The EB1 Homolog Mal3 Stimulates the ATPase of the Kinesin Tea2 by Recruiting It to the Microtubule*\[S\]

Received for publication, December 3, 2004, and in revised form, January 18, 2005
Published, JBC Papers in Press, January 23, 2005, DOI 10.1074/jbc.M413620200

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Tea2 is a kinesin family member from *Schizosaccharomyces pombe* that is targeted to microtubule tips and cell ends in a process that depends on Mal3. Constructs of Tea2 containing the motor domain only or the motor domain plus the N-terminal extension are monomeric, whereas a construct including the first predicted coiled coil region is dimeric. These constructs have a low basal rate of ATP hydrolysis of &lt;0.1 s\(^{-1}\), but microtubules stimulate the rate of ATP hydrolysis to a maximum of &lt;15 s\(^{-1}\). Hydrodynamic analysis of Mal3 indicates that it is dimeric. Mal3 is known to associate with Tea2, and analysis with the above Tea2 constructs indicates that the principal site of interaction of Mal3 with Tea2 is the N-terminal extension, although a weaker interaction is also observed with the motor domain alone. In parallel to the binding studies, Mal3 strongly stimulates the ATPase of constructs containing the N-terminal extension by decreasing the *K*\(_{\text{cat,MT}}\) for stimulation by microtubules but only weakly stimulates motor domains without the N-terminal extension. Mal3 reduces the *K*\(_{\text{cat,MT}}\) values without affecting the *k*\(_{\text{cat}}\) value at saturating microtubule level. Binding of Mal3 to microtubules induces an increase in the binding of Tea2 and a reciprocal stimulation of Mal3 binding by Tea2 is also observed.

Tea2 is a plus end directed motor that drives sliding of axonemes when adsorbed to a glass surface. The sliding rate is initially unaffected by Mal3, but axonemes stop moving on continued exposure to Mal3.

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\[S\] The on-line version of this article (available at http://www.jbc.org) contains a movie.

† This work is dedicated to the memory of Heidi Browning who will be greatly missed.

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\[S\] The abbreviations used are: MT, microtubule; Nte, N-terminal extension; Trx, thioredoxin; Ni-NTA, nickel-nitrilotriacetic acid; ACES, 2-(2-amino-2-oxoethylamino)ethanesulfonic acid; AMP-PNP, adenosine 5'-[(β,γ-imino)triposphate.

* This work was supported by National Institutes of Health Grant NS28562. The costs of publication of this article were defrayed in part by the payment of page charges. This article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

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or Tip1, but Mal3 is required for targeting of Tea2 and Tip1 (7, 8). Human EB1 can substitute for Mal3 to restore accumulation of Tea2 at the MT end, (7) and Mal3 can substitute for at least some functions of EB1 (18, 19).

Because of limited sensitivity, previous work in cells focused on observation of the large assemblies (dots) that have multiple copies of the fluorescently labeled components in the same location and are therefore bright and punctate. Recently it has become possible to observe smaller accumulations of these proteins as speckles (8). This has indicated that speckles containing fluorescently labeled Tea2 do move actively with a variable rate. Their movements are mainly, but not exclusively, toward the plus ends of the MTs. Moving speckles are also observed with fluorescently labeled Tip1; the Tip1 speckles colocalize with speckles of Tea2, and their movement is dependent on Tea2 (8). Similar moving speckles have been observed with the homologous proteins in budding yeast (17). These results have lead to the suggestion that Tip1 is moving as a cargo of Tea2 toward the plus end where it may be deposited as a complex in conjunction with Mal3. Although Mal3 accumulates at MT ends, there is also a large cytoplasmic pool of Mal3 (15) and fluorescently labeled Mal3 is observed along the length of the MTs (7, 8, 15).

As an initial stage in the analysis of how such complex and dynamic assemblies can be generated and targeted to specific cellular locations, we have begun an investigation of the enzymatic and motile properties of Tea2 and how Tea2 is influenced by interaction with Mal3. This has directly demonstrated that Tea2 is a plus end directed motor and that Mal3 stimulates ATP hydrolysis by recruiting Tea2 to the MT, without a significant effect on the $k_{\text{cat}}$ at saturating MT levels.

