Mechanism of Transcription Factor Recruitment by Acidic Activators*

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Many transcriptional activators are intrinsically unstructured yet display unique, defined conformations when bound to target proteins. Target-induced folding provides a mechanism by which activators could form specific interactions with an array of structurally unrelated target proteins. Evidence for such a binding mechanism has been reported previously in the context of the interaction between the cancer-related c-Myc protein and the TATA-binding protein, which can be modeled as a two-step process in which a rapidly forming, low affinity complex slowly converts to a more stable form, consistent with a coupled binding and folding reaction. To test the generality of the target-induced folding model, we investigated the binding of two widely studied acidic activators, Gal4 and VP16, to a set of target proteins, including TATA-binding protein and the Sw1 and Snf5 subunits of the Swi/Snf chromatin remodeling complex. Using surface plasmon resonance, we show that these activator-target combinations also display bi- phase kinetics suggesting two distinct steps. A fast initial binding phase that is inhibited by high ionic strength is followed by a slow phase that is favored by increased temperature. In all cases, overall affinity increases with temperature and, in most cases, with increased ionic strength. These results are consistent with a general mechanism for recruitment of transcriptional components to promoters by naturally occurring acidic activators, by which the initial contact is mediated predominantly through electrostatic interactions, whereas subsequent target-induced folding of the activator results in a stable complex.

Transcriptional activators function by binding specific DNA sequences in the promoter regions of target genes and subsequently recruiting components of the transcriptional machinery via their activation domains (ADs). Activation domains have to be able to interact with multiple structurally unrelated target proteins because eukaryotic genes require successive recruitment of different co-activator proteins (1), and different target genes utilize different repertoires of co-activators, even if they are regulated by the same activator (2, 3). The mechanism by which activator proteins make specific interactions with structurally distinct target proteins is, however, poorly understood.

Many activation domains are known to be intrinsically unstructured but are found in a folded conformation when bound to target proteins (for review, see Ref. 4). Whereas the structures of the initial and final states of some systems have been characterized, the determinants of complementarity, as well as the mechanism of the coupled folding and binding event, remain unknown. Support for the physiological significance of this transition has been provided by studies showing the unstructured nature of activation domains, even in the context of intact activator proteins (5, 6). In addition, mutations, causing substitutions of hydrophobic residues that might be expected to affect folding of the glucocorticoid receptor tau1 activation domain have been shown to cause changes in the activity of the intact receptor protein (7). We have previously reported that the activation domain from the cancer-related c-Myc protein interacts with the TATA-binding protein (TBP) in two steps, such that folding of the c-Myc activation domain is coupled to its interaction with the surface of TBP (8). Coupled binding and folding could explain how activators are able to interact specifically with a large set of structurally different targets in the transcriptional machinery and recruit them to promoters.

The c-Myc activation domain is unusual because it is divided in two protein segments that are interspersed with sequences that destabilize c-Myc by targeting its proteasome-mediated degradation (9, 10). Unlike the c-Myc activation domain, many activation domains consist of short protein segments that are rich in acidic amino acids. Two examples of acidic transcriptional activators that display extensive structural flexibility in their activation domains are Gal4 and VP16 (5, 11, 12). Gal4 regulates the genes required for galactose metabolism in yeast (13). The C-terminal activation domain of Gal4 also contains the interaction site for Gal80, which inhibits Gal4 activity under non-inducing conditions (14–17). VP16 is a co-activator involved in activation of immediate early viral genes in herpes simplex-infected cells, and the C-terminal domain has been identified as an activation domain (18, 19).

Acidic activators have been proposed to interact with target proteins by complementary electrostatic interactions mediated via their acidic residues, without adopting defined structures (20, 21). However, it has also been demonstrated that critical hydrophobic amino acids are essential for both target factor binding and the activating potential of acidic activation domains, whereas mutating individual acidic residues has little or no effect (7, 11, 22–26). These results underscore the importance of critical hydrophobic residues in the target interaction.
that could be coupled to the conformational changes referred to earlier. Indeed, the Gal4 and Gen4 acidic activation domains have been shown to adopt β-sheet conformation under acidic conditions in vitro, and mutational analysis indicates that this is a structural requirement for the activation function of Gal4 (11, 27). However, a relatively recent report indicates that whereas conformational changes may be important for interaction with Gal80, they may not be significant for either the activation activity of Gal4 or its interaction with TBP (21).

