Dissociation of Clathrin from Coated Vesicles by the Uncoating ATPase*

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The uncoating ATPase has been shown to dissociate clathrin from both clathrin-coated vesicles and synthetic clathrin baskets (Rothman, J. E., and Schmid, S. L. (1986) Cell 46, 5–9). In the present study, we investigated the mechanism of action of the uncoating ATPase using intact coated vesicles isolated from bovine brain. We observed an initial burst of uncoating followed by much slower steady-state uncoating. The initial burst of uncoating was essentially stoichiometric with each molecule of uncoating ATPase apparently binding to one leg of the clathrin triskelion. When the enzyme was preincubated with equimolar ADP, Pi, and ATP, rather than just ATP alone, both the initial burst and the slow steady-state uncoating were markedly inhibited, suggesting that the combination of ADP and Pi is a strong competitive inhibitor of ATP binding. However, kinetic studies suggested that ADP and Pi dissociates from the enzyme relatively rapidly unless clathrin is also bound to the enzyme. These results suggest that, after the uncoating ATPase rapidly removes a stoichiometric amount of clathrin while ATP is hydrolyzed at the active site, slow release of ADP and Pi from the resulting enzyme-clathrin-ADP-Pi complex limits the rate at which further uncoating occurs.

When cells are stressed by an increase in temperature, a number of different proteins, known collectively as the heat shock proteins, are selectively produced by the cell. One of the most prominent of these proteins is the 70-kDa protein, which has been observed in a wide variety of species from yeast to man (1, 2). It is now clear that, rather than being only a single 70-kDa protein, there is, in fact, a whole class of these proteins. For example, yeast has been shown to have at least eight different genes that code for 70-kDa proteins (3). Some of these proteins are only produced during heat shock while others are produced constitutively by the cell and are essential for yeast viability. In addition to having molecular masses of about 70 kDa, thus far all of the 70-kDa proteins that have been purified appear to bind ATP unusually tightly which has allowed them to be isolated with essentially complete purity on an ATP affinity column (4).

Although it is clear that the 70-kDa proteins play a role in both normal cellular processes and in the heat shock phenomenon, their specific functions in the cell are not yet understood. The first defined function observed for a 70-kDa heat shock protein was found serendipitously when it was noted that the clathrin uncoating ATPase, discovered by Rothman and his collaborators (5, 6), was a member of the '70-kDa class of proteins (7). In vivo studies have shown that during endocytosis the protein clathrin is stripped from clathrin-coated vesicles within minutes after they have budded off of the plasma membrane (8). Rothman and Schmid (9) found that the uncoating ATPase is able to strip clathrin from coated vesicles in vitro and suggested that the uncoating ATPase may cause clathrin dissociation in vivo in an ATP-dependent reaction.

This finding that the uncoating ATPase strips clathrin from coated vesicles in an ATP-dependent reaction along with the observation that the 70-kDa heat shock protein binds to the nucleolus in an ATP-dependent manner during heat shock led Pelham (4) to suggest that the 70-kDa proteins may have the general function of either disaggregating abnormal protein complexes during heat shock or preventing their aggregation in the first place. More generally, it has been suggested that the 70-kDa proteins may act as "molecular chaperones" (10), that is they may serve to keep proteins in a disaggregated state under conditions where they would otherwise form complexes. The recent observations (11, 12) that the 70-kDa proteins may be involved in the translocation of newly synthesized proteins across the membrane of the endoplasmic reticulum is consistent with this view. It has been suggested that the newly synthesized proteins must form a complex with a 70-kDa protein in order to assume the correct conformation for translocation.