### MATERIALS AND METHODS

Domain fragments of Tea2 were cloned by PCR into a modified pET32 vector (Novagen) that encoded thioredoxin (Trx), a His-tag, a thrombin cleavage site, and a Tev protease cleavage site followed by a BamH1 site for insertion of the Tea2 fragment. The non-fusion proteins obtained by cleavage with Tev protease have the tripeptide GGS appended to the C terminus, T2M, T2MN, and T2NMC1 are indicated schematically in Fig. 1 and encode Tea2 residues 2–122, 124–476, 2–476, and 2–524, respectively. Mal3 encodes residues 2–308. All constructs also have Kpn1 and Sph1 sites encoding GTTS appended to the C terminus, except T2NMC1, which has the sequence GTTSGTEFCA appended to the C terminus. All PCR-derived regions were confirmed by DNA sequencing. A His-tagged version of the Tea2 protease was purified by the method of (20) and using the plasmid TPSN kindly provided by J. A. Doudna. General procedures for expression and lysis were as described previously (6, 21). All solutions containing Tea2 constructs were supplemented with $\geq 0.1$ mM ATP. The Trx fusion proteins were purified on Ni-NTA columns and eluted with a step to 100 mM imidazole in B8 buffer (15 mM Bicine, pH 8.0, 2 mM magnesium acetate, and 0.02% mercaptoethanol) with 400 mM NaCl. Tev protease was added to the peak fractions, and they were then dialyzed for 1–2 days against B8 buffer with 400 mM NaCl and washed with a step to 100 mM imidazole in B8 buffer (15 mM Bicine, pH 8.0, 2 mM magnesium acetate, and 0.02% mercaptoethanol) with 400 mM NaCl. Tev protease was added to the peak fractions, and they were then dialyzed for 1–2 days against B8 buffer with 400 mM NaCl and washed with a total of 0.8 ml 25 mM NaCl, and these fractions were pooled as the flow-through fraction (FT). The column was then washed with 1-ml volumes of 25, 150, and 300 mM NaCl.

**Binding of Mal3 to Tea2 Domains**—Small columns of Ni-NTA (0.2 ml) were loaded with 0.5 ml of 50 $\mu$M test protein as a fusion with Trx. The columns were washed with 500 mM NaCl and then shifted to 25 mM NaCl. Essentially all the test protein bound to the column. Mal3 (0.2 ml of 11 $\mu$M) was then loaded in 25 mM NaCl and washed with a total of 0.8 ml 25 mM NaCl, and these fractions were pooled as the flow-through fraction (FT). The column was then washed with 1-ml volumes of 25, 150, and 300 mM NaCl.

**Binding of Tea2 and Mal3 to MTs**—Samples in A25 buffer with 100 mM NaCl and 10 $\mu$M taxol were centrifuged at 20 °C for 25 min at 250,000 $\times$ g. An aliquot was removed for the supernatant fraction, and the remainder of the supernatant was aspirated. The tube and surface of the pellet were not rinsed, and the pellet fraction was obtained by adding SDS-PAGE sample buffer to the tube.

**Motility**—Attempts to obtain motility of Tea2 constructs by direct adorption to glass coverslips were unsuccessful. Motility was observed when T2NMC1 was coupled to a segment of the tail of conventional kinesin that is required for tight binding of conventional kinesin-1 to glass surfaces. Coupling via a disulfide bond (24) was between T2NMC1 and a fusion protein of the first PDZ domain of InaD and residues 910–937 of *Drosophila* kinesin-1. Coverslips were washed with soap and then treated sequentially for $>2$ h with 2 N KOH and 3 N HCl, rinsed in water, and air-dried. A flow chamber was constructed using double-sided tape. After adsorption of Tea2, the chamber was blocked with 0.4 mg/ml casein in A25 buffer (without mercaptoethanol) with 60 mM KCl and 1 mM MgATP, and then salt-washed with an aquchen axoneme were introduced in the same buffer. Motility was observed at room temperature (21–25 °C) by differential interference contrast microscopy following a final flush with the same buffer to remove unadsorbed axonemes.

