Effect of ATP on the Kinetics of Microtubule Assembly*

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We investigated the role of ATP in the assembly of microtubules. Tubulin, prepared by chromatography on DEAE-cellulose, was nearly devoid of nucleoside diphosphokinase activity. ATP induced assembly in such preparations for a single assembly/disassembly cycle; then further assembly could not be induced by ATP unless the system was supplemented with additional GTP. This suggests that the E-site must contain GTP for polymerization and ATP interacts at a different site on tubulin.

Although tubulin can be assembled into microtubules in 1.0 mM GTP, the inclusion of 0.2 mM ATP along with the GTP increases the rate and extent of assembly. The enhancement increased with increasing ATP concentrations. The inclusion of 0.2 mM ATP reduced the critical concentration for tubulin assembly from 1.5 to 0.9 mg/ml. Analysis of assembly rate versus protein concentration suggested that ATP also affects nucleation.

Aggregates of tubulin rings formed by warming tubulin in the presence of 1.0 mM ATP and 5.0 mM Mg²⁺ were capable of initiating assembly in a solution of tubulin which was not able to polymerize. Furthermore, the extent of microtubule formation was dependent on the concentration of aggregated rings added to the solution.

We propose that ATP interacts with tubulin at a binding site that is distinct from the N- and E-sites that bind GTP. A function of ATP binding is to stimulate the formation of tubulin rings as nucleation centers for polymerization.

Nucleotides play a critical role in the assembly of microtubules (1–5). The tubulin dimer possesses two distinct guanine nucleotide binding sites (6). At one site (N-site), GTP is bound nonexchangeably; the function of this site remains obscure. Considerably more is known about the E-site where 1 mol of bound guanine nucleotide can exchange with exogenous nucleotides. It is firmly established that GTP bound at the E-site is hydrolyzed to GDP during the assembly of microtubules (4, 7, 8). GDP bound at the E-site appears to support microtubule formation of aggregates of tubulin rings (18) which are thought to function as nucleation centers in microtubule polymerization.

The role of ATP is not clearly defined. ATP can promote microtubule assembly (12, 13) through the action of an NDP kinase¹ which can phosphorylate tubulin-associated GDP at the expense of the γ-phosphate of ATP (4, 14, 15). The NDP kinase activity is not inherent to the tubulin and can be removed from the microtubule protein by ion-exchange chromatography (4, 15–17). Tubulin has been reported to be incapable of binding ATP (3, 14) although there is more recent evidence to the contrary (18).

There is evidence that ATP functions in a regulatory capacity in microtubule assembly. ATP was shown to alter the assembly characteristics and stability of microtubules (15–21). Increased rates of microtubule assembly and disassembly at steady state occurred in the presence of physiological concentration of ATP (20). We reported that ATP induces the formation of aggregates of tubulin rings (18) which are thought to function as nucleation centers in microtubule polymerization.

There are several models for microtubule assembly which incorporate ringlike aggregates of tubulin as nucleation sites (22–24). Kinetic studies (25, 26) are consistent with a condensation-polymerization mechanism where there is an initiation phase (lag) followed by an elongation phase. Electron microscopy reveals that rings, initially present, disappear early in the polymerization process (23). Rings could uncoil to become protofilaments which aggregate laterally to form a microtubule. Bryan, however, claims that rings are not obligatory intermediates in microtubule formation (26) although they might have appeared transiently during the assembly pathway (27) and, therefore, overlooked in his analysis.

We previously showed that, at elevated Mg²⁺ concentrations, ATP can induce the formation of tubulin rings (18). We now report that ATP can promote microtubule formation in the absence of NDP kinase activity and that it can significantly alter the assembly kinetics. We propose that ATP effects nucleation and provides further support for the existence of an ATP binding site on tubulin independent of the GTP binding sites.

