MuB, a protein essential for replicative DNA transposition by the bacteriophage Mu, is an ATPase that assembles into a polymeric complex on DNA. We used total internal reflection fluorescence microscopy to observe the behavior of MuB polymers on single molecules of DNA. We demonstrate that polymer assembly is initiated by a stochastic nucleation event. After nucleation, polymer assembly occurs by a mechanism involving the sequential binding of small units of MuB. MuB that bound to A/T-rich regions of the DNA assembled into large polymeric complexes. In contrast, MuB that bound outside of the A/T-rich regions failed to assemble into large oligomeric complexes. Our data also show that MuB does not catalyze multiple rounds of ATP hydrolysis while remaining bound to DNA. Rather, a single ATP is hydrolyzed, then MuB dissociates from the DNA. Finally, we show that “capping” of the enhanced green fluorescent protein-MuB polymer ends with unlabeled MuB dramatically slows, but does not halt, dissociation. This suggests that MuB dissociation occurs through both an end-dependent mechanism and a slower mechanism wherein subunits dissociate from the polymer interior.

DNA transposition is used by many organisms to regulate the dispersion of their genetic material throughout a host cell population (1, 2). The bacteriophage Mu transposition reaction is initiated by the binding of the transposase protein MuA to specific sites at the ends of the phage genome (3). MuA then assembles into a series of nucleoprotein complexes, collectively referred to as transpososomes, that are responsible for catalyzing the DNA processing reactions required to complete transposition (4–7). A second protein, the ATPase MuB, is also required to complete the transposition reaction (8, 9).

MuB performs multiple functions during transposition, including stimulating assembly of the transpososomes, stimulating the MuA catalyzed DNA processing reactions, influencing the selection of the DNA target site, and regulating the disassembly of the transpososome (10–13). MuB does not share extensive sequence similarity with any other known protein. However, like many other ATPases, MuB does contain two conserved amino acid motifs, Walker A and B boxes, that are essential for the binding and hydrolysis of nucleotide triphosphates (14, 15). In the presence of ATP, MuB assembles into large oligomeric complexes on DNA, making the bound DNA an efficient target for strand transfer (16, 17). MuB accumulates on DNA that is not bound by MuA, resulting in a strong preference for transposition to occur at DNA sites at least 10–20 kb from MuA-bound regions (12, 18). This phenomenon is called target immunity and provides a critical mechanism for Mu to prevent disruption of its genome through an aberrant transposition event.

We have established a system that allows us to observe single complexes of MuB bound to an immobilized molecule of DNA (17). We have also demonstrated that MuB forms a large oligomer on DNA and that these oligomers were tightly bound to A/T-rich sequences (17). These same A/T-rich regions were preferentially used as transposition targets by the MuA transposase. Based on these observations, we proposed that MuB could exist in either a high- or low-affinity state and conversion to the high-affinity conformation was dictated by the base composition of the DNA to which the polymer was bound (17).

In this study, we further examine the assembly and disassembly of MuB target complexes bound to surface-tethered molecules of DNA. We demonstrate that polymer assembly is initiated by a nucleation event in which a small unit of MuB binds to A/T-rich DNA in a relatively stable state. This is followed by the sequential net addition of small subunits to the growing complex. When complexes are assembled in the ATP-bound state, the subunits within each complex are unable to exchange ATP for ATPγS without dissociating from DNA, providing further evidence for our previous hypothesis that ATP hydrolysis and DNA dissociation are directly and obligatorily coupled. Finally, the disassembly rates of individual enhanced green fluorescent protein (EGFP)-MuB polymers were dramatically decreased when the ends of the polymers were protected by the addition of unlabeled MuB. We propose that the MuB polymers can disassemble by at least two pathways: either by the dissociation of MuB from the ends of the individual polymers or by the relatively slow dissociation of MuB subunits from the interior of the polymeric complex.