Both the Gal4 and VP16 activation domains interact with TBP with an affinity that correlates with their potency (26, 28–31). We have recently shown that they can also recruit the Swi/Snf chromatin remodeling complex to activated promoters via interactions with specific segments within the Swi1 and Snf5 subunits of the complex (32–34). These activator-interacting segments are not required for the overall integrity of the Swi/Snf complex, but when both are deleted, interaction with activators is severely reduced. This reduction correlates with a similar reduction in the activation potential of Gal4 in yeast strains containing the defective Swi/Snf complex (34). The mechanisms by which these segments of the Swi/Snf complex interact with activator proteins have not been studied.

Here we investigate whether the target-induced folding model for the mechanism of activator-target protein interactions that we previously suggested in the context of the interaction between c-Myc and TBP could be a general mechanism for the interaction of acidic transcriptional activators with a broader group of target proteins. Using surface plasmon resonance (SPR), we have focused on binding of the two highly acidic natural activators Gal4 and VP16 to the yeast target proteins TBP, Swi1, and Snf5. The results demonstrate that the target-induced folding model is applicable to a broader range of activator-target protein interactions.

**EXPERIMENTAL PROCEDURES**

**Plasmids and Proteins—**pRSET-Snf5, which encodes amino acids 1–334 of yeast Snf5 with an N-terminal His6 tag, has been described previously (34) (referred to hereafter as Snf5ABD). pRSET-Swi1 encodes amino acids 329–547 of yeast Swi1 with an N-terminal His6 tag and was constructed as described for pRSET-Snf5 (34) (Swi1ABD). pT7yD encodes a full length yeast TBP (35). pGST-VP16 encodes amino acids 413–490 of VP16 (VP16AD), deletion mutant pGST-VP16AD456 additionally lacks residues 456–490 (VP16sto456) (18), and pGST-Gal4 encodes amino acids 769–881 of Gal4 (Gal4AD) (32, 36). Each construct has an N-terminal GST tag.

All proteins in this study were produced by overexpression in *E. coli* strain BL21/DE3/pLys by induction with 1 mM isopropyl β-D-thiogalactoside. His-tagged Snf5ABD and Swi1ABD were expressed from fresh transformants in SOC medium at 37 °C and 25 °C, respectively. Cell pellets were resuspended in 20 mM Tris, pH 7.4, and 100 mM NaCl supplemented with Complete Protease Inhibitor Mixture. They were subsequently treated essentially as described previously (8). All proteins were frozen immediately following purification and subsequently stored at −70 °C until use. Protein concentrations were determined using Coomassie Protein Assay Reagent (Pierce). All proteins were at least 90–95% pure as judged by SDS-PAGE.

**SPR Analysis—**Kinetic experiments were performed on a BIAcore2000 instrument under control of the BIAcore control 3.1 software (BIACORE, Uppsala, Sweden). Amine-coupling kits and GST capture kits were purchased from BIACORE. The following is common to all experiments. Approximately 5000 resonance units (RU) of anti-GST antibodies were immobilized onto flow cell surfaces of CM4 sensor chips (BIACORE) using amine coupling chemistry. Prior to experiments, the binding capacity of immobilized antibodies was tested by capturing GST. Unless otherwise stated, experiments were carried out at 25 °C, using HBS supplemented with 10 mM MgCl2 as running buffer. GST and the GST-tagged activators were separately immobilized onto the sensor chip, after which 75 μM of target protein was injected at a rate of 20 μL/min. After each cycle, flow cell surfaces were regenerated by two pulses of regeneration buffer (10 mM glycine, pH 2.2) (BIACORE). All response data were collected at a rate of 1 Hz, manually aligned along the x-axis and y-axis, and processed by double referencing (i.e. subtraction of background binding to the affinity tag (GST), as well as adjustment for flow cell-specific bulk effects by subtraction of buffer injection). Curve fitting was performed using BIAevaluation 3.0.2 software (BIACORE) and a sequential two-step model as previously described (8).