MATERIALS AND METHODS

Tubulin was prepared from fresh bovine brains using DEAE-cellulose (Whatman DE52) chromatography according to Weisenberg et al. (6). This was performed either by the batch procedure as described or on a DEAE column (5.0 × 4.0 cm) using a step elution of 1 column volume of 0.25 mM NaCl in PM buffer (10 mM phosphate, 5 mM Mg²⁺, pH 7.0) and then eluting the tubulin with 1 column volume of 0.5 mM NaCl in PM buffer. The latter procedure yielded a product with more consistently low NDP kinase activity. Tubulin was stored at −20 °C in PM buffer containing 1.0 mM succrose. All buffers used in the purification also contained 0.1 mM GTP. For assembly experiments, tubulin was dialyzed overnight against approximately 400 volumes of assembly buffer (10 mM 2-(N-morpholino)propanesulfonic acid, 1.6 mM ethylene glycol bis(β-aminoethyl ether)-N,N,N',N'-tetraacetic acid, 0.6 mM MgCl₂, 0.5 mM KCl, 0.02% NaN₃, pH 7.0).

¹The abbreviations used are: NDP kinase, nucleoside diphosphokinase; PM, phosphate-magnesium; HPLC, high pressure liquid chromatography; AMP-PNP, adenylyl imidodiphosphate.
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3.4 M glycerol, pH 6.4) containing the appropriate concentration of Mg++. For assembly, the indicated nucleotides were added and then the sample was warmed to 37 °C in a thermostatically controlled cuvette chamber of a Cary 15 recording spectrophotometer. The increase in turbidity was monitored at 350 nm.

For purposes of comparison, tubulin was prepared by repeated cycles of assembly and disassembly according to the method of Sheland et al. (28). The procedure was performed both with and without (29) glycerol. The products were stored at -80 °C.

Sodium dodecyl sulfate-polyacrylamide gel electrophoresis was performed with vertical slab gels containing 7.5% acrylamide following the procedure of Laemmli (30).

NDP kinase activity was determined by direct measurement of GTP produced from GDP and ATP with the use of high pressure liquid chromatography. PM buffer was made 0.5 mM in ATP and GDP. Protein was added and the mixture was incubated for 30 min at 37 °C. (The assay was shown to be linear with time and protein concentration over the range used.) The reaction was stopped and protein removal was facilitated by the addition of 100% trichloroacetic acid to a final concentration of 5% followed by centrifugation. Nucleotides were separated and quantified on a reversed phase column (Altex, Ultrasphere-ODS, 25 cm) equilibrated with 5 mM tetrahydroammonium phosphate (Altex), 30 mM KH2PO4, 4% CH3CN. Nucleoside di- and triphosphates are well separated with a linear gradient of 20-35% CH3CN. NDP kinase activity was also determined by measuring ADP production in a coupled enzyme assay (31).

All nucleotides were obtained from Sigma (>98% pure). Protein concentration was determined by the method of Lowry et al. (32). Electron microscopy was performed in a Philips EM301. Samples were fixed with 2-4% glutaraldehyde, placed onto carbon-coated grids, and negatively stained with 1.5% uranyl acetate.

RESULTS

Microtubule protein prepared by cycles of assembly and disassembly contains approximately 15-25% non-tubulin proteins (33, 34). When tubulin is purified by chromatography on DEAE-cellulose, most of the microtubule accessory proteins are removed and the final product is greater than 98% tubulin. Fig. 1 demonstrates the relative purity of the ion-exchange-purified tubulin used in the present experiments (lane 1) compared to tubulin prepared by two cycles of assembly/disassembly in the presence and absence of glycerol (lanes 2 and 3, respectively). Pointed out in the figure are the protein bands which correspond to the high molecular weight and τ proteins. It is clear that the tubulin preparation used here was virtually free of contaminating proteins.

A protein that is found in many tubulin preparations is an NDP kinase. This activity can be removed from tubulin by chromatography on ion-exchange resins (4, 15, 17). That the NDP kinase was depleted in our preparation is illustrated in Table I. NDP kinase activity was determined by direct analysis with HPLC and by the coupled enzyme assay for tubulin fractions prepared by DEAE chromatography and by the cycle procedure. It is clear that the NDP kinase activity was substantially reduced in our preparations compared to once and twice cyclcd tubulin. It is interesting to note that when assayed by HPLC, the activity in cycled tubulin was 20% or less of the value determined by the coupled enzyme assay. Whereas the latter assay will respond to various kinases and phosphatases, the HPLC assay is specific for the formation of GTP from ATP and GDP. The discrepancy may indicate that several kinases and/or phosphatases accompany tubulin during the purification.

