The ubiquitous transcription factor Oct-1 stimulates basal transcription from the mouse mammary tumor virus (MMTV) promoter by binding to octamer-related sequences present in the proviral long terminal repeat. The mechanism of transcriptional activation by Oct-1 was investigated using in vitro transcription assays with a HeLa cell nuclear extract depleted of endogenous Oct-1. Oct-1-mediated transcriptional activation could be reconstituted by addition of bacterially expressed recombinant Oct-1 protein. The stimulatory effect of Oct-1 was observed only when the protein was present during formation of transcription preinitiation complexes and not when added to fully assembled complexes. Furthermore, assembled MMTV preinitiation complexes were resistant to inhibition by a competitor oligonucleotide containing MMTV octamer-related elements that could eliminate Oct-1-mediated stimulation when present during the assembly process. The time course of transcription complex assembly revealed that Oct-1 increases the number of templates on which functional transcription complexes form. Finally, experiments designed to exploit the sensitivity of discrete steps in transcription complex assembly to the anionic detergent Sarkosyl demonstrated that Oct-1 must be present during formation of an early intermediate in the assembly process.

Transcription of the proviral genes of mouse mammary tumor virus (MMTV) is induced by several classes of steroid hormones and is also modulated by negative regulatory elements (NREs) that repress basal activity of the promoter (for a review, see Ref. 1). The hormone response elements (HREs), which are specifically recognized by steroid hormone-receptor complexes, have been localized to MMTV long terminal repeat (LTR) sequences between about –200 and –80 (2–9). Several NREs have been characterized within LTR sequences, including a promoter-distal NRE (–427 to –363) (10–13) and a more promoter-proximal NRE, imbedded within the HRE, which by itself has little or no effect on transcription but which enhances repression mediated by the distal NRE (12, 14). These regulatory sequences modulate the transcriptional activity of the MMTV basal promoter, which contains sequences immediately 3’ of the initiation site that are recognized by a nuclear protein termed initiation site binding protein, a TATA element (centered near –30), a binding site for nuclear factor 1 (NF-1) (centered near –70), and two functional elements related to the octamer consensus (ATGCAAAT) between the TATA element and NF-1 binding site (15, 16).

The octamer motif has been shown to be an important regulatory sequence in many other promoters, and several proteins that are capable of specifically binding the octamer element have been identified. One protein, termed Oct-1 (17), is ubiquitously expressed, and a smaller protein, termed Oct-2 (18), is found predominantly in B lymphocytes. Although Oct-1 and Oct-2 recognize the same consensus sequence, they regulate different sets of genes (for a review, see Ref. 19). Oct-1 activates snRNA promoters as well as some mRNA promoters (e.g. histone H2B) (20, 21), and Oct-2 activates B cell-specific mRNA promoters (22). Oct-1 also acquires an ability to activate an immunoglobulin promoter in conjunction with a B cell-restricted protein (23, 24). Mutational studies have defined domains of octamer proteins that are important for transcriptional activation (21, 25), but the mechanism(s) by which these proteins activate transcription is poorly understood.

The octamer-related elements in the MMTV promoter have been shown to be important in both basal and steroid hormone-induced transcription in vivo (15, 26). Brüggemeier et al. (27) have also demonstrated the importance of MMTV octamer-related elements in progesterone receptor-induced transcription in vitro. In addition, we have shown that affinity-purified HeLa Oct-1, as well as bacterially expressed recombinant Oct-1 (rOct-1), recognizes MMTV octamer-related sequences and that addition of Oct-1 to an Oct-1-depleted HeLa nuclear extract selectively increases basal transcription from a template containing wild-type MMTV octamer sequences relative to a template containing mutations in octamer-related elements, demonstrating that Oct-1 acts as an important factor in basal transcription from the MMTV promoter (28).

In the present study, we have used in vitro transcription assays to show that Oct-1 participates at an early step in transcription preinitiation complex assembly on the MMTV promoter.