**RESULTS**

Oligomeric Characterization—Tea2 is similar to BimC and some other kinesin superfamily members in that it has an N-terminal type motor domain near its N terminus but also has an additional domain (N-terminal extension (Nte)) before the motor domain as indicated in Fig. 1. The rest of the molecule contains two predicted coiled coil regions and a final C-terminal non-coiled coil region. Constructs of the motor domain and adjoining regions were cloned, expressed, and purified to determine the basic enzymatic and motile properties of Tea2 and their interaction with Mal3. T2M is a minimal construct that contains the full motor domain and neck linker and corresponds to monomeric DKH346 or DKH357 of conventional kinesins.

**FIG. 1. Tea2 constructs.** T2NMC1 contains the motor domain of Tea2 (oval); the Nte is indicated by a solid line, and the first coiled coil region is indicated by an open box. The N and C termini are close in space for the motor domain with a docked neck linker (28), and consequently the Nte and coiled coil region are indicated as originating from the same side of the motor domain. T2NMC1 is dimeric, and oligomerization is likely due to the coiled coil region as the other constructs that lack this region are all monomeric.
Mal3 was loaded in 25 mM NaCl and determined by SDS-PAGE analysis of the eluent fractions as described under "Materials and Methods." Mal3 was loaded in 25 mM NaCl and washed sequentially with 25, 150, and 300 mM NaCl. The preoad Mal3 sample is indicated by Pre, and the flow-through in 25 mM NaCl is indicated by FT. Loading of each fraction was adjusted so that the protein concentration would equal that of the preoad (Pre) if the recovery was 100% in that fraction.

Localization of Mal3 Binding Site on Tea2—Previous work indicated that Mal3 bound to Tea2 (7, 8). The site of binding on Tea2 was investigated by measuring the binding of Mal3 to Tea2 constructs attached to a Ni-NTA column as His-tagged fusion protein or with thioredoxin alone bound to the column, as indicated in Fig. 2. In the absence of a fusion protein or with thioredoxin alone bound to the column, the Mal3 passed through the column without binding. When T2M was bound, much of the Mal3 still passed through, but the Mal3 passed through the column with the same concentration and diffusion coefficients of 3.8 S and 4.5 \( \times 10^{-7} \) cm²/s, respectively. These values are essentially identical to those of the corresponding kinesin-1 and BimC constructs and indicate that T2M and T2NM are monomeric, whereas T2NMC1 is predominantly dimeric at the concentration of 0.1 mg/ml at which the sedimentation was performed. Mal3 has sedimentation and diffusion coefficients of 3.8 S and 4.5 \( \times 10^{-7} \) cm²/s, respectively, which result in a calculated molecular mass of 76 kDa. The predicted mass of a Mal3 monomer is 35.5 kDa, and thus Mal3 is dimeric in solution. These results also indicate that the Mal3 dimer is highly asymmetric. In comparison to the globular standards, Mal3 elutes close to catalase (200 kDa) on gel filtration but shifts dramatically to migrate close to ovalbumin (45 kDa) on sedimentation.

MT-stimulated ATPase—MTs stimulate the ATPase rate of Tea2 as illustrated in Fig. 3. There is a progressive increase in MTs affinity for T2M, T2NM, and T2NMC1 as indicated by a decrease in \( K_{\text{M}} \) for the MT concentration required for half-saturation of the stimulation by MTs when measured at the same ionic strength. With 25 mM KCl, T2NM has a \( K_{\text{M}} \) value of 0.26 \( \mu \)M, whereas the value for T2M is too weak to accurately measure (Fig. 3B) and the value for T2NMC1 is too tight to accurately measure due to mutual depletion (data not shown). At 100 mM KCl where the binding of T2NM is too weak to measure, T2NMC1 still has a \( K_{\text{M}} \) value of 1.6 \( \mu \)M (Fig. 3A). Both T2NM and T2NMC1 have \( k_{\text{cat}} \) values at saturating levels of MTs of \(-15\) s⁻¹.