**EXPERIMENTAL PROCEDURES**

*Total Internal Reflection Fluorescence Microscopy*—The total internal reflection fluorescence microscope was a custom-designed system built around a Nikon TE300 Eclipse and has previously been described previously (17). In brief, an argon laser (Spectra-Physics model 543; 488 nm, 100 mW) was focused through a custom-made fused silica prism (J.R. Cumberland, Inc.) onto a flow-cell containing the immobilized target complexes. Fluorescence images were collected through an objective lens (100× Plan Apo, numerical aperture 1.4; Nikon) and captured with an intensified CCD (Pentamax ICCD; Roper Scientific) and image acquisition controlled by Metamorph software (Universal Imaging Corp.). Flow cells were assembled as described previously (17) from fused silica microscope slides (Esco), glass coverslips (Fisher), and

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The abbreviations used are: EGFP, enhanced green fluorescent protein; ATPγS, adenosine 5′-O-(3-thio)triphosphate.
acrylic transfer tape (25 μm thick, 3 m). Before use, flow cells were coated with neutravidin (Pierce) and then rinsed with disassembly buffer (20% glycerol, 25 mM Tris, pH 8.0, 150 mM NaCl, 10 mM MgCl₂, 10 mM dithiothreitol, 10% glucose, 0.05 mg/ml glucose oxidase (USB), 0.01 mg/ml catalase (Calbiochem), and 2 mg/ml casein (Sigma)) and incubated for 30 min. Biotinylated DNA and EGFP-MuB were prepared as described (17). Target complexes were assembled on ice using 10 μM biotinylated DNA, 0.1 μM EGFP-MuB, 2 mM ATP, 10 mM MgCl₂, 150 mM NaCl, 20% glycerol, 25 mM Tris, pH 8.0, 2 mM dithiothreitol, and 0.1 mg/ml bovine serum albumin. Reactions were incubated for 10 min at 25 °C and then chilled on ice until use. For immobilization, the target complexes were diluted 1:10 in wash buffer containing 2 mM ATP and injected into a neutravidin-coated flow cell. Buffer flow was stopped for 10 min to allow binding, and unbound complexes were removed by resuming buffer flow. Image processing and data analysis were performed as described previously (17).

**RESULTS**

**Assembly of Single MuB Target Complexes**—We have reported the development of a total internal reflection fluorescence microscope system for monitoring the behavior of single EGFP-labeled MuB polymers bound to DNA (17). In brief, biotinylated molecules of λ DNA were tethered to the surface of a flow cell, and buffer flow was used to extend the DNA horizontally so that the entire DNA molecule was parallel to the surface of the flow cell. We were then able to observe EGFP-MuB bound to the immobilized DNA by illuminating the sample with total internal reflection.

To examine the assembly of single MuB polymers on the tethered DNA molecules under pre-steady-state conditions, we employed a strategy similar to that previously used for moni-
onitoring the disassembly of single polymers (17). First, the mi-
croscope objective was focused on the imaging plane, and as-
sembly was initiated by rinsing the naked DNA molecules with
2 mM ATP and various concentrations of EGFP-MuB (Fig. 1
A). Progress of the reaction was monitored by capturing images at
defined intervals after initiating assembly and quantitating
the fluorescence signal emitted from EGFP-MuB bound to the
individual DNA molecules (Figs. 1B and 2A). Initial evaluation
of the assembly behavior showed that both the rate and extent
of assembly were dependent upon the concentration of EGFP-
MuB (Fig. 1B). At higher concentrations of EGFP-MuB, 250
and 500 nM, assembly was very rapid, but the details of the
mechanism were obscured at the earliest time points of the
experiment because of the high concentration of free protein
relative to the DNA-bound protein (data not shown). At lower
concentrations, 50 and 100 nM, assembly was much slower,
requiring longer than 15 min to reach a steady state. These
conditions may be more representative of
in vivo
conditions, in
which where DNA is always expected to be in excess over MuB.
The lower concentration of EGFP-MuB made it possible to
distinguish early events in the assembly reaction because of
the reduced background fluorescence from the free protein.
Therefore, we chose to analyze the assembly mechanism in
more detail at lower concentrations of protein.

The time course presented in Fig. 2A shows an example of
the assembly reaction on a single molecule of \( \lambda \) DNA with 50 nM
EGFP-MuB. At the outset of the reaction, no fluorescence signal
was detected on the DNA molecule (Fig. 2A, \( t = 0 \) min). As
the reaction progressed, the molecule of DNA gradually became
visible because of the binding of EGFP-MuB (Fig. 2A). The
gradual increase in fluorescence signal at distinct sites on the
DNA indicated that MuB bound to the DNA as small units,
rather than large preassembled complexes. During assembly,
MuB did not become uniformly distributed on the DNA; rather,
there were very distinct clusters of intense fluorescence signal.
We have previously shown that during dissociation from DNA
molecules saturated with MuB, the regions corresponding to
A/T-rich sites were the most tightly bound (17). These same
A/T-rich regions corresponded to the sites on \( \lambda \) DNA where
MuB oligomer assembly occurred preferentially.