**EXPERIMENTAL PROCEDURES**

Depletion of Oct-1 from HeLa Nuclear Extract—Nuclear extracts were prepared by slight modifications (28) of previously described methods (29, 30). Nuclear extract in buffer D (20 mM HEPES (pH 7.9), 100 mM KCl, 0.2 mM EDTA, 20% glycerol, 3 mM DTT, 0.5 mM phenylmethylsulfonyl fluoride) was loaded on a phosphocellulose (Whatman P11) column equilibrated with the same buffer and eluted with buffer D containing a total of 1 M KCl. This phosphocellulose fraction was dialyzed against buffer D and loaded onto an MMTV octamer-specific DNA affinity column. Preparation of the DNA affinity column was as described previously (28). The flow-through fraction from the DNA affin-
ity column was used as Oct-1-depleted extract. Depletion was confirmed by gel electrophoresis mobility shift assay and Western blot analysis as described (28).

Preparation of Recombinant Oct-1—Oct-1 was expressed via the bacteriopeptide T7 promoter in Escherichia coli BL21(DE3) containing pLYS5 and purified as described previously (28). The final protein preparation was approximately 70–80% pure as judged by SDS-polyacrylamide gel electrophoresis and Coomassie Blue staining.

In Vitro Transcription—DNA templates were supercoiled plasmids pMBPT3 and pTLS(−59/)−38 (28). These templates contain MMTV LTR sequences from −109 to +14 linked to T-free cassettes that allow synthesis of RNA transcripts in the absence of added UTP. Both plasmids contain point mutations at +8 and +11 that maintain the T-free nature of the transcribed sequence, and pTLS(−59/)−38 also contains linker scanning mutations in MMTV octamer-related elements between −59 and −38 that eliminate Oct-1 binding and decrease transcription in vivo (15, 31) and in vitro (28).

For in vitro transcription assays, HeLa nuclear extract or Oct-1-depleted extract (60 μg of protein) was incubated for 30 min on ice in a total volume of 25 μl containing 20 μM HEPES (pH 7.9), 1 μM EDTA, 12.5 μM MgCl2, 20% glycerol, 100 mM KCl, and 2 mM DTT. This incubation with DTT was empirically shown to prevent loss of transcription activity upon storage of nuclear extracts at −80 °C. DNA templates (0.1 pmol each) and diethylpyrocarbonate-treated distilled water were added to a final volume of 44 μl, and the mixture was incubated at 30 °C for 60 min (unless stated otherwise in the figure legends) to allow assembly of transcription complexes. RNA synthesis was initiated by addition of 6 μl of U-free NTP mix (6 mM ATP, 6 mM GTP, 50 μM CTP, and 10 μC of 32P-CTP). Sarkosyl was added to the transcription reactions as indicated in the figure legends to inhibit transcription complex assembly and limit transcription reactions to a single round (32, 33). RNA synthesis was terminated after 30 min at 30 °C by addition of 350 μl of a solution containing 50 mM Tris-HCl (pH 7.5), 1% SDS, 5 mM EDTA, and 25 μg/ml RNAse. The mixture was extracted with phenol-chloroform, and the RNA was precipitated with ethanol in the presence of 0.3 mg sodium acetate. Transcripts were fractionated by electrophoresis on an 8% polyacrylamide gel containing 7 M urea and were visualized by autoradiography. Quantitation was performed with a Fujix BAS 2000 PhosphorImager (Fuji) and was corrected for background in each lane. For the reactions with Oct-1-depleted extracts, rOct-1 was added as described in the figure legends.

The sequence of the random oligonucleotide used as a control for the experiment in Fig. 2 was 5′-GATCCAGTCTGATCAGACTG-3′.

RESULTS

Oct-1 Functions during Preinitiation Complex Assembly—To study the effects of Oct-1 on MMTV promoter activity, we have developed an Oct-1-responsive in vitro transcription system based on HeLa cell nuclear extract depleted of endogenous Oct-1 (28). Our transcription assay is generally performed in two stages: template DNA is incubated with nuclear extract proteins to allow assembly of transcription preinitiation complexes, and then appropriate NTPs (including 32P-CTP) are added to allow RNA synthesis. A low concentration of the anionic detergent Sarkosyl (0.02–0.025%) is added with (or within 2 min following) the NTPs to inhibit further transcription complex assembly and limit transcription to a single round (32, 33), and thus the number of transcripts synthesized is directly proportional to the number of functional preinitiation complexes present at the time of NTP addition. Transcription templates consist of supercoiled plasmids containing MMTV promoter sequences linked to synthetic T-free cassettes that allow RNA synthesis in the absence of added UTP (28). T-free cassettes of different size allow transcription from different templates to be compared in a single assay.