Mal3 Stimulates MT-ATPase of Tea2 at Low MT Concentration—Mal3 strikingly stimulates the rate of ATP hydrolysis for T2NM and T2NMC1 at subsaturating levels of MTs but has little effect on the \( k_{\text{cat}} \) value at saturation as indicated in Fig. 3. The magnitude and rate of onset of the activation are illustrated in Fig. 4. The slope of the decrease in NADH absorption with time measures the ATPase rate with the coupled assay system, and the low slope before addition of Mal3 indicates a low ATPase rate. On addition of Mal3, the slope and therefore ATPase rate markedly increases. The onset of the activation is fast and occurs within the few seconds required for the response of the coupled enzyme system (the response to the pulse of ADP illustrates the response time of the assay). The dependence of the activation on the MT concentration is given by the open symbols in Fig. 3, and the results indicate that activation is only observed at subsaturating levels of MTs. Mal3 also stimulates T2M (Fig. 3B) but to a greatly reduced degree.

Low amounts of Mal3 are sufficient for maximum stimulation of T2NM and T2NMC1, and even a large excess of Mal3 is not inhibitory as indicated in Fig. 5. A and B. T2M also exhibits a concentration dependent activation by Mal3 (Fig. 5C). Although these experiments had to be performed at differentionic strengths and MT concentrations due to the different affinities of the motors, it is interesting to note that the stimulation of the ATPase by Mal3 saturates at a concentration that is approximately stoichiometric to the concentration of MTs in each case. Thus, this stimulation is likely due to binding of Mal3 along the length of the MT and any specific effect due to preferential interaction of Mal3 with the plus ends would likely be at too low a stoichiometry to contribute significantly to the total rate. At higher ionic strength where the binding of Mal3 and Tea2 to MTs is weaker, greater than stoichiometric amounts of Mal3 are required (data not shown). The activation by Mal3 is not a generalized effect on kinesin motor domains, because addition of Mal3 produces only monotonically increasing inhibition with the DKH357 motor domain of conventional kinesin-1 (Fig. 5C).

Mal3 and Tea2 Mutually Recruit Each Other to the MT—The decreased \( K_{\text{M}} \) value for Tea2 in the presence of Mal3 suggests that Mal3 increases the net affinity of Tea2 for the MT. This is confirmed by direct MT binding studies with T2NM that are summarized in Fig. 6. In the absence of MTs, only trace amounts of Tea2 or Mal3 are observed in the pellet fraction due...
Tea2 Activation by Mal3

ATP hydrolysis was followed using the coupled enzyme system of pyruvate kinase and lactic dehydrogenase that results in the oxidation of NADH and a decrease in absorbance at 340 nm. Reaction was initiated just before the beginning of data acquisition by addition of T2NMC1 and MTs to 0.02 and 0.22 μM, respectively, in 100 mM KCl in A25 buffer with 2 mM P-enolpyruvate, 1 mM MgATP, 0.15 mM NADH, and high concentrations of pyruvate kinase and lactic dehydrogenase (0.083 and 0.036 mg/ml, respectively) for rapid response to ADP release. At the indicated times, the response of the coupled enzyme system was tested by addition of ADP to 0.01 mM, and then Mal3 was added to 0.28 μM.

Tea2 is also illustrated by lanes 2–5 in Fig. 6B with increasing amounts of Mal3. The binding of Tea2 to the MT produces a reciprocal recruitment of Mal3 as indicated by lanes 8–15 in Fig. 6B. In the absence of Tea2, the majority of the Mal3 is not bound to the MTs (Fig. 6B, lane 10 versus lane 11), whereas more Mal3 is bound when Tea2 is present (Fig. 6B, lanes 12 versus lane 13). When AMP-PNP induces high binding of Tea2 to the MT, then the binding of Mal3 is even further enhanced with most of the Mal3 bound (Fig. 6B, lane 14 versus lane 15). Lane 12 in Fig. 6A indicates that high amounts of both Tea2 and Mal3 can bind simultaneously to the MT in the presence of AMP-PNP.

Tea2 Is a Plus End-directed Motor with Time-dependent Inhibition by Mal3—Axonemes slide along a Tea2 coated surface in 60 mM KCl with a broad distribution of velocities at 0.057 ± 0.015 μm/s for axonemes that are gliding smoothly. To determine the polarity of movement of Tea2, an initial observation was made of the direction of sliding of individual axonemes, and then beads with adsorbed kinesin-I were introduced into the flow cell. The beads were always observed to move toward the trailing end of the axoneme. Because kinesin-I is a plus end-directed motor, this indicates that the trailing end of the axoneme in Tea2-dependent sliding is the plus end and that Tea2 is thus also a plus end-directed motor.