The level of NDP kinase remaining in our preparation was not sufficient for ATP-supported microtubule assembly. Fig. 2 illustrates what happened when tubulin was warmed in the presence of 1 mM ATP as the only added nucleotide. There was the usual turbidity increase resulting from microtubule formation as verified by electron microscopy. It is important to note that no assembly occurred without added ATP. When the sample was cooled to disassemble the microtubules and then rewarmed to 37 °C, no further assembly took place. Competency to form microtubules was returned when this sample was supplemented with GTP. We interpret this as follows. Following dialysis, the tubulin contained a small amount of bound GTP. ATP was able to induce microtubule assembly with the subsequent hydrolysis of GTP. Once the GTP was depleted, no further assembly could occur until additional GTP was added. In tubulin preparations containing sufficient NDP kinase (i.e. cycle-purified tubulin), ATP alone

![Fig. 1. Sodium dodecyl sulfate gel electrophoresis of tubulin preparations. Tubulin, prepared by: 1, chromatography on DEAE-cellulose, 2, cycle procedure with glycerol, and 3, cycle procedure without glycerol, was run on an sodium dodecyl sulfate-polyacrylamide gel (7.5%) to display the impurities. HMWs, high molecular weight proteins.](http://www.jbc.org/)
can lead to several cycles of assembly through the regeneration of GTP from GDP.

Two additional points were revealed by this experiment. First, the addition of ATP was capable of inducing assembly in a system where this would not otherwise have occurred. Second, it is apparent that in order for ATP to support assembly, the E-site must contain GTP which then becomes hydrolyzed. Therefore, ATP cannot be interacting with tubulin at this site.

The addition of small amounts of ATP to tubulin in the presence of GTP increased the rate and extent of microtubule assembly. Fig. 3 shows the polymerization profile of two tubulin samples that contained 1.0 mM GTP and which were identical in every other respect except that one had ATP added to 0.2 mM. The ATP was added as the magnesium salt so the total free Mg$^{2+}$ level should have remained constant. The sample with ATP assembled with a shorter lag, a faster elongation rate, and a higher plateau value. By electron microscopy, the two preparations were indistinguishable, showing mostly microtubules whether ATP was added or not. When the experiment was repeated keeping the total free MF level should have remained constant.

An increase in the concentration of ATP resulted in a proportional increase in the level of assembly. This is illustrated in Fig. 4 in which the plateau level of turbidity is plotted as a function of ATP addition. To demonstrate that this effect is not a result of added Mg$^{2+}$ or the increased level of nucleotide triphosphate, the same experiment was performed in which comparable amounts of Mg$^{2+}$ or GTP were added. The addition of these factors had little or no effect (Fig. 4), confirming that ATP was responsible for the enhancement of polymerization.

The presence of ATP resulted in a reduction of the critical concentration for microtubule assembly. Two tubulin samples were prepared in assembly buffer (with 1.0 mM GTP) and ATP was added to one to a final concentration of 0.2 mM. These samples were polymerized by warming to 37 °C until they reached maximum turbidity, and then they were depolymerized by cooling to 4 °C. Dilution and repolymerization were carried out for several cycles. Fig. 5 presents the final change in turbidity as a function of the tubulin concentration. Extrapolation to zero turbidity change yields the critical concentration ($C_c$) (38). Including ATP in the assembly buffer decreased the $C_c$ from 1.5 to 0.9 mg/ml.

In Fig. 6, the rate of assembly is plotted as a function of the concentration of added ATP. Two curves are presented corresponding to assembly in 1.0 mM GTP or 1.0 mM GTP plus 0.2 mM ATP. The data obtained from assembly in GTP alone fall on a straight line while those with added ATP are distinctly nonlinear. The significance of this will be discussed later.

To test whether ATP hydrolysis was required for the enhancement of assembly, a comparison was made of the assembly kinetics for samples containing 1.0 mM GTP or 1.0 mM GTP plus 0.2 mM AMP-PNP, a nonhydrolyzable ATP analog. The polymerization curves were essentially the same, or perhaps in the sample containing the AMP-PNP slightly slower
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FIG. 5. The effect of ATP on the critical concentration for microtubule formation. The extent of tubulin assembly in 1.0 mM GTP was measured with (○) and without (●) 0.2 mM ATP at the stated concentrations as described in the text.