We have assessed transcription from two MMTV promoter-containing templates. One template (pMBPT3) contains wild-type MMTV sequences from −109 to +14 (with the exception of two base changes introduced to maintain the T-free cassette (28)) and generates a U-free transcript of 172 nucleotides, while the second template (pTLS(−59/)−38) contains the same MMTV promoter region with mutations in octamer-related elements and generates a U-free transcript of 151 nucleotides (Fig. 1A). In nuclear extract containing endogenous levels of Oct-1, the wild-type template was transcribed 2- to 3-fold more efficiently than the template with the octamer mutations (28) (and see Fig. 2). After depletion of Oct-1 by specific DNA-affinity chromatography, the levels of transcription from the two templates were essentially identical (Fig. 1B, lane 1). Addition of purified rOct-1 to the depleted extract at the beginning of transcription complex assembly (time zero in Fig. 1B) resulted in a 14-fold stimulation of wild-type MMTV promoter activity, while transcription from the octamer-mutated template was stimulated only 4-fold (Fig. 1B, compare lanes 1 and 2). The observed stimulation of the mutated promoter was somewhat surprising. It is possible that the mutated promoter retains some affinity for Oct-1, or, perhaps more likely, that
Oct-1 is inefficiently recruited via interactions with other components of the transcription complex. Most importantly, in the transcription system reconstituted with rOct-1, the wild-type promoter was transcribed 3.5-fold more efficiently than the promoter was transcribed 3.5-fold more efficiently than the wild-type template. However, we show below that Oct-1 is fully functional at this concentration of Sarkosyl when the protein is added before preinitiation complex assembly (see Fig. 5). These experiments demonstrate that following transcription complex assembly, the MMTV promoter is resistant to stimulation by Oct-1.

MMTV Preinitiation Complexes Are Refractory to Inhibition by an Octamer Oligonucleotide—A complementary approach to determining the ability of assembled MMTV preinitiation complexes to be affected by Oct-1 was provided by an oligonucleotide competition assay. The oligonucleotide used contained MMTV sequences from –67 to –32 and thus encompassed both of the functional Oct-1 binding sites in the MMTV promoter (28, 31). This MMTV octamer oligonucleotide is specifically recognized by Oct-1 in a gel electrophoresis mobility shift assay (28) and can specifically inhibit binding of Oct-1 to the MMTV promoter in DNase I footprinting experiments (data not shown). Transcription from the wild-type MMTV promoter (pMBPT3) in a nuclear extract containing endogenous levels of Oct-1 was inhibited when the MMTV octamer oligonucleotide was present during transcription complex assembly (Fig. 2A, lanes 2–5), while an octamer oligonucleotide lacking an Oct-1 binding site did not significantly inhibit MMTV transcription (Fig. 2A, lane 6). Inhibition was dependent on the presence of octamer elements in the promoter, as the octamer oligonucleotide had no effect on transcription from the template containing mutations in this region (pTLS(–59/–38)) (Fig. 2A, lanes 2–5). Thus, in the presence of 50 pmol of the octamer oligonucleotide, transcription from the wild-type template decreased to a level similar to that observed from the octamer-mutated template (Fig. 2B). The inhibition of transcription by the MMTV octamer oligonucleotide most likely results from titration of Oct-1 from the template onto the competitor oligonucleotide.

This oligonucleotide-competing assay was used to assess the susceptibility of assembled MMTV preinitiation complexes to inhibition by the MMTV octamer oligonucleotide. Preinitiation complexes were assembled on MMTV templates by incubation in HeLa nuclear extract for 2 h. The MMTV oligonucleotide was then added, and the reaction mixture was incubated for an additional time prior to initiating transcription by the addition of NTPs and 0.025% Sarkosyl to limit transcription to a single round (Fig. 3, bottom). Under these conditions, wild-type MMTV promoter activity was not inhibited by the oligonucleotide (Fig. 3, lanes 3–5), and the level of transcription was similar to that observed in the absence of the octamer oligonucleotide (Fig. 3, lane 1). As expected, the presence of the oligonucleotide competitor during transcription complex assembly completely inhibited the stimulatory effect of Oct-1 (Fig. 3, lane 2). Resistance to the oligonucleotide competitor occurs despite our observation that purified rOct-1 dissociates from the MMTV promoter with a t1/2 of approximately 10 min in the presence of excess competitor oligonucleotide in a footprinting assay (data not shown). However, the effect of other components of the transcription complex on the dissociation rate of rOct-1 is difficult to assess.
Oct-1 Promotes MMTV Transcription Complex Assembly