To determine the influence of Mal3 on movement, a similar procedure was followed in which an initial observation of Tea2-dependent movement was made, and then varying concentrations of Mal3 were introduced into the flow cell. When flushed with 0.25 μM Mal3, the axonemes initially continued moving at...
a similar rate but gradually slowed down (often with pauses) and then stopped so that no axonemes were still moving after 5–10 min (see the movie in the supplemental material). When flushed with higher concentrations of Mal3, the lag before movement stopped was shortened. A prominent feature of the inhibition by Mal3 was that many of the axonemes that had been gliding smoothly before addition of Mal3 lost attachment to the surface along their length and remained attached only at their trailing plus ends (see the movie in the supplemental material). Of 59 axonemes that were tracked, 7 detached during the addition of Mal3 and were swept away by the flow. The remainder came to a stop with 22 attached to the coverslip surface along their whole length and 30 attached only by their plus ends. When conventional kinesin-1 (DKH960 (26)) was adsorbed to the coverslip instead of Tea2, a much higher Mal3 level of 5 μM had little effect on the sliding velocity in 60 mM KCl (0.59 ± 0.06 μm/s without Mal3 versus 0.56 ± 0.4 μm/s with Mal3).

**DISCUSSION**

The Tea2 motor constructs have ATPase properties that are similar to those of several other kinesin family members, although the $k_{cat}$ value of $15 \text{ s}^{-1}$ at saturating levels of MTs are slower than the value of $60 \text{ s}^{-1}$ for conventional kinesin motor domains (25) and $25 \text{ s}^{-1}$ for BimC (6). As also observed for conventional kinesin, Tea2 constructs lacking the C-terminal coiled coils are monomeric, but T2NNMC1 with the first coiled coil region is dimeric. The purified protein moves toward the plus end of MTs in an *in vitro* motility assay, as anticipated from its classification as an N-terminal motor, the mainly plus end-directed movement of Tea2 speckles in *vivo* (8) and its physiological role. The average sliding velocity of 0.054 μm/s in 60 mM KCl is slower than for conventional kinesin (0.59 μm/s) in agreement with the lower $k_{cat}$ value for Tea2. The relative decrease in sliding velocity for Tea2 is greater than the relative decrease in $k_{cat}$ value, but Tea2 exhibited a broad range of sliding rates, and the observed average in 60 mM KCl may underestimate the true sliding rate for optimal conditions.

At subsaturating concentrations of MTs, Mal3 strikingly stimulates the ATPase rate of Tea2 constructs that contain the Nte and weakly stimulates T2M without the Nte (Fig. 3). Because binding to MTs with coupled release of ADP is expected to be the rate-limiting step at low MT concentrations, this stimulation suggests that Mal3 is acting by increasing the binding of Tea2 to the MT. Such recruitment of Tea2 to the MT by Mal3 would not result in a net increase of the ATPase rate if Tea2 was inhibited from rapidly releasing ADP in the ternary complex of MTs with Mal3 and Tea2. The lack of inhibition by high levels of Mal3, however, indicates that this is not a factor and that cyclic ATP hydrolysis by Tea2 is fully active in the ternary complex. Direct binding studies demonstrate that the Nte of Tea2 is sufficient for tight interaction with Mal3 (Fig. 2) and that the interaction of Tea2 with Mal3 results in their mutual recruitment to the MT (Fig. 6). Many kinesins contain an auxiliary MT binding site outside of the motor domain (see Ref. 6) that can increase the net affinity of the motor for the MT. Mal3 also provides an auxiliary MT binding site that increases the affinity of the motor for the MT, but Mal3 provides the auxiliary MT binding site intramolecularly rather than intramolecularly.

