Effect of F-Actin upon the Binding of ADP to Myosin and Its Fragments*

Margery C. Beinfeld† and Anthony N. Martonosi§

From the Department of Biochemistry, St. Louis University School of Medicine, St. Louis, Missouri 63104

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The effect of F-actin upon the binding of ADP to rabbit skeletal muscle myosin, heavy meromyosin, and subfragment 1 was studied by equilibrium dialysis, ultracentrifuge transport, and light scattering techniques. Both myosin and H-meromyosin (HMM) bind a maximum of approximately 1.6 mol of ADP/mol of protein, while S-1 binds approximately 0.9 mol of ADP/mol of protein. The affinity for ADP of all three preparations was similar at a given ionic strength (approximately $10^{-9} \text{ M}^{-1}$ at 0.05 M KCl) and decreased with increasing ionic strength. Under conditions similar to those used for the measurement of ADP binding, the binding sites of myosin, HMM, and subfragment 1 (S-1) are saturated with actin at molar ratios of 2, 2, and 1 mol of actin monomer/mol of protein, respectively, as determined by light scattering, ultracentrifuge transport, and in the case of myosin by ATPase measurements. F-actin was found to inhibit ADP binding, but even at an actin concentration at least twice that required for saturation of myosin, HMM, or S-1, significant ADP binding remained. This ADP binding was inhibited by $10^{-4}$ M pyrophosphate. The observations are consistent with the formation of an actomyosin-ADP complex in which actin and ADP are bound to myosin at distinct but interacting sites.

During the contraction cycle, binding of ATP dissociates actomyosin followed by ATP hydrolysis and the formation of a myosin product complex (2). When the myosin head interacts with a new actin molecule, the products of the reaction are released from the active site and the crossbridges develop tension (2). In agreement with this scheme actomyosin in solution is readily dissociated by ATP (3), and F-actin decreases the affinity of ADP (4). PP, (5, 6), and ATP (7) for myosin. As 2 mol of actin (8), ADP (4, 9), ATP (7, 10), and PP, (6, 11) are bound per mol of myosin, each of the myosin heads is likely to contain one actin and one closely related nucleotide binding site (6, 8, 12).

The structural and functional identity of the two sets of nucleotide and actin binding sites is being debated. Scatchard plots of ADP interaction in the presence of Mg$^{2+}$ are usually interpreted in terms of a single set of noninteracting sites. Differences between the two nucleotide binding sites were inferred from the ultraviolet difference spectra of myosin induced by ATP, ADP, or PP, (14) and from the stochiometry of the rapid initial burst of ATP hydrolysis (14, 15). Indirect indications of cooperativity between the two sets of sites came from kinetic data (16) and from electron spin resonance measurements (17).

We have reported earlier that the binding of ADP to myosin is inhibited by actin (4). Surprisingly, even at actin to myosin ratios higher than that required for the saturation of myosin, significant ADP binding remained. As the precise relationship of the two ADP binding sites of myosin to the actin binding would have significant implications upon the mechanism of crossbridge movement during muscle contraction, the effect of actin upon the ADP binding of myosin was investigated in detail.

**EXPERIMENTAL PROCEDURES**

Myosin was isolated from white rabbit skeletal muscle as described earlier (4) and fractionated with (NH$_4$)$_2$SO$_4$, according to Kielley and Bradley (18). The protein fraction obtained between 40 and 50% (NH$_4$)$_2$SO$_4$ saturation was collected and stored at $-20^\circ$ as an (NH$_4$)$_2$SO$_4$ paste or after dialysis for the removal of (NH$_4$)$_2$SO$_4$ in a solution of 0.3 M KCl/50% glycerol, pH 7.5. The results obtained with myosins stored under either conditions were identical. The ATPase activity was monitored periodically and all preparations were used within 2½ months.

Heavy meromyosin (HMM)$^*$ was prepared by the method of Lowey

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†Recipient of a postdoctoral research fellowship from the Missouri Heart Association.

‡To whom all correspondence should be sent.

$^*$The abbreviations used are: HMM or H-meromyosin, heavy meromyosin; S-1, subfragment 1.
and Cohen (19). Fresh preparations of heavy meromyosin were either used within 1 week or immediately lyophilized in sucrose according to Yount and Koshland (20) and stored at -20°.