Oct-1 Modulates the Efficiency of Assembly of Functional Preinitiation Complexes—The simplest interpretation of the experiments presented in Figs. 1–3 is that Oct-1 stimulation of MMTV transcription in vitro is mediated during the assembly of preinitiation complexes. To assess whether Oct-1 affects the kinetics of assembly of preinitiation complexes, the time course of assembly was determined (Fig. 4). Wild-type (pMBPT3) and octamer-mutated (pTLS(−59/−38)) MMTV promoter templates were incubated with Oct-1-depleted HeLa nuclear extract supplemented with rOct-1 (360 ng). NTPs and Sarkosyl (0.025% (w/v)) were then added and the incubations were continued for an additional 30 min. B, quantitation of transcription activity. Each point represents the average of two experiments like that described in A. Transcription signals were normalized to the signal from pMBPT3 in lane 4 and expressed as relative transcription. Curves are fit to a first order reaction.

Oct-1 Acts Early in Transcription Complex Assembly—Kinetic and inhibitor studies with the adenovirus major late promoter have allowed the process of transcription initiation to be divided into several discrete functional steps (32–34, see Fig. 5B). The first step is rate-limiting, can occur in the presence of 0.015–0.025% Sarkosyl, and leads to the formation of an intermediate complex that is not competent for transcription initiation. For the adenovirus major late promoter, this intermediate has been termed the template-committed complex since its formation results in preferential transcription relative to a second template added later in the assembly pathway. The conversion of the intermediate complex to a rapid-start complex, which is functionally equivalent to what we have termed the preinitiation complex in this report, occurs in a relatively rapid second step that is blocked by 0.015–0.025% Sarkosyl. Upon addition of NTPs, the rapid-start complex can rapidly initiate RNA synthesis. This third step is inhibited by concentrations of Sarkosyl greater than about 0.02% Sarkosyl (but resistant to 0.007%). We have not determined whether the intermediate complex on the MMTV promoter has the properties of a template-committed complex.

Fig. 4. Time course of preinitiation complex formation on the MMTV promoter, A, autoradiograph of U-free transcripts. Preinitiation complexes on pMBPT3 and pTLS(−59/−38) templates were allowed to form for the indicated times in the presence of Oct-1-depleted nuclear extract supplemented with rOct-1 (360 ng). NTPs and Sarkosyl (0.025% (w/v)) were then added and the incubations were continued for an additional 30 min. B, quantitation of transcription activity. Each point represents the average of two experiments like that described in A. Transcription signals were normalized to the signal from pMBPT3 in lane 4 and expressed as relative transcription. Curves are fit to a first order reaction.

Fig. 5. Role of Oct-1 in discrete steps in preinitiation complex assembly distinguished by Sarkosyl sensitivity, A, autoradiograph of U-free transcripts. The experimental design is shown below the autoradiograph. At time 0, pMBPT3 and pTLS(−59/−38) templates were incubated in Oct-1-depleted nuclear extract in the presence of a low (L) (0.007%, lanes 1 and 2) or medium (M) (0.02%, lanes 3–7) concentration of Sarkosyl. At 0.02% Sarkosyl, the conversion of the intermediate complex to the rapid-start complex is blocked (see B). After 60 min, the reactions were diluted 3-fold so that the Sarkosyl concentration either remained constant (L → L, lanes 1 and 2; M → M, lane 3) or decreased from 0.02% to 0.007% (M → L, lanes 4–7). Sarkosyl dilution allows the conversion of the intermediate complex to the rapid-start complex (see B). NTPs were added to initiate RNA synthesis either at the time of dilution (lanes 1–6) or 5 min after dilution (lane 7). In each reaction, the Sarkosyl concentration was adjusted to 0.025% 2 min after NTP addition to limit transcription to a single round. rOct-1 (360 ng) was present in some reactions (lanes 2, 3, and 5–7) and was added either at time 0 (lanes 2, 3, and 5) or at the time of Sarkosyl dilution (lanes 6 and 7). The time of Oct-1 addition is denoted with an asterisk at the top of each lane. B, steps in RNA polymerase II transcription initiation as defined by differential sensitivity to Sarkosyl. Formation of an intermediate complex is slow and insensitive to 0.015–0.025% Sarkosyl. Conversion to the rapid-start complex is fast and sensitive to 0.015–0.025% Sarkosyl. The third step, conversion to a stably initiated complex in the presence of NTPs, is also fast and sensitive to Sarkosyl concentrations greater than 0.1%.