Interpretation of these results will depend highly on determination of the structure of the ternary complex. For example it is possible that the MT binding domain of Mal3 and the motor domain of Tea2 may bind simultaneously to the same tubulin dimer on a MT or that they may interact with neighboring tubulin binding sites, especially at low levels of binding to the MT. Tea2 must still be able to bind to the MT when high levels of Mal3 are already bound because the maximum ATPase is not inhibited under these circumstances. However, it is also possible that binding of high levels of Tea2 (such as when AMP-PNP is present) displaces Mal3 from the MT so that Mal3 remains tethered to the MT through binding to the Nte of Tea2 but is not directly interacting with the MT.

Previous gel filtration studies with a fusion protein of maltose-binding protein and EB1 indicated that it had a large Stokes radius and was possibly tetrameric (27). Analysis presented here with Mal3 indicates that it is dimeric but does have a larger Stokes radius than expected for a globular dimer because it is highly asymmetric. The dimeric nature of Tea2 and of Mal3 suggests that their interaction may produce a heterotetramer with a Mal3 dimer acting as a cross-link between Tea2 motor domains. Alternatively, the multivalency of these proteins could result in large open networks, especially given the fact that several other proteins are known to interact as well. In particular, Tip1 is also dimeric and binds both to the MT binding domain of Mal3 and to the C-terminal region of Tea2 (as well as binding independently to MTs), and this could lead to extensive cross linking and the formation of the large “dots” at the ends of MTs.

The initial binding equilibria between Mal3 and MTs or Tea2 are established rapidly as indicated by the lack of a lag on addition of Mal3 in Fig. 4. Thus the continuation of rapid sliding for many axonemes after addition of 0.25–0.5 μM Mal3 indicates that the initial reversible binding of Mal3 has little effect on the sliding velocity. This is consistent with the lack of inhibition of the ATPase of Tea2 by high levels of Mal3 and by the ability of Mal3 and Tea2 to bind simultaneously to MTs (Fig. 6A, lane 12). The failure of high levels of Mal3 to inhibit sliding of conventional kinesin-1 also indicates that Mal3 does not produce road blocks when bound to the MT. This could be due either to the binding of Mal3 being so rapidly reversible that it is readily displaced by processive kinesin-1 dimers or because binding of Mal3 is not strongly competitive with binding of kinesin motor domains. The observed inhibition of monomeric DKH357 by Mal3 in Fig. 5A under conditions where the rate is limited by MT-stimulated ADP release suggests that the interaction may be at least partially competitive. But even in this case, the inhibition is not pronounced and may still be sufficiently reversible as to not influence the rate-limiting step during processive movement of a dimer of conventional kinesin-1.

The slow onset of inhibition of sliding by Mal3 and the preferential attachment of the inhibited axonemes to the surface at their plus ends suggest that these effects are due to some process that is subsequent to initial binding of Mal3 along the length of the axoneme. One possibility is that it is due to a time and concentration dependent accumulation of high local concentrations of Mal3 and possibly Tea2 at the plus end of the axoneme. Further work with fluorescently labeled Mal3 and Tea2 should be informative in this regard.

**REFERENCES**

Tea2 Activation by Mal3

Supplement: Tea2Movie1.mov

Analysis was performed by DIC microscopy on an Olympus XL71 microscope with a 60x 1.45 NA lens and 2.5x projection lens. An Argus20 was used to obtain a running average of 8 frames from the video output of a PicIII intensified video camera and images were captured at 4 seconds per frame. An out of focus background image was subtracted from each frame using Image J. The total elapsed time of the movie is 8 minutes and the view is 32 µm wide.

The movie begins with control movement in buffer alone for 2.5 minutes. The flow cell was then flushed with buffer containing 0.25 µM Mal3 during the black frames and the movement in the present of Mal3 was monitored for the remainder of the movie.

Two axonemes that are moving to the right were tracked over the whole period. After addition of Mal3, they both initially continued moving smoothly with a velocity that was slightly greater than before addition of Mal3. By the end of the movie, however, they had both stopped moving. The shift is usually not gradual, but abrupt as with the lower axoneme that moved at a high rate until it stopped moving just before the end of the movie. No further movement of this axoneme was observed at longer times. The upper axoneme exhibited pauses of varying lengths before it came to a final stop with only the trailing end still attached to the surface.
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doi: 10.1074/jbc.M413620200 originally published online January 23, 2005

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