuclear extract depleted of octamer-binding proteins. The extract was depleted of OBPs by passage over an octamer element affinity matrix, and the depletion of octamer-binding proteins confirmed by gel mobility shift assay (data not shown). DRA in vitro transcription using nondepleted B cell extracts as a source of transcription factors resulted in the correct 595-nucleotide run-off transcript (left arrow) (Fig. 2C, lanes 1 and 4), whereas octamer-depleted B cell extracts produced no DRA transcription (lanes 2 and 5). It is critical that any differences in OTF-1 and OTF-2 activity would not be due to different amounts of these proteins used for reconstitution, therefore equivalent binding activity was determined by titration of these OBP-enriched fractions in a gel mobility shift assay (data not shown). B cell and HeLa cell OBPs containing similar amounts of OTF-1 binding activity were added to the depleted transcription reaction (see Fig. 2A). B cell OBPs could partially reconstitute transcription (lane 3), whereas HeLa cell OBPs could not (lane 6). The partial reconstitution with B cell OBPs averaged 35% as quantitated relative to the level of internal 494-nucleotide standard (arrowhead) in three separate experiments as determined by densitometric scanning of autoradiographs. When the same type of experiment was done using a plasmid containing a mutated octamer element as a transcription template, no effect of B cell OBPs was seen (data not shown). As a control to demonstrate that the HeLa cell OBPs were functional, we determined that HeLa OBPs could reconstitute octamer-depleted transcription from the TH2A elements and either a wild type or mutant octamer element upstream of the bacterial CAT gene. The TH2A-CAT plasmid contains 248 bp of the rat testis H2A histone promoter upstream of the CAT gene (42) and was kindly provided by Dr. Chi-Bon Chue (University of North Carolina, Chapel Hill, NC). The adenovirus major late promoter-CAT plasmid contains 170 bp of the adenovirus major late promoter upstream of the CAT gene (43), and was kindly provided by Dr. Al Baldwin (University of North Carolina, Chapel Hill, NC). Template DNA was linearized with NcoI, and precipitated prior to use in the reactions. The reaction consisted of template DNA (1.5 μg), 0.4 mM each ATP, UTP, and CTP, 1 mM creatine phosphate, 0.14 mM EDTA, 1 μl of [α-32P]GTP, and 15 μl of extract in a final volume of 25 μl. Transcription was allowed to proceed for 60 min at 30 °C. The reaction was stopped by adding 275 μl of stop solution (8 M urea, 0.5% SDS, 10 mM Tris-HCl, pH 8.0, 10 mM EDTA) plus 1000 cpm of an internal standard radiolabeled 494-nucleotide SmaI transcript to detect differences in recovery as previously published (39). After extraction with phenol:chloroform:isoamyl alcohol (20:20:1), the phenol layer was back-extracted with 165 μl of urea extraction buffer (7 M urea, 350 mM NaCl, 10 mM Tris, pH 8.0, 10 mM EDTA, 1% SDS) plus 25 μg of RNA carrier. The supernatants were combined and re-extracted with phenol:chloroform:isoamyl alcohol and precipitated with 95% ethanol. After washing in 70% ethanol, the RNA was heated at 90 °C for 2 min and electrophoresed on a 4% polyacrylamide gel containing 7 M urea. The gel was then autoradiographed.

**FIG. 1.** A, schematic representation of wild type and mutant MHC class II DRA reporter plasmids. pWToct contains 109 bp of the DRA promoter placed upstream of the bacterial chloramphenicol acetyltransferase reporter gene. A 66-bp oligonucleotide coding for the DRA X, Y, and octamer elements was cloned into the 5'Δ3'-CAT plasmid. pMUToct is identical to pWToct except mutant octamer sequence replaces wild type octamer sequence. B, schematic representation of OTF-2 expression plasmid, pCMV5-OTF-2. Human OTF-2 cDNA was cloned into the compatible cloning site of the plasmid pCMV5. pCMV5 contains the human cytomegalovirus early promoter, a multiple cloning site linker, and transcription termination and polyadenylation signals from human growth hormone.
OTF-2 Regulates DRA Gene Transcription