Characterization of the Gal4AD-TBP, Gal4AD-Swi1ABD and VP16AD-Swi1ABD interactions was performed on flow cell 2, using flow cell 1 for the characterization of target proteins. The binding capacity of immobilized antibodies was tested by capturing VP16AD, with the following concentration of target proteins: 0.21, 0.43, 0.85, 1.7, and 3.4 μM Swi1ABD and 0.21, 0.41, 0.83, 1.7, and 3.3 μM TBP. Three consecutive injections of buffer control were run prior to injection of dilution series of target proteins, which were followed by a fourth buffer control injection. An average of three injections was used for double referencing. The running buffer was supplemented with 2% glycerol, and analyte samples were diluted to 2% glycerol prior to injection. Temperature experiments on Gal4AD-Swi1ABD were performed using the running buffer described above containing 300 mM NaCl.

Reproducibility was assessed as follows. Results obtained by repeating the 150 mM NaCl experiment at 25 °C on Gal4AD-TBP at different time points and on different sensor chips varied by <11% for the fast phase, 18% for the slow phase, and 27% for overall affinity. For duplicates on parallel flow cells in VP16AD-TBP experiments, results generally varied by <11% for the fast phase, 26% for the slow phase, and 26% for overall affinity.

**RESULTS**

**Acidic Activators Bind Target Proteins in Two Major Kinetic Steps—**We used SPR to monitor the interaction of target proteins with activation domains that were immobilized on the surface of a sensor chip. Using this technique, it is possible to measure the time-dependent association of target proteins in the buffer stream with immobilized activation domains as they flow over the chip surface. Furthermore, because the continuous flow of target protein solution can be exchanged for buffer alone following association, it is possible to measure the dissociation kinetics of complexes that have been formed on the chip surface. The plots (sensograms) are then subjected to regression analysis in order to extract the kinetic and equilibrium constants according to a selected binding model. To determine whether acidic activators bind to target proteins by a mechanism similar to that proposed for the c-Myc-TBP interaction, we measured the kinetics of VP16AD, the binding to TBP as well as protein segments containing the ABs of Swi1 and Snf5. Fig. 1 displays the characteristic bi-phasic time course of VP16AD interacting with all three target proteins. The mutant VP16AD456, which has impaired transcriptional activity (18, 37) and displays severely reduced affinity for Swi1 and Snf5 (33, 34), is included as a control. As expected, this mutant (Fig. 1, gray line) also produces a significantly reduced SPR response. A two-step model reproduces the experimental data well (Fig. 1, dashed line) and demonstrates that activator-
target complex formation is consistent with an initial fast step followed by a subsequent slower step.

**Increased Temperature Favors the Slow Phase but Generally Does Not Affect the Fast Initial Phase**—The kinetics of activator binding to a dilution series of target proteins was measured as a function of temperature. Fig. 2 provides an overview of the binding data by showing sensograms for the highest concentration of the respective target proteins interacting with each activator at the extremes of temperature tested. All activator-target combinations interact with bi-phasic kinetics under all conditions tested. Higher temperature (black line) significantly increases the rate of the slow association phase but does not markedly affect the dissociation rate or the fast association phase, resulting in an increased rate of complex formation.

To investigate the nature of the respective interaction phases, equilibrium affinity constants for initial and subsequent phases were obtained as a function of temperature and subjected to a van’t Hoff analysis. Increased temperature (black line) significantly increases the rate of the slow association phase but does not markedly affect the dissociation rate or the fast association phase, resulting in an increased rate of complex formation.

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Table 1

| Activator-target interaction | \(\Delta H_{\text{slow}}\) | \(\Delta H_{\text{fast}}\) | \(\Delta H_{\text{overall}}\) |
|-----------------------------|----------------|----------------|----------------|
| Gal4AD-TBP                  | 52 ± 18        | 1 ± 4          | 52 ± 16        |
| VP16AD-TBP                  | 39 ± 4         | -3 ± 8         | 35 ± 12        |
| Gal4AD-Swi1ABD              | 172 ± 53       | -53 ± 8        | 118 ± 45       |
| VP16AD-Swi1ABD              | 72 ± 14        | 16 ± 13        | 93 ± 9         |

\(a\) 300 mM NaCl.