Close inspection of the DNA molecules, such as those shown
in Fig. 2A, revealed that the initiation of assembly did not occur
simultaneously at the different positions on the DNA. Rather,
the assembly of the different MuB oligomers seemed to be
initiated at slightly different times, even though they were all
on the same molecule of DNA (Figs. 2, A and B, and 3B). This
suggested that the assembly reactions were started by individ-
ual nucleation events that led to the formation of larger MuB
polymers. Most of the largest oligomers were assembled at the
positions corresponding to the A/T-rich sites, but there did not
seem to be a conserved order in which assembly was initiated
at the different positions on the DNA (Fig. 3 and data not
shown).

To measure the rate of assembly after nucleation, we moni-
tored the increase in photon emission from individual EGFP-
MuB polymers over time (Fig. 2B; see Ref. 17 for details of
signal quantitation). The fluorescent signal from the individual
polymers of MuB shown in Fig. 2A increased linearly over time,
corresponding to an estimated rate of ∼8–14 molecules of EGFP-MuB per minute. The assembly rates for 232 different MuB polymers exhibited an estimated average net assembly rate of ∼12 molecules of MuB per minute (Fig. 2C). Some of the polymers seemed to assemble much faster than others, with rates up to 38 molecules per minute. However, it was possible that in these instances there may have been two or more closely juxtaposed polymers that we could not spatially resolve. Taken together, these results suggest that assembly of the MuB oligomers was initiated by a stochastic nucleation event involving smaller order oligomers of MuB. This was followed by incorporation of additional MuB during the continued assembly of the complexes.

Stochastic Nucleation and Oligomer Stability—To examine the details of the early nucleation events, we aligned the images of nine different DNA molecules captured at either 3 or 15 min after initiating the assembly reactions (Fig. 3A). This showed that the sites on the DNA at which the early nucleation events occurred could vary quite dramatically from molecule to molecule. However, the distribution pattern of MuB on different DNA molecules did not differ greatly in reactions that were allowed to reach steady state. Fig. 4 shows the MuB distribution patterns on four different molecules of DNA 30 min after assembly was initiated. In this case, the images were captured at 5-min intervals, starting from the 30-min time point and continuing until 60 min. These images show that once the reactions have reached steady state, the distribution of MuB is similar on the different DNA molecules and remains constant (Fig. 4). The punctate pattern of MuB binding observed in Fig. 4 also correlates with the ATP distribution in λ DNA, and we have shown previously that during disassembly, EGFP-MuB remains more tightly bound to λT-rich regions of the λ DNA (17).

Not all of the polymers we observed continued to increase in size during the course of the experiment. For example, compare the polymers at the 3.5 and 5.5 μm positions in Fig. 3B. Both polymers began to assemble at ∼2 min after initiating the reaction (Fig. 3B). From 4 to 6 min, both polymers increase in size at similar rates. However, after 6 min, the polymer at the 3.5-μm position continued to assemble, whereas the signal from the 5.5-μm region no longer increased and the polymer itself could no longer be clearly resolved after ∼7 min. Together, these results indicated that over time only the most stably bound DNA sites remained bound by large MuB polymers and that the MuB bound to the less preferred sites eventually dissociated from the DNA.

**ATP Hydrolysis Is Linked to Dissociation of MuB from DNA**—We have proposed previously that MuB is unable to catalyze more than a single round of ATP hydrolysis before dissociating from DNA (17, 19). To further support this hypothesis, we examined the effects of ATPγS on the dissociation rate of MuB initially bound to DNA in the presence of ATP. MuB is unable to efficiently hydrolyze ATPγS, and bulk experiments have shown that when complexes of MuB are assembled in the presence of ATPγS, the protein is essentially irreversibly bound to the DNA (16, 19). Using the total internal reflection fluo-
cence microscope, we have previously shown that MuB initially bound to λ DNA in the presence of ATPγS does not dissociate even after washing for several hours in the absence of any nucleotide (17). Based on these previous results, we reasoned that if MuB were capable of catalyzing multiple rounds of ATP hydrolysis without dissociating from DNA, this would be revealed as the complete inhibition of the disassembly reaction when the complexes assembled with ATP were chased with ATPγS (Fig. 5A).