FIG. 6. Dependence of assembly rate on tubulin concentrations. Tubulin samples in assembly buffer and 1.0 mM GTP were polymerized with 0.2 mM ATP (●) and without (○) ATP. Assembly was monitored as the change in turbidity at 350 nm. Initial rates were determined as the slope of the best line drawn through the linear portion of the assembly curves.

FIG. 7. Aggregated rings of tubulin are capable of initiating microtubule formation. Tubulin was prepared in assembly buffer with 2.0 mM Mg$^{2+}$ and 1.0 mM GTP such that polymerization would not occur. At the point indicated, various amounts of preformed aggregated rings prepared by assembly in 1.0 mM ATP and 5.0 mM Mg$^{2+}$ were added. Inset: the initial rate of assembly was plotted against the amount of the rings fraction added.

and less (by ~5%) extensive assembly was observed. When this analog was added to the level of 1.0 mM with 5.0 mM Mg$^{2+}$, conditions which lead to aggregated ring formation in the presence of ATP (18), no turbidity developed upon warming to 37 °C and no ring formation was observed by electron microscopy.

ATP induces the formation of aggregates of tubulin rings which are believed to function as nucleation centers in tubulin polymerization. If these are true nucleation sites, their addition to a solution of tubulin ought to initiate microtubule formation. Tubulin was prepared in assembly buffer with GTP. The protein concentration and Mg$^{2+}$ level were arranged such that spontaneous assembly could not occur at 37 °C, or if it did, it would do so at a very slow rate. To such preparations of tubulin, aliquots of preformed aggregated rings, prepared as described (18), were added at the times indicated in Fig. 7. The increase in turbidity indicated that the formation of microtubules resulted from this addition. Furthermore, the rate of assembly was dependent upon the concentration of rings (inset). A control experiment demonstrated that the small amount of ATP which was added with the ring fraction was not responsible for the initiation of assembly.

DISCUSSION

ATP as the only added nucleotide was capable of inducing microtubule assembly through a mechanism that does not involve transphosphorylation. The experiment described in Fig. 2 demonstrated that there was not sufficient NDP kinase present to support assembly and that ATP appears to interact at a site independent of the exchangeable GTP site.

That ATP does indeed stimulate the assembly rate is borne out by comparison of assembly kinetics in the presence and absence of a small amount of ATP. With the GTP level at 1.0 mM (well in excess of tubulin), and ATP at 0.2 mM, the rate and extent of polymerization were dramatically increased over those of an identical sample of tubulin with GTP but without ATP. Electron microscopy proved that there was extensive microtubule formation in both cases under the conditions used. A critical factor here was the Mg$^{2+}$ concentration of 2.5 mM. We previously described (18) a similar enhancement of tubulin assembly in 1.0 mM ATP compared to 1.0 mM GTP, when both samples were at 5.0 mM Mg$^{2+}$. At this level of Mg$^{2+}$, the polymers formed with ATP were extensively aggregated tubulin rings whereas polymerization with GTP produced only microtubules. It is clear that the Mg$^{2+}$ concentration is an important factor in the microtubule assembly process. ATP may be acting to favor the formation of tubulin rings which are forced to aggregate by high Mg$^{2+}$ levels, and are thereby prevented from proceeding onto the further stages in the assembly of microtubules. If rings are indeed structures for the nucleation of microtubule formation, an influence of ATP on ring formation would be consistent with the shorter lag time and increased assembly rate which were observed when a small amount of ATP was added.

Microtubule formation has been described as a nucleated
condensation mechanism (25, 35) involving a critical concentration below which net polymerization cannot occur. It is supposed that nuclei form first, and then tubulin dimers add onto the nuclei in an elongation reaction. The theoretical treatment of such a mechanism was developed by Oosawa and Higashi (36) to describe the polymerization of actin. In this treatment, the critical concentration \( C_c \) is seen to be the inverse of the equilibrium association constant \( (K_p) \) of the polymer. Thus

\[
C_c = \frac{1}{K_p} = k_{+1}/k_{-1} \tag{1}
\]

where \( k_{+1} \) and \( k_{-1} \) are rate constants for depolymerization and polymerization. Numerous factors including microtubule accessory proteins, have been reported to influence the critical concentration (24, 37, 38), and we show here that small amounts of ATP reduce \( C_c \). The latter observation implies that ATP increases \( k_{+1} \), the rate of addition of tubulin dimers onto the ends of microtubules, or it decreases \( k_{-1} \), the rate of release of dimers, or both.