If the 70-kDa proteins are indeed acting as molecular chaperones, it is important to understand their mechanism of action. From their extensive investigation into the mechanism of action of the uncoating ATPase, Rothman and Schmid (9, 13) suggested a rather complex mechanism of action for the uncoating ATPase involving two nucleotide-binding sites and two different sites of binding of the uncoating ATPase to clathrin. One of the reasons that they postulated such a complex mechanism was to explain their observation that the uncoating ATPase appeared to cause repeated cycles of uncoating rather than just removing a stoichiometric amount of clathrin in a single cycle of uncoating. Although they apparently observed an initial burst of ATP hydrolysis when the enzyme with bound ATP was first mixed with clathrin baskets, they did not observe a stoichiometric burst of clathrin release. Rather, the rate of clathrin release appeared to be nearly linear with time, with the enzyme repeatedly binding to the artificial clathrin baskets, releasing clathrin into solution, and then returning to the baskets to remove more clathrin molecules (6, 9). It is possible that these experiments were affected by the use of tritium-labeled artificial clathrin baskets. This substrate may be rather unstable under the

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conditions of the uncoating assays, as noted by Rothman and co-workers (6).

In the present study, we investigated the mechanism of action of the uncoating ATPase using intact coated vesicles isolated from bovine brain. Our results show that when the uncoating ATPase is first preincubated with ATP and then mixed with coated vesicles there is an initial burst of uncoating followed by much slower steady-state uncoating. The initial burst of uncoating is essentially stoichiometric with each enzyme apparently binding to one heavy chain of the clathrin triskelion. If both ADP and Pi are present in addition to ATP, the initial burst of uncoating is strongly inhibited as is the slow steady-state uncoating. However, kinetic studies suggest that ADP and Pi dissociate from the enzyme relatively rapidly unless clathrin is also bound to the enzyme. These data suggest a model where the enzyme with bound ATP interacts with the coated vesicles and then rapidly removes a stoichiometric amount of clathrin while ATP at the active site is hydrolyzed. This is followed by slow release of ADP and Pi from the resulting enzyme-clathrin-ADP-Pi complex; the rate of release limits the rate at which further uncoating can occur.

MATERIALS AND METHODS

Coated vesicles were made from bovine brain essentially according to the procedure of Nandi et al. (14). A 12% sucrose-D2O ultracentrifugation step (SW 28 rotor, 100,000 × g for 3 h) was used in the purification of the coated vesicles. The coated vesicles were usually stored at a clathrin concentration of ~15 pM.

The uncoating ATPase was prepared from bovine brain according to the procedure of Schlossman et al. (6) with the following modifications. After the brain was homogenized in buffer A (0.1 M Mes (pH 6.5), 0.1 mM EGTA, 0.5 mM MgC$_2$, and 3 mM Na$_2$) and spun (10,000 × g for 45 min), the supernatant was dialyzed overnight in buffer B (25 mM imidazole (pH 7.0), 1 mM dithiothreitol). After clarification of the dialysate, it was applied to a DEAE-Sephacel elution step provided a yellowish colored protein peak which was salt steps as described by Schlossman et al. (6). The 0.15 M NaCl elution step provided a protein band on SDS gels.

The enzyme stock solution was routinely made 1 mM in ATP after its concentration was determined. From 600 g of brain tissue, we routinely obtained 15–20 mg of purified uncoating ATPase. Protein concentrations were determined from absorbance using the following molecular weight and extinction coefficients: for clathrin, M$_r$ 620,000 and ε$_{280}$ = 1.2 (15); for uncoating ATPase, M$_r$ 70,000 and ε$_{280}$ = 6.2. The latter extinction coefficient was determined by measuring both the amino acid composition and absorbance of the same sample of uncoating ATPase.

The assays measuring the extent of clathrin dissociation by the uncoating ATPase were performed by using ultracentrifugation to separate the dissociated clathrin from the clathrin bound to the coated vesicles. In these assays, enzyme was mixed with coated vesicles in buffer C (pH 7.0) using either 1 or 5 mM ATP with the free magnesium concentration always maintained at 1 mM. The added volume of buffer A and coated vesicles (in buffer A) was maintained at 10% of the total volume of the reaction mixture. Unless otherwise indicated, the uncoating assays contained an ATP-regenerating system, consisting of 15 mM phosphocreatine and 30 units/ml creatine phosphokinase (Sigma). The reaction mixture was typically incubated for 30 min at 25 °C and then 0.5 ml of the mixture was spun for 10 min at 300,000 × g at 4 °C in the TL 100 centrifuge (Beckman). The top 80% of the supernatant was removed and run on SDS gels to quantify the clathrin in the supernatant.