HMM S-1 fragments were prepared according to Young et al. (21) by cryptic digestion of heavy meromyosin and separation of the fragments by gel filtration through a Sephadex G-200 column. The fractions containing the S-1 fragment were pooled and concentrated by precipitation from a 60% saturated (NH₄)₂SO₄ solution. The precipitate was dissolved in 0.05 M KCl and 0.1 M Tris-HCl, pH 7.6, dialyzed against the same solution to remove (NH₄)₂SO₄ and reapplied to the column for final purification. The freshly prepared S-1 fractions were used within 1 week. In some cases S-1 was prepared according to Margossian and Lowery (8). The data obtained with the two types of preparations were similar.

Actin was prepared from acetone-dried muscle powder as described earlier (22). After 3 cycles of polymerization and depolymerization the actin was stored in the G-form in 2 x 10⁻⁵ M ATP, pH 7.8, at 4° and used within 1 week after preparation. Before use the actin was polymerized with 0.1 M KCl and the unbound nucleotides were removed either by treatment with Dowex 1-X2-100 resin (23) or by extensive dialysis against three changes of 0.1 M KCl/5 mM imidazole, pH 7.5, over a 18-hour period. It was ascertained that under the conditions of the experiment no significant exchange of F-actin-bound nucleotide occurred (4, 24). The F-actin was essentially free of unbound ATP, as judged by the rapid rise of light scattering (90°), to levels characteristic of actomyosin upon mixing F-actin with myosin. Under the same experimental conditions the addition of as little as 10⁻⁷ M ATP to actomyosin solutions caused a transient but significant decrease in the intensity of scattered light.

The binding of [³²P]ADP to myosin and its fragments was measured by three different methods.

**Equilibrium Dialysis**

Equilibrium dialysis was performed essentially as described earlier (4, 6). Despite the elaborate precautions taken to ensure the purity of myosin and actin preparations, trace amounts of myokinase and AMP-deaminase contamination caused a breakdown of ADP into ATP and IMP during the prolonged equilibration dialysis in the presence of Mg²⁺. The extent of ADP decomposition was measured following the separation of isotopically labeled nucleotides by high voltage electrophoresis on Whatman No. 3MM paper in 0.03 M sodium citrate buffer (pH 5.0) at 50 volts/cm for 1 to 2 hours with adequate cooling. The nucleotide spots were cut out and the radioactivity counted as the method of Leightfield and Eigner (25). Some of the reported binding data were corrected for ADP decomposition which was usually less than 90% of the total ADP in an equilibrium dialysis experiment of 12 to 20 hours duration at 3-5°. With the centrifuge transport method (see below) no correction was required because of the shorter incubation time.

**Ultracentrifuge Transport**

The protein solution containing 50 mM Tris-HCl buffer, pH 8.0, 1.0 mM MgCl₂, and [³²P]ADP in varying concentration was placed in an ultracentrifuge tube of 3-ml volume and centrifuged at 80,000 x g for 4 to 12 hours at 5° in a Spinco No. 40 preparative rotor. The contents of the centrifuge tubes were carefully removed in several layers (0.4 to 0.5 ml each) and the protein content (28) and [³²P]ADP radioactivity of each layer were measured. The ADP concentration of each layer and of the centrifuge tube were measured with a Nikon profile projector using the actin-free H-meromyosin (0.23 mg/ml) or H-meromyosin (0.23 mg/ml) with increasing concentrations of actin. The sedimentation of light scattered at 90° was measured in a medium of 0.6 M KCl, 44 mM Tris, pH 9.0, and 1.5 mM MgCl₂. The buffer solutions were filtered through a Millipore filter to remove dust and the protein solutions were centrifuged periodically to free them from aggregated material.

The usual procedure was to mix fixed concentrations of myosin (0.2 mg/ml) or H-meromyosin (0.25 mg/ml) with increasing concentrations of actin. The relative intensity of the 90° scattering of actomyosin or actomonomer solutions after deduction of the scattering intensity of myosin and actin measured separately gave the value of ΔAM, the differential scattering attributable to actomyosin formation. The value of ΔAM was usually similar to the decrease in light scattering produced by the addition of 10⁻⁶ M ATP to actomyosin, which is denoted as ΔATP.