B cell nuclear extract was depleted of octamer-binding proteins by passage over an octamer DNA affinity column. This octamer-binding protein-depleted extract did not support DRA run-off transcription (see C). Octamer-binding proteins enriched from B cells (which contain OTF-2 and OTF-1) or from HeLa cells (which contain OTF-1) were assessed for their ability to reconstitute DRA gene transcription. B, gel mobility shift of octamer-binding proteins enriched from B cell and HeLa cell nuclear extracts. B cell (lane 1) or HeLa cell (lane 2) nuclear extracts, and octamer-binding proteins enriched from B cells (lanes 3–5) or octamer-binding proteins enriched from HeLa cells (lanes 6–8) were incubated with a radiolabeled octamer DNA probe in the presence of no competitor (lanes 1–3 and 6), wild type octamer competitor (lanes 4 and 7), or mutant octamer competitor (lanes 5 and 8). The resulting complexes were electrophoresed on a 6% nondenaturing Tris glycine polyacrylamide gel. C, functional analysis of B cell or HeLa cell OBPs in DRA in vitro transcription. DRA in vitro transcription using B cell nuclear extracts (lanes 1 and 4) or B cell nuclear extracts depleted of octamer-binding proteins (lanes 2, 3, 5, and 6). B cell OBPs were added to lane 3, HeLa cell OBPs were added to lane 6, and no OBPs were added to lanes 2 and 5. The correct DRA run-off transcription product (left arrow) is indicated. As a control for HeLa cell OBP

gene (Fig. 2C, lanes 7 and 8). These experiments demonstrate that B cell-derived OBPs can partially reconstitute octamer-depleted DRA gene transcription, whereas HeLa cell-derived OBPs cannot. Additionally, this effect is dependent on an intact octamer element.

Cloned OTF-2 Protein Positively Affects DRA Gene Transcription in Vitro—Octamer-binding proteins enriched from B cells were able to partially reconstitute octamer-depleted DRA transcription, in contrast to octamer-binding proteins enriched from HeLa cells. Since B cells make OTF-2 protein and HeLa cells do not, this suggests that OTF-2 was responsible for the reconstitution of octamer-depleted DRA gene transcription that we had observed. We tested this directly using cloned OTF-2 protein. For these experiments, a plasmid coding for human OTF-2 was utilized as a source of recombinant OTF-2 protein. OTF-2 protein was transcribed and translated in vitro from this plasmid in the sense orientation, resulting in the correct OTF-2 band as assessed by gel mobility shift assay (Fig. 3A, lane 2). As a comparison, nuclear extracts from the B cell line Namalwa (lane 1) gave the expected OTF-1 (upper arrow) and OTF-2 (lower arrow) shifted bands. The recombinant OTF-2 shifted band was specific since it could be competed with an excess of wild type octamer element (lane 3) but was not competed with the same molar amount of mutant octamer element (lane 4). Protein transcribed and translated from the same plasmid, but in the antisense orientation did not result in a specific OTF-2 protein (lane 5).

This antisense protein was used as a control for the cloned OTF-2 protein in the DRA transcription experiments. The extra bands in the sense and antisense recombinant OTF-2 lanes (lanes 2 and 5; arrowheads) are specific octamer-binding proteins produced by the rabbit reticulocyte lysate (compare lanes 3 and 4). HeLa cells do not contain OTF-2 protein (see Fig. 2B) and were used as a source of general transcription factors. We (5) have previously reported that HeLa extracts can only minimally support transcription from the DRA promoter. Cloned OTF-2 protein added to HeLa nuclear extracts allows for increased DRA gene transcription (Fig. 3B, lane 2, left arrow) over a control extract to which antisense OTF-2 has been added (lane 1). The arrowhead corresponds to the 494-nucleotide internal standard. When a plasmid containing a mutated octamer element is used as a template for transcription, OTF-2 protein in the sense or in the antisense direction has no effect (data not shown). As another control for specificity, we examined the effect of recombinant OTF-2 protein on transcription from the adenovirus major late promoter (right arrow), which does not contain an octamer element in its promoter. Recombinant OTF-2 protein in the sense or in the antisense direction had no effect on adenovirus major late promoter transcription (lanes 3 and 4). Thus, OTF-2 plays a positive role in DRA gene transcription. This effect is also dependent on an intact octamer element.