The Coomassie Blue intensity of the protein bands on the SDS gels were quantified using the LKB ultracron XL laser densitometer. The SDS gels, which were 4–20% mini-gels (from Integrated Separation Systems), always contained a standard amount of clathrin. This clathrin standard was prepared either by 0.5 M Tris extraction of the coated vesicles at pH 7.0 (16) or by dialysis of the coated vesicles against 10 mM Tris (pH 8.5) (17) followed by centrifugation of the stripped vesicles. Preparation of column-purified clathrin was made from the 10 mM Tris-extracted clathrin on a Superose 6 column (18) using 0.5 M Tris (pH 8.0) as the elution buffer. By using the same extinction coefficient for clathrin with all these preparations, the 0.5 M Tris-clathrin and the 10 mM Tris-clathrin had 60 and 80% of the purity, respectively, of the column-purified clathrin. Although we made pure clathrin to calibrate the 10 mM Tris- and 0.5 M Tris-clathrins, we did not routinely use pure clathrin as our standard because we found that the pure clathrin was extremely unstable, unlike the former clathrin preparations. This instability appeared to be due to proteolysis. In all cases, the standard we used in calculating the concentration of dissociated clathrin was corrected back to the concentration of pure clathrin.

HPLC chromatography was used to analyze the breakdown of ATP in our solutions. The buffer used was 0.6 M sodium acetate, 0.075 M sodium phosphate (pH 7.4). The column used was Synchropak AX100 (Syn Chrom, Inc.) and the HPLC was the Hewlett Packard HP 1090 liquid chromatograph.

RESULTS

We began our study of the uncoating ATPase by examining its ability to dissociate clathrin from coated vesicles at 25 °C (pH 7.0) in the presence of MgATP and buffer C. The extent of clathrin dissociation was determined by first separating the dissociated and bound clathrin by centrifugation. Less than 10% of the free clathrin and less than 20% of the uncoating ATPase sedimented during centrifugation while more than 95% of the vesicles sedimented. Following centrifugation, the free clathrin was quantified by measuring the intensity of the clathrin band on SDS gels.

The heavily loaded gel in Fig. 1 shows our preparations of uncoating ATPase and coated vesicles. The uncoating ATPase (lane 2) was 95% pure, as determined by scanning the bands on the SDS gel. Fig. 24 shows the ability of the uncoating ATPase to remove clathrin from the coated vesicles. Fig. 24, lanes 1 and 2, shows that in the absence of the uncoating ATPase the vesicles were very stable under the conditions of the uncoating reaction. Even with prolonged incubation of the coated vesicles in buffer C, less than 5% of

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1 The abbreviations used are: Mes, 2-(N-morpholino)ethane-sulfonic acid; EGTA, [ethylenbis(oxycylenenitritilo)]tetraacetic acid; SDS, sodium dodecyl sulfate; HPLC, high pressure liquid chromatography.
The dissociation of clathrin from coated vesicles and quantitation of the dissociated clathrin. A, coated vesicles (0.32 

μM in clathrin) were incubated in buffer C for 30 min at 25 °C. This incubation was carried out in the presence of 1 mM MgATP and absence of uncoating ATPase (lane 1), in the presence of uncoating ATPase (1.0 μM) and absence of MgATP (lane 3), and in the presence of both uncoating ATPase (1.0 μM) and MgATP (lane 5). The coated vesicles were spun and the supernatant from the reaction mixture given in lanes 1, 3, and 5 are shown in lanes 2, 4, and 6, respectively. B, standard curve of relative absorbance of the clathrin protein heavy chain, obtained from scanning the Coomassie Blue intensity of a SDS gel containing clathrin of different concentrations (0.16–0.64 μM).