In the ultracentrifuge technique the meromyosin or subfragment 1 were mixed with increasing concentrations of F-actin and centrifuged in the Spinco model E analytical ultracentrifuge at 52,640 rpm at 5-10°. F-actin and its complexes with H-meromyosin or subfragment 1 sediment rapidly leaving the unbound H-meromyosin and subfragment 1 behind. The concentration of the unbound enzyme was measured with a Nikon profile projector using the actin-free H-meromyosin or subfragment 1 solutions as reference.

The light scattering and ultracentrifuge data were supplemented with measurements of the effect of F-actin upon the Mg²⁺-moderated ATPase activity of myosin.

**Measurement of ATPase Activity**

The enzymatic activity of myosin, H-meromyosin, and subfragment 1 was measured under the following conditions. Mg²⁺-moderated ATPase—Activity was measured in a medium of 50 mM Tris buffer, pH 8.0, 0.05 to 0.6 M KCl, 5 mM MgCl₂, 5 mM ATP, and enzyme in a final concentration of 0.2 mg/ml. The incubation time was 10 to 60 min at 25°.

**Actin Activation of Myosin ATPase**—The medium contained 0.03 M KCl, 10 mM imidazole buffer, pH 7.5, 1 mM MgCl₂, 2.5 mM ATP, 0.2 mg of myosin/ml, and actin in varying concentrations. The incubation was carried out at 25° for 2 to 10 min.

**Ca²⁺-activated ATPase**—The medium contained 0.06 M KCl, 50 mM Tris buffer, pH 8.0, 5 mM CaCl₂, 5 mM ATP, and 0.10 to 0.20 mg of enzyme/ml in a final concentration. Incubation was usually carried out at 25° for 2 to 10 min.

**EDTA-activated ATPase**—The medium contained 0.6 M KCl, 5 mM EDTA, 50 mM Tris buffer, pH 8.0, 5 mM ATP, and enzyme in a final concentration of 0.05 to 0.1 mg/ml. Incubation was carried out at 25° for 2 to 10 min.

The reaction was stopped in each system by the addition of trichloroacetic acid to a final concentration of 2% followed by centrifu-
ation and determination of the orthophosphate liberated according to Fiske and Subbarow (27).

Materials

N-ethylmaleimide was obtained from Sigma Chemical Co. ATP and ADP were purchased from P-L Biochemicals Inc. Adenosine 5'-diphosphate-[8-14C]trilithium salt was the product of Schwarz BioResearch (Schwarz/Mann) Orangeburg, New York.

RESULTS

Binding of F-Actin and ADP to Myosin—Myosin or its subfragments bind ADP with relatively high affinity which is influenced by the ionic strength of the medium (4). The affinity constant of ADP binding to myosin is approximately $2 \times 10^7 \text{ M}^{-1}$ at low ionic strength (Fig. 1A) and decreases to approximately $1.7 \times 10^6 \text{ M}^{-1}$ at a KCl concentration of 0.6 M (Fig. 1C). The maximum number of ADP binding sites is 1.4 at low ionic strength (Fig. 1A) and 1.60 and 1.70 at KCl concentrations of 0.3 M and 0.6 M, respectively (Fig. 1, B and C). This is in essential agreement with previous reports (4, 9) and is usually taken to indicate the existence of two ADP binding sites per mol of myosin. Myosin is in the form of filamentous aggregates at low ionic strength and forms true solution only at a KCl concentration above 0.3 M. The marked deviation of the Scatchard plot from linearity at low ionic strength (Fig. 1A) may be related in part to the aggregated state of the proteins and in part to ADP decomposition which affects the free ADP especially at low ADP concentrations.

Addition of F-actin to myosin in actin monomer to myosin mole ratios ranging from 0 to 4 causes an inhibition of ADP binding (Fig. 1 A to C). At low ionic strength the inhibition is largely noncompetitive as indicated by the marked change in the maximum number of ADP binding sites as the concentration of F-actin increases. Even at an actin/myosin ratio of 4, a significant amount of ADP remained bound to myosin. At this actin concentration the actin binding sites of myosin as judged from the effect of actin upon the Mg$^{2+}$-moderated ATPase activity of myosin are fully saturated (Fig. 2). At 0.3 M KCl (Fig. 1B) or 0.6 M KCl concentration (Fig. 1C) the inhibitory effect of F-actin upon the ADP binding appears competitive up to an actin to myosin ratio of 2 where on the basis of light scattering measurements (Fig. 3) myosin is fully saturated with actin.