**Increased Ionic Strength Inhibits the Fast Initial Phase but Favors the Subsequent Slow Phase**—The kinetics of activator binding to a dilution series of target proteins was measured as a function of NaCl concentration. An overview of the binding data is shown by the sensograms for the highest concentration of the respective target proteins interacting with each activator at the extremes of NaCl concentrations tested (Fig. 4). As shown for the effect of temperature (Fig. 2), the interaction kinetics of each of the activator-target combinations remains

**Table 1**

| van’t Hoff enthalpies calculated from data shown in Figs. 3 and 6 |
|---------------------------|----------------|----------------|
|                           | \(\Delta H_{\text{slow}}\) | \(\Delta H_{\text{fast}}\) | \(\Delta H_{\text{overall}}\) |
| Gal4AD-TBP                | 52 ± 18        | 1 ± 4          | 52 ± 16        |
| VP16AD-TBP                | 39 ± 4         | -3 ± 8         | 35 ± 12        |
| Gal4AD-Swi1ABD            | 172 ± 53       | -53 ± 8        | 118 ± 45       |
| VP16AD-Swi1ABD            | 72 ± 14        | 16 ± 13        | 93 ± 9         |

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bi-phasic under all the conditions studied. Increased ionic strength (gray line) significantly reduces the amplitude of the overall response without significantly affecting the relative contribution of the fast and slow association phases, as would be expected if the initial step was largely electrostatic in nature.

When plotted as the natural logarithms of the calculated affinity constants for each binding phase as a function of NaCl concentration, the data demonstrate that increasing ionic strength results in a reduction of the magnitude of the initial fast phase for all activator-target combinations (Fig. 5, A–D). Contrary to this inhibitory effect on the initial fast phase, increasing ionic strength favored the subsequent slow phase.

**Effect of Increased Ionic Strength and Temperature on the Overall Affinity of Acidic Activators for Target Proteins—**The different nature of electrostatic and hydrophobic interactions makes it difficult to predict the effect of increased ionic strength and temperature on the overall affinity of activator-target interactions. Fig. 6A shows that the overall affinities for all the interactions tested increase with increased temperature. The slopes of these plots again yield a positive van’t Hoff enthalpy for binding. The overall reaction is therefore also entropy-driven. The effect of increasing ionic strength on overall affinity (Fig. 6B) is more heterogeneous. Gal4AD displayed an increased overall affinity for both TBP and Swi1ABD with increasing ionic strength. VP16AD behaves differently when binding Swi1ABD and TBP. The VP16AD-Swi1ABD interaction is similar to the Gal4AD pattern with both targets, although in the case of VP16AD binding to Swi1ABD, the increase in overall affinity with increased ionic strength is subtle. However, the interaction of VP16AD with TBP is sensitive to increasing ionic strength and thus shows a trend opposite to that observed for the other interactions.

**DISCUSSION**

Here we have tested the generality of the target-induced folding model, using the activation domains of two naturally occurring acidic activators, Gal4 and VP16, which are frequently used as models in studies of activator function. In addition to the well-established binding partner TBP, two recently defined activator-binding domains within the Swi1 and...
Snf5 subunits of the Swi/Snf chromatin remodeling complex were also studied. The target-induced folding model that we proposed previously to account for the interaction between c-Myc and TBP entails three main requirements. First, the interaction between activator and target occurs in two main steps. Second, the initial step is rapid and dependent on electrostatic interactions between the proteins. Third, the second interaction step is slower and entropy-driven, suggesting the involvement of protein folding. All three of these conditions are fulfilled by all the interactions between the acidic activators and target proteins tested here. We therefore propose that the target-induced protein folding model is applicable to a broad range of activator-target interactions.

It has been suggested that activation domains of acidic activators, including VP16 and Gal4, function as “acid blobs” that recruit target proteins to promoters via low affinity, nonspecific electrostatic interactions (20, 21). Our results provide a more complete context for understanding the role played by electrostatic forces in interactions between acidic activation domains and the target factors they recruit. According to our model, electrostatic interactions dominate during the initial binding phase, but they generally have a subordinate role in the subsequent entropy-driven phase, where formation of the final complex depends on hydrophobic interactions. This is consistent with our observation that increased temperature, which increases the efficiency of the entropy-driven step, stabilizes formation of the final complex. Our model is consistent with genetic studies showing both the importance of overall acidity and the crucial role played by specific individual hydrophobic residues but not individual acidic residues in activation domain function (7, 22, 23, 30). We conclude that whereas electrostatic interactions might sometimes increase the rate of complex formation, the affinity of interactions between acidic activators and the factors they recruit depends on hydrophobic interactions that are likely to be associated with folding of the participating activation domains.