FIG. 2. The dissociation of clathrin from coated vesicles and quantitation of the dissociated clathrin. A, coated vesicles (0.32 

μM in clathrin) were incubated in buffer C for 30 min at 25 °C. This incubation was carried out in the presence of 1 mM MgATP and absence of uncoating ATPase (lane 1), in the presence of uncoating ATPase (1.0 μM) and absence of MgATP (lane 3), and in the presence of both uncoating ATPase (1.0 μM) and MgATP (lane 5). The coated vesicles were spun and the supernatant from the reaction mixture given in lanes 1, 3, and 5 are shown in lanes 2, 4, and 6, respectively. B, standard curve of relative absorbance of the clathrin protein heavy chain, obtained from scanning the Coomassie Blue intensity of a SDS gel containing clathrin of different concentrations (0.16–0.64 μM).

The clathrin was dissociated in the absence of the uncoating ATPase. Furthermore, as shown in lanes 3 and 4, in the presence of uncoating ATPase, there was no significant dissociation of clathrin in the absence of ATP. On the other hand, lanes 5 and 6 show that, in the presence of both uncoating ATPase and ATP, there was extensive dissociation of clathrin from the coated vesicles. Under the conditions of this experiment about 80% of the clathrin was dissociated from the coated vesicles by the uncoating ATPase, and almost all of the uncoating ATPase remained in the supernatant with the dissociated clathrin. To quantify the concentration of clathrin both in the coated vesicles and in the supernatant, we used a clathrin standard curve (Fig. 2B). The clathrin used in the standard was typically prepared either by high salt extraction with 0.5 M Tris (pH 7.0) (16) or dialysis against 10 mM Tris (pH 8.5) (17).

Fig. 3 shows the uncoating activity at two different enzyme concentrations at both 25 and 37 °C. The data suggest that there was an initial burst of uncoating followed by slow steady-state uncoating (rate = 6 × 10^{-5} s^{-1}). Both the magnitude of the initial burst of uncoating and the rate of the slow steady-state uncoating were proportional to the enzyme concentration. Increasing the temperature increases the steady-state rate of uncoating about 2-fold, but had little effect on the magnitude of the initial burst of uncoating. Note that we only measured the time course of uncoating for 1 h. After that, the ATP level was not maintained, and the level of ADP increased, suggesting that the creatine phosphate level was decreasing.

Fig. 4 shows the initial burst of uncoating as a function of enzyme concentration at 0.55 μM vesicles, 25 °C. The magnitude of the initial burst was determined by extrapolation of the steady-state rate of uncoating to zero time as shown in Fig. 3. The extent of clathrin dissociation from the coated vesicles showed a linear dependence on the uncoating ATPase concentration with about three molecules of uncoating ATPase dissociating one molecule of clathrin. This suggests that during the initial burst of uncoating one clathrin molecule is dissociated when one enzyme molecule binds to each of the three legs of clathrin.

In a preliminary report (19), we had suggested that the uncoating ATPase did not show steady-state uncoating, but only showed stoichiometric uncoating of the coated vesicles. Since these experiments were performed in the absence of the creatine kinase-creatine phosphate ATP-regenerating system, we tested the effect of this ATP-regenerating system on both the initial burst of uncoating and on the steady-state uncoating. Fig. 5 shows that the initial burst of uncoating is about the same in the presence and absence of the ATP-regenerating system, but that the slow steady-state uncoating only occurs when the ATP-regenerating system is present. These data suggest that the products of ATP hydrolysis inhibit the steady-state uncoating reaction. Note that most of the ATP hydrolysis in these experiments is not due to the uncoating ATPase itself, but rather to contaminated ATPases present in the coated vesicles. Under the conditions of the experiment shown in Fig. 5, HPLC analysis showed that about half of the 1 mM ATP initially present was hydrolyzed by the coated vesicles during the first 30 min of the reaction.

In these measurements of the initial clathrin burst, we always used enzyme that had been preincubated with 1 mM ATP. However, if the products of ATP hydrolysis are inhibiting the steady-state rate by decreasing the rate of release of ADP and P_i, then the presence of these products on the enzyme might be expected to inhibit the initial burst, as well as the burst.
as the steady-state rate. To determine the extent to which the products of ATP hydrolysis inhibit the uncoating reaction, we first preincubated the uncoating ATPase with varying amounts of either ADP or P_i, or both ADP and P_i, in the presence of 5 mM ATP. We then determined the magnitude of the initial burst of uncoating which occurred after mixing the uncoating ATPase with coated vesicles. This experiment was done in the absence of an ATP-regenerating system when we measured the inhibition by either ADP alone or both ADP and P_i, but in the presence of an ATP-regenerating system when we measured the inhibition by P_i alone.