Surprisingly, at an actin to myosin mole ratio of 2, under the conditions of these experiments a significant portion of the ADP binding sites remains occupied with ADP, and a further increase in the actin concentration to an actin to myosin ratio of 4 has little effect upon the residual binding to ADP to myosin. This is not attributable to the dissociation of actomyosin by ADP since ADP up to 100 $\mu$M concentration had little effect upon the steady state rate of actin-activated ATP hydrolysis or upon the light scattering of actomyosin solutions measured at 25° in the presence of 0.6 M KCl, 50 mM Tris, pH

![Fig. 2. Actin stimulation of the Mg$^{2+}$-activated ATPase activity of myosin. The rate of Mg$^{2+}$-activated myosin ATPase was measured in the absence and in the presence of increasing concentrations of F-actin, in a medium of 10 mM KCl, 1 mM MgCl$_2$, 10 mM imidazole, pH 7.0, and 2 to 5 mM ATP, at 25°.](http://www.jbc.org/)
8.0, and 1.5 mM MgCl₂ (Table I). ADP can be readily displaced from the actin-insensitive sites in the presence of 10⁻³ to 10⁻⁴ M inorganic pyrophosphate (Fig. 4), in agreement with the previously demonstrated simple competition between these two substrate analogs (4, 6).

Effect of F-Actin upon Binding of ADP to H-Meromyosin—The tendency of myosin to form aggregates below 0.3 M KCl concentration complicates the interpretation of the competition between actin and ADP at physiological ionic strengths. Hence the effect of F-actin on the ADP binding of H-meromyosin was investigated.

Scatchard plots of ADP binding at 0.05 M (Fig. 5A), 0.3 M (Fig. 5B), and 0.6 M (Fig. 5C) KCl concentration indicate inhibition by F-actin with a decrease in the apparent affinity of ADP binding without a significant change in the maximum number of binding sites. The linearity of the Scatchard plots of H-meromyosin·ADP interaction at low ionic strength (Fig. 5A) is in contrast to the marked nonlinearity observed with myosin (Fig. 1A). At an actin-to-H-meromyosin ratio of 2, significant ADP binding remains at free ADP concentrations as low as 1 to 15 μM (Fig. 5A to C, insets), and a further increase in actin concentration to an actin-to-HMM ratio of 4 causes little or no further change in ADP binding.

The stoichiometry of actin-H-meromyosin interaction was investigated by light scattering (Fig. 6) and ultracentrifuge measurements (Fig. 7). Irrespective of ionic strength, clear-cut saturation of H-meromyosin with F-actin was obtained at an actin:H-meromyosin ratio of 2. The presence of ADP up to 10⁻⁴ M concentration had little influence upon the actin·HMM interaction as judged by ultracentrifuge transport (Table I).

Binding of Actin and ADP to Subfragment 1—Myosin and H-meromyosin contain two nucleotide and actin binding sites located on the two head portions of the molecule. The insensitivity of some of the nucleotide binding to actin may indicate that F-actin lowers the affinity of ADP binding at two equivalent sites by a negative interaction between the actin and the nucleotide binding sites. Alternatively, chemical differences between the two sites or steric effects connected with the proximity of the two otherwise identical head portions of the myosin or H-meromyosin molecules may be considered. In order to distinguish between these alternatives the effect of actin upon the binding of ADP to S-1 fragments was studied (Fig. 8).