Fig. 5B shows examples of target complex disassembly during a chase with either no nucleotide (top) or when 2 mM ATPγS was included in the chase buffer. In the absence of nucleotide, the disassembly data could be fit to a single exponential decay curve, each with an apparent rate constant of \(-0.4 \text{ min}^{-1}\) (data not shown). The dissociation rate was the same, \(-0.4 \text{ min}^{-1}\), when ATPγS was included in the chase buffer, and the vast majority of the MuB was able to dissociate even though ATPγS was present (Fig. 5B). This was consistent with our previous conclusions and indicated that the majority of the MuB within the target complex does not hydrolyze multiple molecules of ATP before dissociation. Although most of the MuB dissociated from the DNA when chased with ATPγS, a small fraction (\(-5\%\)) of MuB remained bound to the DNA after the ATPγS chase (Fig. 5B). It is likely that a small fraction of the protein in the assembly buffer bound to ATPγS when inadvertently mixed with the chase buffer during the buffer switch, and this protein could then bind stably to the DNA.

**EGFP-MuB Dissociation Is Slowed in the Presence of Unlabeled MuB** —Previously we proposed a mechanism in which the disassembly of the MuB polymers occurred through an end-dependent reaction (17). This hypothesis was supported by the observation that the polymers containing up to \(-100–200\)
monomers behaved as individual entities and exhibited stochastic behaviors during disassembly (17). However, the disassembly kinetics were not linear, as would be expected for an end-dependent dissociation mechanism. The observed kinetics could be fit to single exponential decays more consistent with a random dissociation mechanism. To further analyze the mechanism of polymer disassembly, we decided to examine the behavior of the MuB target complex when both disassembly and assembly can occur simultaneously. In an attempt to examine the possibility that polymer disassembly was occurring via an end-dependent mechanism, we included unlabeled MuB in the chase buffer. If dissociation were random, then the probability of a particular MuB subunit dissociating from the DNA should be independent of that subunit’s positions within the polymer (i.e. relative to the polymer ends). In this case, the disassembly of the EGFP-MuB polymers should not be influenced by the presence of the unlabeled MuB in the chase buffer. If, however, the disassembly mechanism were not random and occurred via an end-dependent process, then the presence of unlabeled MuB in the disassembly buffer would be expected to influence the dissociation rate of the fluorescent MuB (Fig. 6A).

Target complexes were assembled with 50 nM EGFP-MuB and 2 mM ATP and then chased with buffer containing 2 mM ATP and 500 nM unlabeled MuB (Fig. 6B). At this concentration of unlabeled MuB, the protein is expected to saturate the available DNA sites very rapidly (see Fig. 1). Therefore, the DNA will be covered primarily by unlabeled MuB that is interspersed by shorter polymers of EGFP-MuB. When the unlabeled MuB was included in the chase buffer, the disassembly rate of the EGFP-MuB was dramatically decreased (Fig. 6B).

The dissociation rate of EGFP-MuB was reduced from 0.4 min$^{-1}$ to 0.086 min$^{-1}$, corresponding to an increase of nearly 5-fold in the half-life of the EGFP-MuB bound within the target complex (1.7 versus 8.1 min). These results suggest that the dissociation of MuB from the target complex does not occur via the random dissociation of subunits. On the other hand, it is also possible that saturating the DNA with unlabeled MuB induces some completely different disassembly pathway that does not occur at lower concentrations of protein. However, we believe that this is unlikely because the dissociation rate for EGFP-MuB does not vary over a wide range of protein concentrations (data not shown).

In addition to the slower dissociation rate in the presence of the unlabeled MuB, two other changes were observed in all the DNA molecules examined (>25). First, when the unlabeled MuB entered the flow cell, all of the DNA molecules seemed to increase in length by 1-2 nm (compare the 0- and 2-min data in Fig. 6B). This suggested that binding of MuB results in a slight lengthening of the bound DNA. Second, there was a small increase in the fluorescence signal on the target complex that also coincided with the initial binding of the unlabeled protein (Fig. 6B). We considered the possibility that the increase in the total amount of protein bound to the DNA resulted in a conformational change in the DNA bound EGFP-MuB that may have led to an effective increase in the quantum yield of EGFP-MuB. However, we were unable to observe similar increases in fluorescence intensity in ensemble measurements when unlabeled MuB was titrated into reaction mixes containing EGFP-MuB; therefore, we consider this possibility unlikely (data not shown). A second, more likely, explanation is that the high
concentration of unlabeled protein stimulated the binding of additional EGFP-MuB via cooperative interactions during the initial stage of the buffer exchange.