Fig. 6 shows that preincubation of the uncoating ATPase with P_i in the presence of an ATP-regenerating system caused a significant inhibition of the initial clathrin burst. The slight inhibition that was observed is most likely due to both ADP and P_i, since there is slow production of P_i from the hydrolysis of ATP during the preincubation. However, the results were very different when ADP and P_i, with ATP, were preincubated with the uncoating ATPase. As shown in Fig. 6 (closed circles) there was strong inhibition of the uncoating reaction when the ATP and ADP and P_i concentrations were equal. This suggests that ADP and P_i, together act as a strong competitive inhibitor of ATP binding to the uncoating ATPase.

Since our results showed that the rapid initial burst of uncoating is followed by much slower steady-state uncoating, we were interested in whether the slow steady-state rate is caused by a slow release of ADP and P_i from the uncoating ATPase prior to the rebinding of ATP. To test this, we preincubated the uncoating ATPase with ADP and P_i overnight and then mixed the uncoating ATPase with coated vesicles in the presence of an ATP-regenerating system. The results (shown by open circles in Fig. 7) show that preincubation of the enzyme with the products of ATP hydrolysis caused only a slight decrease in the rate and magnitude of the initial clathrin burst (compare with dotted line in Fig. 7). Note that in this experiment the ATP-regenerating system removed the free ADP and P_i added with the uncoating ATPase almost instantaneously because of the very high concentrations of creatine kinase used in the reaction mixture. Therefore, these results indicate that the release of ADP and P_i from the uncoating ATPase does not account for the slow steady-state rate of uncoating observed after the initial burst.

Although the presence of ADP and P_i on the enzyme had only a slight effect on the initial burst of uncoating, it remained possible that if clathrin as well as ADP and P_i were bound to the enzyme the initial burst of uncoating would be strongly inhibited. To test this possibility, the uncoating reaction was carried out in the absence of an ATP-regenerating system and then the supernatant, which contained uncoating ATPase, stoichiometric clathrin, and ATP (of which, based on HPLC analysis, about 90% had been hydrolyzed to ADP and P_i), was mixed with fresh coated vesicles in the presence of an ATP-regenerating system. As shown by the triangles in Fig. 7, the burst of uncoating was about 80% inhibited (compare with dotted line). On the other hand, the
stead-state uncoating still occurred at about the same rate. These data suggest that the complex of uncoating ATPase-clathrin-ADP-Pi, which resulted from the first round of uncoating, decomposed at the slow steady-state rate when exposed to an ATP-regenerating system and fresh coated vesicles. This in turn suggests that product release from the enzyme-clathrin-ADP-Pi complex may be the rate-limiting step in the uncoating reaction.

**DISCUSSION**

This study has examined the ability of the uncoating ATPase to dissociate clathrin from clathrin-coated vesicles *in vitro*. Most of our results are in agreement with the findings of Rothman, Schmid, Bracil and co-workers (6, 8, 9, 10, 20–25) who first discovered and characterized the uncoating ATPase. Specifically, we confirmed that the uncoating ATPase dissociates clathrin from coated vesicles in an ATP-dependent reaction. However, we found that this uncoating reaction occurs in two phases: a rapid initial burst of uncoating occurs followed by a much slower steady-state rate of uncoating.

The occurrence of a rapid initial burst of uncoating prior to the slow steady-state uncoating suggests that, following incubation of the enzyme with ATP, the enzyme is primed to dissociate clathrin rapidly from the coated vesicles. The initial burst of uncoating appears to require three enzyme molecules to dissociate one clathrin molecule. Clathrin molecules are triskelions, that is each molecule is composed of three legs (24). Therefore the stoichiometry of the initial burst fits with the idea that one enzyme molecule must bind to each of the three clathrin legs to dissociate a clathrin molecule from a coated vesicle.