At KCl concentrations ranging from 0.05 to 0.6 M, S-1 fragments bound close to 1 mol of ADP/100,000 g of protein. The affinity constant of ADP binding was 2.4 × 10⁶ M⁻¹ at 0.05 M KCl (Fig. 8A) and decreased with increasing KCl concentration to 5.4 × 10⁵ M⁻¹ (Fig. 8C). Maximum inhibition of ADP binding was obtained at an actin to S-1 molar ratio of 1 where approximately one-half of the ADP bound by S-1 in the absence of actin was displaced from the binding site (Fig. 8, insets). Increasing the actin to S-1 mole ratio from 1 to as high as 8 mol of actin/mol of S-1 fragment did not cause a significant change in the amount of ADP bound to S-1 at free ADP concentrations ranging from 1 to 15 μM (Fig. 8, insets). Direct measurement of the binding of F-actin to S-1 fragments in the analytical ultracentrifuge showed that at an actin to S-1 mole ratio of 1 and above, no detectable free S-1

![Fig. 3. Light scattering measurements of actin-myosin interaction.](https://example.com/f3.png)

Light scattering measurements were carried out as described under "Experimental Procedures" in a medium of 0.6 M KCl, 44 mM Tris, pH 8.0, and 1.5 mM MgCl₂. ○, O, ΔAM; ■, ΔATP.

![Fig. 4. The effects of pyrophosphate upon the binding of ADP to actomyosin.](https://example.com/f4.png)

The ADP binding was measured by ultracentrifuge transport in a medium of 0.3 M KCl, 50 mM Tris, pH 8.0, and 1 mM MgCl₂. ○, myosin alone; □, actomyosin (A/M = 2.0); ■, actomyosin (A/M = 2) + 10⁻⁴ M inorganic pyrophosphate.

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Table 1

| ADP concentration | Ultracentrifuge | Light scattering |
|-------------------|----------------|-----------------|
|                   | Acto-S-1 (A/S-1 = 0.8) | Acto HMM (A/HMM = 1.3) | Acto HMM (A/HMM = 2.0) | Actomyosin (A/M = 2.0) |
| mM                | % S-1 bound | % HMM bound | % HMM bound | % myosin bound |
| 0                 | 80          | 40           | 86           | 100          |
| 0.1               | 82.5        | 46.6         | 76           | 88           |
These observations follow the pattern described for myosin and H-meromyosin. Saturation of S-1 fragments with F-actin causes the displacement of approximately one-half of the bound ADP from the active site. The remaining ADP binding is unaffected by actin even at high actin/S-1 molar ratios.

It is unlikely that the ADP binding observed with saturating concentrations of F-actin is due to the nonspecific interaction of ADP with S-1 fragments since the ADP binding is inhibited by $10^{-6}$ to $10^{-5}$ M inorganic pyrophosphate, a substrate analog known to interact with the nucleotide binding site of myosin (Fig. 10). Scatchard plots of ADP binding in F-actin-free systems indicate a single set of noninteracting sites with an affinity constant of the order of $10^4$ to $10^5$ M which is much higher than the expected affinity for nonspecific anion binding sites.

The apparent insensitivity of a portion of the ADP binding of S-1 to F-actin is not attributable to the dissociation of acto-S-1 complex by ADP since 0.01 to 0.1 mM ADP had no significant effect upon the concentration of free S-1 fragments in a system containing 0.8 mol of actin/mol of S-1 protein (Table I).

**DISCUSSION**

The observations presented in this report confirm earlier findings (4, 8, 9) about the existence of two ADP and two actin binding sites in myosin or H-meromyosin and one of each in subfragment 1. The equilibrium constant of ADP binding at the two sites of myosin is similar.

F-actin inhibits the binding of ADP to myosin but significant ADP binding occurs even at actin to myosin ratios where...
myosin is fully saturated with actin. Similar observations were made with H-meromyosin and with subfragment 1, indicating that the results are not attributable to the formation of myosin filaments or to steric interference between the two heads of a myosin molecule. The ADP binding observed in the presence of a saturating concentration of F-actin is inhibited by $10^{-5}$ to $10^{-6}$ M inorganic pyrophosphate, and the inhibitor constant of pyrophosphate calculated from the competitive inhibition is similar to the previously determined (6) dissociation constant of pyrophosphate for the active site of myosin. Therefore, the F-actin-resistant ADP binding presumably occurs at the active site.

Exchange of the bound nucleotide of F-actin is not likely to account for the F-actin-insensitive ADP binding since the extent of such exchange is limited (24, 28, 29). Furthermore, inorganic pyrophosphate nearly completely inhibited the F-
actin-insensitive ADP binding of actomyosin although its affinity for F-actin is negligible (23).