In Vivo Role of Recombinant OTF-2 Protein on DRA Gene Transcription—To further address the effect of OTF-2 protein expression on DRA gene transcription, an OTF-2 expression plasmid, pCMV5-OTF-2 (Fig. 1B), was co-transfected into the human T cell line HSB or into HeLa cells along with DRA-CAT reporter plasmids pWToct or pMUToct. HSB and HeLa cells lack endogenous OTF-2 protein and DRA expression is negligible (33). The reporter plasmids contain wild function, in vitro transcription from the TH2A CAT plasmid was analyzed using HeLa cell extracts depleted of octamer-binding proteins (lanes 7 and 8) without (lane 7) or with (lane 8) added HeLa cell OBPs. The correct TH2A run-off transcription product (right arrow) is indicated. The arrowhead indicates the 494-nucleotide internal standard included in all reactions.
type or mutant octamer elements in the context of 109 bp of
DRA promoter sequence (see Fig. 1A). As a control for the
OTF-2 expression plasmid, the same expression plasmid with-
out OTF-2 sequence was used (pCMV5). The effect of OTF-
2 protein on DRA promoter function was determined by
comparing the levels of CAT enzyme produced by pWToct in
the presence of the OTF-2 or the control CMV expression
plasmids. A representative result of the transient transfection
analysis using HSB cells (Fig. 4) is shown. DRA-CAT expres-
sion was induced 2.6-fold by CMV-OTF-2 in HSB cells (com-
pare lanes 2 and 1). We have repeated the experiments using
HSB cells 4 times and we have consistently observed a 2–3-
fold induction of CAT expression by OTF-2. To determine if
the effect of OTF-2 is dependent on the octamer element, a
reporter plasmid containing a mutant octamer element, pMUToct,
was used for similar experiments (Fig. 4, lanes 3 and 4). OTF-2 had little effect on CAT expression from the
pMUToct plasmid, demonstrating that OTF-2 induction of
DRA-CAT expression requires an intact octamer element.
This effect was also observed when the similar experiment
was performed using HeLa cells (data not shown).

**DISCUSSION**

Transcriptional regulation is an important mechanism for
the control of class II MHC gene expression. Several cis-
acting elements in the promoters of MHC class II genes have
been defined that mediate this transcriptional control. The
octamer element, ATTTGCGAT, is one of the cis elements
defined that is necessary for DRA gene transcription in B
cells, but not in non-B cells that express class II molecules
(6). This conclusion was reached because mutagenesis of the
octamer element in DRA greatly diminished gene expression
in B cell lines but not in DRα non-B cell lines. We had
previously demonstrated by gel mobility shift assays that two
proteins bind to the DRA octamer element, one B cell-specific
and the other not (33). These were thought to be the octamer-
binding proteins OTF-2 and OTF-1, respectively. Although
both OTF-2 and OTF-1 were capable of binding to the DRA
promoter in vitro, it was not known whether these octamer-
binding proteins functionally contributed to DRA gene tran-
scription. In this report, we demonstrated the role of OTF-2
protein in DRA gene transcription.

OTF-2 protein function was assessed in both a cell-free in
vitro transcription system and by in vivo transient transfe-
sion studies. Similar conclusions were obtained using both of
these approaches. Octamer-binding proteins enriched from B
cells which contain OTF-2 could partially restore octamer-
depleted DRA transcription in vitro, whereas those from HeLa
cells which do not contain OTF-2 protein had no effect (Fig.
2). Furthermore, recombinant human OTF-2 protein could
potentiate DRA gene transcription, but to a lesser extent than
enriched cellular OTF-2 (Fig. 3). This result is consistent with
data from other laboratories that suggests a role for an addi-
tional B cell factor in Ig gene regulation (28–30). Perhaps this
same or another B cell factor is also required for optimal
MHC DRA gene transcription. Our in vivo studies demonstrat-
ed that OTF-2 protein enhanced DRA gene transcription
2–3-fold, and that this effect requires an intact octamer ele-
ment (Fig. 4). Together these results demonstrate a role for
the OTF-2 protein in DRA gene transcription.