**DISCUSSION**

The Assembly of the MuB Target Complex—Fig. 7 depicts a possible model describing the assembly behavior of the MuB polymers. The assembly of the MuB polymer is likely to initiate with the binding of a small protomeric unit of ATP-MuB to the DNA molecule (Fig. 7, step 1). MuB binds to A/T-rich DNA in a relatively stable configuration, allowing continued assembly of the complex (Fig. 7, step 2). MuB binds G/C-rich regions less tightly and does not assemble into large complexes at these sites. Under the conditions used for our experiments, EGFP-MuB is preincubated with ATP and is therefore already in the ATP-bound protomeric state. The oligomeric state of MuB in the absence of DNA is highly dependent upon the concentrations of both ATP and MuB (16, 19). Therefore, the concentrations of these reaction components are likely to influence the size of the MuB protomer, which initially binds to the DNA. Although we do not directly observe the individual binding events in the experiments presented here, it is clear from our data that nucleation and continued assembly involve smaller order oligomers of MuB. After assembly has begun, the MuB subunits located at the interior of the polymer may dissociate more slowly than those located at the end (Fig. 7, step 3). This aspect of the model is based on the observation that the EGFP-MuB polymers dissociate more slowly when the polymer ends are blocked by chasing with unlabeled MuB. Finally, polymer extension beyond the A/T-rich DNA into the G/C-rich DNA is unfavorable because MuB binds less tightly to the G/C-rich regions.

In this model, there are two important aspects that influence MuB polymer assembly. First, is the kinetically preferred binding to A/T-rich DNA sequence. This observed sequence dependence leads to a characteristic pattern of MuB binding on the λ DNA, which reflects the base composition of the DNA (Fig. 4 and Ref. 17). We also believe that polymer assembly is mediated through an initial nucleation event. That is, the binding of additional subunits becomes energetically more favorable after the first MuB protomer binds the A/T-rich DNA. This view is supported by previous experiments showing that MuB binds cooperatively to DNA (16) and by the data presented in Fig. 3A, top, which shows MuB polymers soon after the initiation of assembly (i.e. nucleation) on several different DNA molecules. At the time point (3 min) shown in this panel, we estimate that the individual polymers of MuB contain approximately 100 molecules of protein (see Fig. 1B). If nucleation only represented binding to a kinetically preferred site (i.e. A/T-rich), then it is unlikely that the individual molecules of DNA would display any significant variation in the position of the MuB polymers at this stage of the reaction. Rather than showing distinct, relatively large MuB clusters at defined sites within the A/T-rich DNA, the protein would be more evenly dispersed throughout the entire A/T-rich region. Unfortunately, we do not have the resolution required to directly resolve individual binding events, and we can now only infer that each individual polymer initiated from a single nucleation event based on the wide variations in positioning at this early stage of the reaction. Therefore, it is formally possible that each polymer will result from multiple, independent binding events.

ATP Hydrolysis and Target Complex Disassembly—We have previously proposed that ATP hydrolysis is directly coupled to polymer disassembly such that each MuB monomer within the polymer hydrolyzes one molecule of ATP before dissociation from the complex (17, 19). The new evidence presented here confirms our earlier hypothesis by showing that the majority of
the MuB within the target complexes was unable to exchange bound ATP (or ADP) for ATPγS.

The inability of MuB to turnover multiple ATP molecules while remaining bound to DNA also has important implications regarding the interactions between MuA and MuB that are necessary to establish target immunity. Previously proposed models for ATP hydrolysis suggested that MuB was capable of hydrolyzing multiple ATP molecules without dissociating from DNA (12, 16). According to this model, ATP hydrolysis was necessary but not sufficient for MuB dissociation from DNA, and the additional impact of the MuA-MuB interaction was needed for the dissociation of MuB from DNA (16). This conclusion was based on several observations. First, in previous studies, we have used ATPγS to “freeze” the distribution of MuB between immune and non-immune targets that was established in the presence of ATP and MuA. We thought that this was possible because it seemed as though nucleotide exchange could occur without obligatory dissociation of MuB from the DNA. The results presented here are inconsistent with this previous interpretation. A critical difference between the previous experiment and the experiments presented here is that in the previous experiments, any MuB that dissociated from the DNA could also re-bind to the DNA. In the experiments presented here, once a molecule of MuB dissociates from the DNA, it is removed from the reaction by the continuous flow of buffer and thus can not re-bind to the DNA molecules. It is possible that in the previous experiments, when dissociation and re-binding can take place concurrently in the presence of ATPγS, the MuB distribution pattern tends to maintain itself. This would be possible if re-binding to DNA next to a pre-existing polymer is faster than de novo nucleation at a site on the DNA unbound by MuB. This hypothesis is supported by the observation that MuB binds cooperatively to DNA (16). Our current data support a much simpler model in which MuA need only stimulate the turnover by MuB of 1 bound molecule of ATP. This would lead to rapid dissociation of MuB from the DNA and provides MuA with an efficient mechanism for preventing MuB from accumulating at sites near the Mu ends.