Hanson et al. (20) observed that actin forms lateral aggregates at high (70 mM) MgCl₂ concentration. Aggregation of actin would explain the incomplete inhibition of ADP binding at high actin concentration, since myosin would interact only with actin molecules on the surface of these aggregates. However, at the MgCl₂ concentration used in these experiments (1 to 1.5 mM) such aggregation is negligible and the saturation of myosin or its fragments with F-actin shows clear stoichiometry.

The most likely explanation of the observed effects of F-actin upon the ADP binding is that F-actin and ADP are bound simultaneously to myosin leading to the formation of an actomyosin-ADP complex. The inhibitory effect of F-actin upon the ADP binding may be explained by the negative interaction between the actin binding sites and the distinct nucleotide binding sites, as indicated in the following scheme:

\[
\begin{align*}
\text{F-actin + ADP + Myosin} & \rightarrow \text{Actomyosin-ADP} \\
\text{Actin} & \quad \text{Myosin-ADP} \\
& \quad \text{F-actin} \\
& \quad \text{ADP}
\end{align*}
\]

where \( K_a, K_b, K'_a, \) and \( K'_b \) denote affinity constants. It is assumed that the two nucleotide binding sites located on the two myosin heads are identical and the same is true for the actin binding site. According to the data, binding of F-actin to myosin lowers the affinity of ADP for the nucleotide binding site by at least one order of magnitude (\( K_a \gg K'_a \)), while ADP has apparently little effect upon the binding of F-actin to myosin (5). The ADP binding observed in the presence of a saturating concentration of F-actin is defined by \( K'_a \), the affinity constant of ADP binding to actomyosin. In this interpretation the relationship between the actin and the nucleotide binding sites is not truly competitive but involves some interaction between the two types of sites.

The nucleotide and actin binding sites apparently involve distinct functional groups (31-34), and conditions exist for the selective modification of each set of sites. Treatment of actomyosin with —SH group reagents makes it insensitive to the dissociating effect of ATP without breaking actomyosin links (5). Myofibrils treated in a similar manner lose both their contractility and relaxing response (31). After treatment of actomyosin with a higher concentration of N-ethylmaleimide or iodoacetamide at pH 6.0, complete inhibition of ATPase activity was obtained without dissociation of actomyosin (32-34). The myosin component isolated from these preparations formed actomyosin which was resistant to the dissociating effect of ATP. Finally, N-ethylmaleimide in high concentration inhibits the Ca²⁺- or EDTA-moderated ATPase activity of myosin, but leaves the binding of ATP (35) and ADP (36, 37) relatively unaffected. The selective inhibition of the different functions of myosin by —SH group reagents implies that the catalytic and actin binding sites are separate but related. Conditions may exist for the chemical modification of myosin which leaves the binding of actin and ADP relatively unaffected but eliminates the negative interaction between the actin and nucleotide binding sites.

The observed effects of F-actin upon the ADP binding may also be explained by models based upon differences in the intrinsic affinity and actin sensitivity of the ADP binding sites located on the two heads of myosin. Among early indications of binding site heterogeneity are the stoichiometry of early phosphate burst of ATP hydrolysis (14, 15) and the relationship between the ultraviolet difference spectrum and the binding of ATP, ADP, and pyrophosphate to H-meromyosin (14, 38). These models require special assumptions to fit the data and therefore appear less plausible. In view of the large degree of interpretive freedom permitted by the binding data, the demonstration of two populations of S-1 fragments, each capable of ADP binding but differing in actin sensitivity, would be required to substantiate this possibility.

Simultaneous binding of actin and ADP to myosin was also observed in glycerinated muscle fibers (39). In the absence of ATP, glycerinated fibers are in rigor, i.e. the myosin crossbridges are linked to actin. Yet these fibers bind the same amount of ADP that can be bound during ATP hydrolysis. Maximum ADP binding was achieved without a significant change in the elastic modulus, indicating that ADP was bound to the fibers without dissociating actin-myosin links. This is in agreement with previous observations showing that ADP dissociates actomyosin only at very high concentrations (5).

The existence of actin-insensitive ADP binding to myosin in living muscle remains to be established (40). As the bound ADP of actomyosin is readily displaced by ATP it is likely that in living muscle the actomyosin-ADP complex is immediately converted into myosin-ATP.

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