The target-induced folding model suggests that target proteins exert a dominant influence on the ultimate state of the activator-target complex, and thus one might expect to see characteristic binding patterns associated with at least some target proteins. Such a pattern was observed for TBP, for which all changes in experimental variables had essentially the same effects on the binding pattern of both VP16AD and Gal4AD (see Figs. 2 and 4). This suggests that the activators may have similar contacts on TBP, a notion that is supported by previous studies (30, 38). In light of these results, a picture emerges of a relatively predefined TBP scaffold, providing a common surface specialized in activator recognition.

Although all the interactions studied here conform to the two-step target-induced folding interaction model, differential contributions of electrostatic and hydrophobic interactions could be responsible for some interesting differences between the different activator-target interactions. One example is the different behavior of the overall affinities for the VP16AD-TBP and Gal4AD-TBP complexes in response to ionic strength. This behavior correlates with previous affinity data for TBP, measured using other techniques (29, 30). Unlike VP16AD, Gal4AD displays increased overall affinity for both target proteins in increased ionic strength, which suggests that Gal4AD may have some additional unfavorable electrostatic interactions to overcome upon complex formation, compared with VP16AD. Furthermore, the observation that Swi1AD, but not TBP, can support an increased overall affinity with both of these activators at high ionic strength helps to illuminate the individual influence of a target on activator binding despite some potentially unfavorable interactions. Interestingly, whereas the effect of ionic strength on overall affinity appears to depend on both activator and target protein, the magnitude of the effect of temperature appears to depend entirely on the target protein (see Fig. 6, A and B). Higher temperature has a stronger positive effect on the activator complexes with Swi1 than it does with TBP. These binding characteristics of Swi1 could be interpreted as the activator-binding surface being more hydrophobic in nature. Upon activator binding, repulsive electrostatic interactions dominate, compared with TBP. However, because there are multiple ways that both electrostatic and hydrophobic interactions could combine at the interaction interfaces producing the observed differences between activator-target complexes, it is difficult to predict a priori the nature of the interaction surfaces.

In summary, our results indicate that the acidic activator proteins Gal4 and VP16 share a common target protein binding mechanism that is consistent with binding-induced folding of the activator. We propose that, in general, the role of the abundant acidic residues of such activators is primarily to promote a rapid, rather unspecific, initial contact and that specificity relies on hydrophobic interactions that determine the stability of the final complex and the rate with which it forms. However, as shown here, the relative contribution of electrostatic and hydrophobic interactions to complex stability varies for particular activator-target combinations. The model we propose provides a framework that integrates the previously demonstrated importance of both acidic and bulky hydrophobic amino acid residues for target recruitment and the activation potential of acidic activators (11, 23, 25, 26, 30). These results suggest that target-induced folding may be a general mechanism by which transcriptional components are recruited to promoters by naturally occurring activators.