0.1% The Sarkosyl block of the second step can be reversed by diluting the Sarkosyl concentration to 0.005% (33). Transcription complex assembly on the MMTV promoter appears to follow a similar pathway characterized by steps with comparable Sarkosyl sensitivity. In preliminary experiments, we determined that 0.02% Sarkosyl completely inhibited preinitiation complex assembly on the MMTV promoter. Furthermore, we determined that a 3-fold dilution (to 0.007%) reversed this inhibition and allowed formation of functional preinitiation (rapid start) complexes with kinetics much faster than that observed if the incubation at the higher Sarkosyl concentration, which presumably allowed formation of the intermediate complex, had not been performed (data not shown). Therefore, as with the adenovirus major late promoter, transcription complex assembly on the MMTV promoter can be divided into two functional steps, a relatively slow step that is resistant to 0.02% Sarkosyl and a faster step that is sensitive to 0.02% Sarkosyl (but resistant to 0.007%). We have not determined whether the intermediate complex on the MMTV promoter has the properties of a template-committed complex.
Oct-1 Promotes MMTV Transcription Complex Assembly

Described for the adenovirus major late promoter. For several promoters, including MMTV, formation of stable preinitiation complexes that are resistant to challenge by a second template requires a larger set of general transcription factors than the adenovirus major late promoter (35).

The ability to reversibly block the second step in MMTV preinitiation complex assembly made it possible to independently assess the role of Oct-1 in each of the two steps. In one extreme possibility, if Oct-1-mediated stimulation requires that Oct-1 be present during formation of the intermediate complex, then addition of Oct-1 following removal of a 0.02% Sarkosyl block by dilution to 0.007% should have no stimulatory effect. On the other hand, if Oct-1 participates only in the conversion of the intermediate complex to the rapid start complex, then addition of Oct-1 at the time of Sarkosyl dilution should have the same stimulatory effect as when it is present from the beginning of the assembly process.

These possibilities were tested as follows. Wild-type (pMBPT3) and octamer-mutated (pTLS(−59/−38)) MMTV templates were incubated in Oct-1-depleted HeLa nuclear extract containing 0.02% Sarkosyl (denoted as medium (M) concentration in Fig. 5). As described above, this concentration of Sarkosyl was empirically determined to prevent formation of the rapid-start complex on the MMTV promoter. After 60 min, the Sarkosyl was diluted to a concentration of 0.007% (denoted as low (L) concentration in Fig. 5), and NTPs were added. Two minutes after NTP addition, the Sarkosyl concentration was raised to 0.025% to limit transcription to a single round. rOct-1 was added to the reactions either at time zero or immediately following dilution. A control experiment in which the volume dilution maintained the Sarkosyl concentration at 0.02% (M* → M) effectively blocked all transcription, as expected, even when rOct-1 was present from time zero (Fig. 5A, lane 3). However, when the Sarkosyl block was reversed by dilution to 0.007% (M* → L), the number of functional preinitiation complexes formed (lane 5) was comparable to that in a control experiment in which the Sarkosyl concentration was maintained at 0.007% until after NTP addition (L* → L, lane 2). In addition, Oct-1-mediated stimulation of MMTV promoter activity was comparable in the M → L (compare lanes 4 and 5) and L → L (compare lanes 1 and 2) experiments; rOct-1-stimulated transcription from the wild-type template about 4-fold more efficiently than from the octamer-mutated template (17- to 20-fold from the wild-type and 5- to 6-fold from the mutant).