As illustrated in Fig. 6, there is evidence that ATP affects nucleation as well as the critical concentration. Gaskin et al. (25) and others, using preparations of microtubule proteins that contained accessory proteins, reported that microtubule assembly exhibited mixed kinetics of an order only slightly higher than first. For several reasons (25, 38), it was concluded that the kinetics predominantly reflects a rate-limiting step in the nucleation process. As shown in Fig. 6, in the absence of accessory proteins and ATP, the dependence of assembly rate on tubulin concentration was strictly first order (above the \( C_c \)). Apparently, this strictly first order dependence represents some step in nucleation that is not only predominant in the kinetics, but absolutely rate-limiting. This rate limitation, which presumably involves the addition of a tubulin dimer to some intermediate structure, was at least partially relieved by ATP. This was demonstrated by the fact that assembly rates in the presence of ATP were higher and the kinetics was greater than first order (nonlinear curve in Fig. 6), just as it is when accessory proteins are present. Therefore, ATP enhances nucleation, in addition to reducing the critical concentration.

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We demonstrated that ATP interacts directly with tubulin and is responsible for changes in the assembly characteristics. Although the effects of ATP were clear, the mechanism by which ATP influences polymerization remained to be studied. The first question to be addressed was whether ATP hydrolysis is involved. Consistent with the requirement for hydrolysis was the finding that the nonhydrolyzable ATP analog, AMP-PNP, had no effect on the assembly kinetics. Also, this analog would not induce the formation of aggregated rings as did ATP. While these results would be necessary to demonstrate ATP hydrolysis, they are not sufficient to prove the case. Therefore, we looked for the release of inorganic phosphate using \([\gamma^{32}P]ATP\). No hydrolysis was detected over the time necessary for microtubule formation to reach steady state. The fact that AMP-PNP was unable to substitute for ATP can be reconciled to the lack of hydrolysis if ATP is bound with a precise and crucial molecular fit and the difference in structure of the analog prevents such binding.

For some time, tubulin rings have been postulated to be nucleation centers for microtubule formation. Indeed, aggregated tubulin rings induced with ATP proved capable of initiating assembly in a solution of tubulin which otherwise would not have polymerized. A crucial point to be emphasized is that while the Mg\(^{2+}\) concentration was 5.0 mM during the formation of the ring aggregates, it was diluted to 2.5 mM when the aliquot of aggregated rings was added to the tubulin solution. Microtubule formation resulted from this addition. When the Mg\(^{2+}\) concentration was maintained at 5.0 mM in parallel experiments, assembly was aborted by the aggregation of the rings.

Fig. 8 presents a scheme for the assembly of tubulin into microtubules incorporating the interaction of ATP at a third nucleotide binding site. We also illustrate some aspects of the role of magnesium in tubulin polymer formation. Since little information is available on the role of the N-site, GTP was simply placed at this site in every case. When GDP is on the E-site, no polymer formation can occur. With GTP at the E-site and ATP bound at the third site in the presence of 5 mM

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\[^2\] J. K. Zabrecky and R. D. Cole, unpublished results.
Mg$^{2+}$, warming to 37 °C results in the formation of aggregated tubulin rings (18). Tubulin is apparently locked in this polymeric form and prevented from entering the next stage in the formation of microtubules. When the magnesium concentration is lowered, these aggregates break down and the individual rings become nucleation centers for microtubule assembly. With the same nucleotide configuration but at lower Mg$^{2+}$ concentration (2 mM), individual rings are formed and without the hindrance of aggregation they initiate microtubule formation with subsequent E-site GTP hydrolysis. The last line of Fig. 8 indicates that there is not an absolute requirement for ATP, at least not under the conditions used here. In the absence of ATP, microtubule formation proceeds through the formation of rings as intermediates with E-site GTP hydrolysis, but with ATP bound to the tubulin, ring formation and microtubule assembly are speeded and the incorporation of tubulin into microtubules is more extensive.