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REFERENCES

1. Cosma, M. P., Tanaka, T., and Nasmyth, K. (1999) Cell 97, 299–311
2. Gregory, P. D., Schmid, A., Zavari, M., Munsterkotter, M., and Horz, W. (1999) EMBO J. 18, 6407–6414
3. Gregory, P. D., Schmid, A., Zavari, M., Lui, L., Berger, S. L., and Horz, W. (1999) Mol. Cell 4, 495–505
4. Dyson, H. J., and Wright, P. E. (2002) Curr. Opin. Struct. Biol. 12, 54–60
5. Grossmann, J. G., Sharff, A. J., O’Hare, P., and Luisi, B. (2001) Biochemistry 40, 6267–6274
6. Ayed, A., Mulder, F. A. A., Yi, G. S., Lu, Y., Kay, L. E., and Arrowsmith, C. H. (2001) Nat. Struct. Biol. 8, 756–760
7. Almlof, T., Gustafsson, J. A., and Wright, A. P. H. (1997) Mol. Cell. Biol. 17, 934–945
8. Hermann, S., Berndt, K. D., and Wright, A. P. (2001) J. Biol. Chem. 276, 40127–40132
9. Flinn, E. M., Busch, C. M., and Wright, A. P. H. (1998) Mol. Cell. Biol. 18, 5961–5969
10. Flinn, E. M., Wallberg, A. E., Hermann, S., Grant, P. A., Workman, J. L., and Wright, A. P. (2002) J. Biol. Chem. 277, 23399–23406
11. Leuther, K. K., Salmeron, J. M., and Johnston, S. A. (1993) Cell 72, 575–585
12. Usugui, M., Nyangule, O., Lu, H., Levine, A. J., and Verdin, G. L. (1997) Science 277, 1310–1313
13. Johnston, M. (1987) Microbiol. Rev. 51, 458–476
14. Ma, J., and Ptasgne, H. (1997) Cell 85, 847–853
15. Ma, J., and Ptasgne, H. (1997) Cell 85, 137–142
16. Johnston, M., Fluck, J. S., and Pepton, T. (1994) Mol. Cell. Biol. 14, 3834–3841
17. Rohde, J. R., Trinh, J., and Sadowski, I. (2000) Mol. Cell. Biol. 20, 3880–3886
18. Triesenberg, S. J., Kingsbury, R. C., and Mcknight, S. L. (1988) Genes Dev. 2, 718–729
19. Preston, C. M., Frame, M. C., and Campbell, M. E. M. (1988) Cell 52, 425–434
20. Sigler, P. B. (1988) Nature 333, 210–212
21. Ansari, A. Z., Reece, R. J., and Ptasgne, H. (1998) Proc. Natl. Acad. Sci. U. S. A. 95, 13543–13548
22. Almlof, T., Wright, A. P. H., and Gustafsson, J. A. (1995) J. Biol. Chem. 270, 17538–17540
23. Cress, W. D., and Triesenberg, S. J. (1991) Science 251, 87–90
24. Lin, J. Y., Chen, J. D., Elenbaas, B., and Levine, A. J. (1994) Genes Dev. 8, 1235–1246
25. Regier, J. L., Shen, F., and Triesenberg, S. J. (1993) Proc. Natl. Acad. Sci. U. S. A. 90, 883–887
26. Sullivan, S. M., Horn, P. J., Olson, V. A., Koop, A. H., Niu, W., Ebrist, R. H., and Triesenberg, S. J. (1998) Nucleic Acids Res. 26, 4487–4496
27. Van Hory, M., Leuther, K. K., Kodakef, T., and Johnston, S. A. (1993) Cell 72, 587–594
28. Ingles, C. J., Shales, M., Cress, W. D., Triezenberg, S. J., and Greenblatt, J. (1991) *Nature* **351**, 588–590
29. Stringer, K. F., Ingles, C. J., and Greenblatt, J. (1990) *Nature* **345**, 783–786
30. Melcher, K., and Johnston, S. A. (1995) *Mol. Cell. Biol.* **15**, 2839–2848
31. Nedalkov, Y. A., and Triezenberg, S. J. (2004) *Arch. Biochem. Biophys.* **425**, 77–86
32. Neely, K. E., Hassan, A. H., Wallberg, A. E., Steger, D. J., Cairns, B. R., Wright, A. P. H., and Workman, J. L. (1999) *Mol. Cell* **4**, 649–655
33. Neely, K. E., Hassan, A. H., Brown, C. E., Howe, L., and Workman, J. L. (2002) *Mol. Cell. Biol.* **22**, 1615–1625
34. Prochasson, P., Neely, K. E., Hassan, A. H., Li, B., and Workman, J. L. (2003) *Mol. Cell* **12**, 983–990
35. Hoopes, B. C., Leblanc, J. F., and Hawley, D. K. (1992) *J. Biol. Chem.* **267**, 11539–11547
36. Carrozza, M. J., John, S., Sil, A. K., Hopper, J. E., and Workman, J. L. (2002) *J. Biol. Chem.* **277**, 24648–24652
37. Berger, S. L., Cress, W. D., Cress, A., Triezenberg, S. J., and Guarente, L. (1990) *Cell* **61**, 1199–1208
38. Kim, T. K., Hashimoto, S., Kelleher, R. J., Flanagan, P. M., Kornberg, R. D., Horikoshi, M., and Roeder, R. G. (1994) *Nature* **369**, 252–255
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