Interestingly, the plot of the initial burst of uncoating *versus* enzyme concentration is linear even at low ratios of enzyme to clathrin. This would not be expected if the uncoating ATPase bound non-cooperatively to the clathrin, since at low ratios of enzyme to clathrin presumably the enzyme would scatter around various clathrin molecules and would be unable to dissociate any of them. Therefore the observation that three enzyme molecules dissociate one clathrin molecule, even at low ratios of enzyme to clathrin, suggests that the uncoating ATPase binds cooperatively to the clathrin triskelion, although much more work will be necessary to prove that this is indeed the case.

Following the initial burst of uncoating, we observed a slow steady-state uncoating reaction which occurred only in the presence of an ATP-regenerating system. We previously missed the occurrence of this slow steady-state uncoating reaction (19) because we did not have an ATP-regenerating system present in our reaction mixture. Since coated vesicles themselves have a high intrinsic ATPase activity, ATP is hydrolyzed quite rapidly in our reaction mixture. In addition, a relatively low level of ADP and P₈ has a strong inhibitory effect, with almost complete inhibition of the uncoating reaction occurring when equimolar ATP and ADP + P₈ are present in the reaction mixture. Interestingly, both ADP and P₈ must be present to obtain this inhibition, because ADP alone has little or no effect in the presence of equimolar ATP. These results indicate that the combination of ADP and P₈ binds with very strong affinity to the uncoating ATPase, possibly with even greater affinity than ATP.

The observation that an initial burst of uncoating precedes the slow steady-state uncoating reaction implies that a slow step in the uncoating reaction must occur after the initial burst of uncoating is complete. Since ADP and P₈ bind so strongly to the uncoating ATPase, it seemed possible that this slow step might involve the release of the products of ATP hydrolysis from the enzyme. If this were the case, preincubation of the enzyme with ADP and P₈ should prevent the initial burst of uncoating from occurring. However, when we pre-equilibrated the enzyme with ADP and P₈, we found that the initial burst of uncoating still occurred, but with a slightly slower rate. Therefore, although ADP and P₈ bind very tightly to the enzyme, they apparently dissociate from the enzyme quite rapidly.

Since dissociation of ADP and P₈ alone did not prevent the initial burst of uncoating, we tested whether the initial burst of uncoating was strongly inhibited, but not the steady-state uncoating. These data suggest that product release from the uncoating ATPase-clathrin-ADP-P₈ complex may be the rate-limiting step in the uncoating ATPase reaction. On this basis, we suggest the following model for the action of the uncoating ATPase in which Cl₃bound is the clathrin triskelion bound to the coated vesicles, Cl₃dissociated is the dissociated triskelion, and UC is the uncoating ATPase.

In this model, the enzyme with bound ATP first interacts with the coated vesicles and removes a stoichiometric amount of clathrin while ATP at the active site is hydrolyzed. This part of the cycle occurs rapidly and explains the initial burst of uncoating that occurs when the enzyme is preincubated with ATP and then mixed with coated vesicles. Following the initial burst of uncoating, ADP and P₈ are slowly released from the resulting enzyme-clathrin-ADP-P₈ complex which accounts for the slow steady-state uncoating reaction which follows the initial burst of uncoating. ATP then dissociates the bound clathrin allowing further cycles of uncoating to occur.

One aspect of the uncoating process which our model does not consider is what occurs at low pH or high Mg concentration where the coated vesicles activate the ATPase activity of the uncoating ATPase but no uncoating occurs (23). This activation might be consistent with our model if the rate of ADP and P₈ release from the uncoating ATPase-clathrin complex were actually faster when the clathrin remained attached to the coated vesicles than when it dissociated. We are currently carrying out ATPase studies to determine the relationship between the ATPase activity of the uncoating ATPase and the uncoating process. We are also testing whether an uncoating ATPase-clathrin-ADP-P₈ complex is, indeed, present during the normal steady-state uncoating reaction as our model predicts.

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