We have previously proposed a model for the disassembly of the DNA-bound MuB oligomers in which dissociation (or addition) of new subunits occurs at the end-units of the polymer (17). Herein, we show that when oligomers of EGFP-MuB are bound to the target DNA and then chased with unlabeled MuB, the dissociation rate of the fluorescent subunits is much slower than was observed in the absence of the unlabeled protein. Taken together with our previous results, these data suggest that there may be at least two pathways for disassembly: an end-dependent dissociation mechanism along with an internal dissociation mechanism that is one-fifth the rate of the end-dependent mechanism. The combination of these two pathways could yield the apparent exponential decay kinetics previously observed during target complex disassembly (17, 19).

**Target Immunity and the Distribution of MuB—**Our results highlight the non-random distribution of the polymeric MuB complexes on DNA as an additional level of regulation during the selection of a target site during Mu transposition. MuB displays a clear preference for binding to AT-rich regions of DNA. This preference for MuB binding to AT-rich regions is also reflected in the distribution of transpososome insertion sites in vitro and in vivo (17, 20, 21).

The non-random distribution of sites at which MuB can assemble into large polymers, and therefore acceptable sites for transpososome insertion, has important implications for target immunity. That is, target immunity does not have to prevent insertions over the entire 38-kb phage genome. Rather, it only has to function at what may be a limited number of AT-rich “hotspots” present within the phage genome. MuA could in effect “ignore” any MuB that is not assembled into a large polymeric complex, and if no “hotspots” are present within a given region of DNA, then it is possible that no additional protection would be required to prevent transposition into this DNA. Interestingly, the Mu genome is itself relatively free of any extended regions of AT-rich sequences and the only areas that have regions that would seem to be potential “hotspots” for MuB binding are nearest to the ends of the phage genome (data not shown). The central 30 kb of the Mu genome has no extended sequences, which would seem to preferentially support the formation of the MuB target complexes.

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**REFERENCES**

1. Craig, N. L. (1997) *Annu. Rev. Biochem.* **66**, 437–474

2. Mizuuchi, K. (1992) *Annu. Rev. Biochem.* **61**, 1011–1051

3. Craigie, R., Mizuuchi, M., and Mizuuchi, K. (1984) *Cell* **39**, 387–394

4. Craigie, R., and Mizuuchi, K. (1987) *Cell* **51**, 493–501

5. Surette, M. G., Buch, S. J., and Chaconas, G. (1988) *Cell* **49**, 253–262

6. Baker, T. A., and Mizuuchi, K. (1992) *Genes Dev.* **6**, 2221–2232

7. Mizuuchi, M., Baker, T. A., and Mizuuchi, K. (1992) *Cell* **70**, 303–311

8. Chaconas, G., Giddens, E. R., Miller, J. L., and Gloor, G. (1985) *Cell* **41**, 857–865

9. Chaconas, G., Gloor, G., and Miller, J. L. (1985) *J. Biol. Chem.* **260**, 2626–2669

10. Baker, T. A., Mizuuchi, M., and Mizuuchi, K. (1991) *Cell* **65**, 1003–1013

11. Levchenko, I., Yamauchi, M., and Baker, T. A. (1997) *Genes Dev.* **11**, 1561–1572

12. Adzuma, K., and Mizuuchi, K. (1988) *Cell* **53**, 257–266

13. Adzuma, K., and Mizuuchi, K. (1989) *Cell* **57**, 41–47

14. Nakayama, C., Leung, P. C., and Harshey, R. M. (1988) *J. Biol. Chem.* **263**, 10851–10857

15. Yamauchi, M., and Baker, T. A. (1998) *EMBO J.* **17**, 5509–5518

16. Adzuma, K., and Mizuuchi, K. (1991) *J. Biol. Chem.* **266**, 6159–6167

17. Greene, E. C., and Mizuuchi, K. (2002) *Mol. Cell* **9**, 1079–1089

18. Manna, D., and Higgins, P. N. (1999) *Mol. Microbiol.* **32**, 585–606

19. Greene, E. C., and Mizuuchi, K. (2002) *EMBO J.* **21**, 1477–1486
Visualizing the Assembly and Disassembly Mechanisms of the MuB Transposition Targeting Complex

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