Although the effect of OTF-2 did not seem very dramatic,
contributing about 2–3-fold enhancement of DRA transcrip-
tion, it was very reproducible and the extent of OTF-2 con-
tribution was similar using two very different methods, i.e. in
vitro cell-free transcription and in vivo transient transfection.
This level of effect was not unexpected since protein binding
to the octamer element was thought to act in concert with
DNA-protein interactions at other known regulatory elements
of the DRA gene, including the W, X, and Y elements (4).
We have already previously demonstrated a positive func-
tional role for one Y element-binding protein YE BP, in DRA
gene transcription in B cells (39). It will be informative to
study the effects of all of the MHC class II promoter-binding
proteins individually as well as in combination with each
other to discern their roles in transcription.

In our studies, the OTF-1 enriched preparation did not

Fig. 3. A, gel mobility shift analysis of in vitro transcribed/trans-
lated human OTF-2. B cell nuclear extract (lane 1) or recombinant
OTF-2 protein obtained from OTF-2 translated in the sense ori-
entation (OTF-2-s; lanes 2–4) or in the antisense orientation (as; lane
5) were incubated with a radiolabeled octamer DNA probe in
the absence of competitor (lanes 1, 2, and 5), wild type octamer competitor
(lane 3), or mutant octamer competitor (lane 4). OTF-1 and OTF-2
shifted complexes are indicated. Arrowheads indicate octamer-bind-
ing proteins produced by the rabbit reticulocyte lysate. The protein-
DNA complexes were electrophoresed on a non-denaturing Tris
glycine polyacrylamide gel. B, functional analysis of recombinant
human OTF-2 protein in DRA in vitro transcription. In vitro
transcription from the DRA promoter (lanes 1 and 2) or the MLP
promoter (lanes 3 and 4) using HeLa nuclear extracts to supply
general transcription factors plus either recombinant OTF-2 protein
obtained from OTF-2 translated in the sense orientation (s; lanes 2
and 4) or in the antisense orientation (as; lanes 1 and 3). The correct
DRA run-off transcription product (left arrow) and the correct MLP
run-off product (right arrow) are indicated. The 494-nucleotide internal
standard (arrowhead) is included in all reactions.

Fig. 4. In vivo role of recombinant OTF-2 protein on DRA
gene transcription. Assay of CAT activity in extracts from HSB
(lanes 1–4) cells transiently co-transfected with either DRA-WToct
(lanes 1 and 2) or DRA-MUToct (lanes 3 and 4) reporter plasmids
and pCMV5 (lanes 1 and 3) or pCMV5-OTF-2 (lanes 2 and 4)
expression plasmids. Ethyl acetate-extracted material was separated
by thin layer chromatography and subjected to autoradiography.
proteins have, however, been shown to bind to DNA sequences in the promoters of their promoter regions (46). Whether or not the octamer element and the OTF-2 protein play a role in MHC class II DNA gene transcription, other human MHC class II genes do not contain an obvious octamer element in their promoters. Interestingly, the DNA homologues in other primates, including old and new world monkeys, also contain octamer elements in similar locations in their promoter regions (46). Whether this suggests that the octamer-binding protein OTF-2 only affects transcription of the DNA gene (and potentially its immune homologues) but not other MHC class II genes, or whether the octamer-binding protein can bind to other DNA sequences in the promoters of these genes remains to be determined. The octamer-binding proteins have, however, been shown to bind to DNA sequences other than the canonical ATTTGCAT octamer element including the TAATGARAT (R = purine) sequence upstream of the herpes simplex virus immediate-early genes (47), the AAGAATAAATTAGA site in the Aβ-globin gene promoter (48), and the heptamer element CTCATGA in the Ig heavy chain promoter (49, 50). On the other hand, if OTF-2 is only able to regulate transcription from the DNA MHC class II promoter, this could explain a possible mechanism for the higher levels of DR protein relative to DP or DQ proteins that are normally found on class II expressing cells (51).

In conclusion, this is the first demonstration that the lymphoid-specific octamer-binding protein, OTF-2, can positively regulate expression of the MHC class II gene, DNA. Using both affinity-enriched protein fractions and in vitro translated OTF-2 protein in an in vitro transcription system, in addition to an in vivo approach, our results established a role for OTF-2 in DNA transcription. Experiments are ongoing to determine the role of other proteins which may interact with OTF-2 in DNA gene expression.

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