Significantly, addition of rOct-1 immediately after the Sarkosyl dilution (M → L*) had no effect on the level of MMTV transcription even after 5 min of incubation with rOct-1 before addition of NTPs (compare lanes 4–7). Thus, in our assays, the effect of Oct-1 on the MMTV promoter appears to be predominantly in the first functionally defined step (formation of the intermediate complex) rather than in the second step (formation of the rapid-start complex).

**DISCUSSION**

Oct-1 Stimulates Assembly of a Functional Preinitiation Complex—Four different experimental results are consistent with the idea that Oct-1 stimulates the assembly of functional transcription complexes on the MMTV promoter. First, Oct-1 was shown to increase MMTV promoter activity in vitro only if it was present while transcription complexes were being assembled; addition of Oct-1 after assembly had no effect on promoter activity (Fig. 1). Second, transcription complexes assembled in the presence of Oct-1 were refractory to inhibition by an MMTV octamer oligonucleotide competitor that binds Oct-1 and prevents Oct-1-mediated stimulation of promoter activity if present during transcription complex assembly (Figs. 2 and 3). Third, the kinetics of transcription complex assembly revealed that Oct-1 increases the number of templates that support transcription (Fig. 4). Finally, Sarkosyl inhibition experiments showed that Oct-1 must be present during an early step in transcription complex assembly (Fig. 5). The lymphoid-specific octamer-binding protein Oct-2 has also been shown to affect transcription complex assembly in vitro (36, 37).

There are at least two non-mutually exclusive mechanisms by which an Oct-1-mediated increase in the number of functional MMTV preinitiation complexes could occur. One possibility is that Oct-1 directs a more efficient assembly of transcription complexes that are functionally equivalent to those assembled in its absence, resulting in an increased number of templates on which such complexes form. Alternatively, transcription complexes assembled in the presence of Oct-1 could be qualitatively different and more likely to lead to productive RNA synthesis than those assembled in its absence. Such a difference could result directly from the presence of Oct-1 or indirectly via Oct-1-mediated recruitment of some additional factor. Both models are consistent with our order of addition, oligonucleotide competition, and Sarkosyl inhibition experiments that indicate that Oct-1 obligatorily enters the complex at an early stage. Preliminary analysis of transcription complex stability and kinetics of RNA synthesis has not revealed any differences between complexes assembled on wild-type and octamer-mutated templates. However, differences not detected by these assays are possible.

Interactions of Oct-1 with Other Transcription Proteins—If the mechanism of Oct-1-mediated stimulation involves increased efficiency of transcription complex assembly, then one or more components of the basal transcription apparatus may be targets of Oct-1 action. Furthermore, the observation that assembly of a stable complex on the MMTV promoter requires TFII D, TFII B, and RNA polymerase II (35) is consistent with the idea that transcription could be affected through interaction with any of these components. Indeed, Zwilling et al. (38) have demonstrated that the POU domains of Oct-1 and Oct-2 can interact with the TATA-binding protein component of TFII D.

Interactions between Oct-1 and other transcription proteins have also been demonstrated (39–43). Of particular significance to our work is the reported interaction between Oct-1 and NF-1 in binding closely spaced sites in the epithelial-specific enhancer of human papillomavirus (44). The MMTV octamer elements are also closely linked to an NF-1 binding site (33). These results suggest interactions between these two transcription factors on the MMTV promoter that can be disrupted by spacing changes. However, using purified recombinant proteins, we have not been able to demonstrate cooperative binding between Oct-1 and NF-1 on the MMTV promoter (data not shown), and interactions between these proteins, if present, appear not to be at the level of cooperative DNA binding.

Further analysis with a more defined transcription system may allow a more detailed determination of the mechanism of transcriptional activation by Oct-1 protein. Our studies have clearly demonstrated that for the activation we observe in vitro, Oct-1 must enter the assembling transcription complex at an early stage and that binding of Oct-1 results in an increase in the efficiency with which functional preinitiation complexes form on the template. It will be particularly interesting to

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2 C. Bral, J. Steinke, S. Kopytek, and D. O. Peterson, unpublished observations.
determine the mechanistic role of Oct-1 in steroid receptor-activated transcription and promoter repression mediated by the MMTV NREs.

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