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REFERENCES

1. Jacobs, M. (1979) in Microtubules (Roberts, K., and Hyams, J., eds) Chapt. 5, pp. 255–277, Academic Press, New York
2. Weisenberg, R. C. (1972) Science 177, 1054-1055
3. Jacobs, M., Smith, H., and Taylor, E. W. (1974) J. Mol. Biol. 89, 455-468
4. Penningroth, S. M., and Kirschner, M. W. (1977) J. Mol. Biol. 115, 643–673
5. Weisenberg, R. C., Deery, W. J., and Dickinson, P. J. (1976) Biochemistry 15, 4248–4254
6. Weisenberg, R. C., Borisy, G. G., and Taylor, E. W. (1968) Biochemistry 7, 4496-4479
7. Kobayashi, T. (1975) J. Biochem. (Tokyo) 77, 1193-1197
8. David-Pfeuty, T., Erickson, H. P., and Pantaloni, D. (1977) Proc. Natl. Acad. Sci. U. S. A. 74, 5372-5376
9. Carlier, M., and Pantaloni, D. (1978) Biochemistry 17, 1908-1915
10. Jameson, L., and Caplow, M. (1980) J. Biol. Chem. 255, 2284–2292
11. Carlier, M., and Pantaloni, D. (1981) Biochemistry 20, 1918-1924
12. Olmsted, J. B., and Borisy, G. G. (1973) Biochemistry 12, 4282–4289
13. Borisy, G. G., Olmsted, J. B., Marcum, J. M., and Allen, C. (1974) Fed. Proc. 33, 167–174
14. Jacobs, M., and Caplow, M. (1976) Biochem. Biophys. Res. Commun. 68, 127–135
15. Zeeberg, B., and Caplow, M. (1978) J. Biol. Chem. 253, 1984–1990
16. Jacobs, M. (1975) Ann. N. Y. Acad. Sci. 253, 562–572
17. Jacobs, M., and Huitorel, P. (1979) Eur. J. Biochem. 99, 613–622
18. Zabrecky, J. R., and Cole, R. D. (1980) J. Biol. Chem. 255, 11981–11985
19. Selkoe, D. J. (1979) Brain Res. 173, 382–386
20. Margolis, R. L., and Wilson, L. (1979) Cell 18, 673–679
21. Kamagai, H., Nishida, E., and Sakai, H. (1979) J. Biochem. (Tokyo) 85, 495–502
22. Erickson, H. P. (1974) J. Supramol. Struct. 2, 393–411
23. Kirschner, M. W., Honig, L. S., and Williams, R. C. (1975) J. Mol. Biol. 90, 253–276
24. Borisy, G. G., Johnson, K. A., and Marcum, J. M. (1976) in Cell Motility (Goldman, R., Pollard, T. D., and Rosenbaum, J. L., eds) Book C, pp. 1093–1108, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY
25. Gaskin, F., Cantor, C. R., and Shelanski, M. L. (1974) J. Mol. Biol. 89, 737–758
26. Bryan, J. (1976) J. Cell Biol. 71, 749–767
27. Kirschner, M. W. (1976) Int. Rev. Cytol. 54, 1–71
28. Shelanski, M. L., Gaskin, F., and Cantor, C. R. (1973) Proc. Natl. Acad. Sci. U. S. A. 70, 765–768
29. Borisy, G. G., Marcum, J. M., Olsted, J. B., Murphy, D. B., and Johnson, K. A. (1975) Ann. N. Y. Acad. Sci. 253, 107–132
30. Laemmli, U. K. (1970) Nature (Lond.) 227, 680–685
31. Parks, R. E., Jr., and Agarwal, R. P. (1973) in The Enzymes (Boyer, P. D., ed) third ed, Vol. 8, pp. 307–333, Academic Press, New York
32. Lowry, O. H., Rosebrough, N. J., Farr, A. L., and Randall, R. J. (1951) J. Biol. Chem. 193, 265–275
33. Kirschner, M. W. (1978) Int. Rev. Cytol. 54, 1–71
34. Murphy, D. B., and Borisy, G. G. (1975) Proc. Natl. Acad. Sci. U. S. A. 72, 2696–2700
35. Johnson, K. A., and Borisy, G. G. (1977) J. Mol. Biol. 117, 1–31
36. Oosawa, F., and Higashi, S. (1967) Prog. Theor. Biol. 1, 28–164
37. Lee, J. C., and Timasheff, S. N. (1977) Biochemistry 16, 1754–1764
38. Lee, J. C., Tweedy, N., and Timasheff, S. N. (1978) Biochemistry 17, 2783–2790
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