The glucocorticoid receptor exists in the cytoplasm of hormone-untrated cells as a complex with the 90-kDa heat shock protein (HSP90). Glucocorticoids induce dissociation of the glucocorticoid binding protein from HSP90 and translocation of the receptor to the nucleus. HSP90 binds to actin filaments, and calmodulin or tropomyosin inhibits the binding. We present here evidence that the HSP90-containing glucocorticoid receptor complexes (8 S receptor) bind to filamentous actin in vitro while the HSP90-free form of the receptor does not. The binding was detectable for both the crude cytosolic fractions and the partially purified 8 S glucocorticoid receptor. Purified HSP90 or tropomyosin completely abolished the binding. Calmodulin also inhibited the binding in a Ca<sup>2+</sup>-dependent manner. From these results, we conclude that the glucocorticoid receptor complex is able to bind actin filaments via the HSP90 moiety. The binding may provide an anchoring mechanism for the glucocorticoid receptor in the cytoplasm.

The glucocorticoid receptor (GCR) is recovered in the cytosolic fraction of hormone-untrated cells as a molecular form with a sedimentation coefficient of 8 S (Holbrook et al., 1983; Vedekis, 1983; Okret et al., 1985). Although the cytosolic 8 S GCR is unable to bind DNA, it is converted into the active form by binding with glucocorticoids (for review, see Pratt, 1987). The activation of the receptor is associated with the transformation of the receptor from 8 S to 4 S (Holbrook et al., 1983; Vedekis, 1988). The 8 S GCR consists of the intrinsic glucocorticoid binding protein and a dimer of the associated protein (Joab et al., 1984; Houseley et al., 1985; Schuh et al., 1985; Mendel et al., 1986; Denis et al., 1987) that has been identified as a heat shock protein (HSP), HSP90 (Catelli et al., 1985; Sanchez et al., 1986, 1987). During the activation process after the hormone binding, HSP90 dissociates from the receptor, and the GCR complex is converted from the 8 S non-DNA binding form to the 4 S DNA binding species (for review, see Pratt et al., 1989; Denis and Gustafsson, 1989). Even after the binding with the ligands, the 8 S GCR is stabilized in a relatively low ionic strength buffer containing transition metal oxyanions such as molybdate (Dahmer et al., 1984). Current evidence suggests that the 8 S GCR is really present in the cytoplasm of living cells (Howard and Distelhorst, 1988), implying that the association of the 4 S GCR with HSP90 is physiologically significant. In fact, the association has been suggested to be necessary for high affinity binding of the GCR with glucocorticoids (Bremser et al., 1990).

HSP90, a 90-kDa heat shock protein, is induced in various organisms by heat shock or other environmental stresses, but is also constitutively expressed in unstressed cells (for review, see Lindquist and Craig, 1988; Schlesinger, 1990). HSP90 exists as a dimeric form under physiological conditions and is distributed throughout the cytoplasm (Koyasu et al., 1986; Lindquist and Craig, 1988). We have noted that ruffling membranes of cultured cells were brightly stained with antibodies against HSP90 and also with phalloidin (Koyasu et al., 1986). In addition, a heat shock-resistant Chinese hamster ovary variant expressing HSP90 to a relatively high degree showed the high level of cell motility (Yahara et al., 1986). These observations raised the possibility that HSP90 interacts with actin filaments. This turned out to be the case, and we have found that HSP90 cross-links actin filaments (Koyasu et al., 1986; Nishida et al., 1986). The binding of HSP90 to actin filaments is inhibited by tropomyosin or by Ca<sup>2+</sup>-calmodulin (Nishida et al., 1986). Taking this finding together with the fact that 8 S GCR consists of a dimeric HSP90, we have hypothesized that 8 S GCR interacts with actin filaments through its HSP90 moiety. In this study, we have examined this hypothesis and clearly showed that 8 S GCR but not 4 S GCR binds actin filaments and that the binding is specifically mediated by HSP90.

**EXPERIMENTAL PROCEDURES**

**Materials**—[6,7-<sup>3</sup>H]Triamcinolone acetonide (TAA) (1570 GBq/mmol) was obtained from Du Pont-New England Nuclear. Phosphocellulose P11 was obtained from Whatman Chemical Separation (Maidstone, United Kingdom), DEAE-Sepharose Fast Flow and Superdex-200 HiLoad from Pharmacia Fine Chemicals AB (Upsala, Sweden), and hydroxyapatite high resolution from Calbiochem. Radioactively labeled TAA and all other analytical reagents were obtained from Sigma.

**Buffers and Cells**—HEPDG + Mo buffer: 25 mM Hepes, 1.5 mM EDTA, 1 mM dithiothreitol, and 10% (v/v) glycerol, pH 7.6, containing 10 mM sodium molybdate. Mouse hepatoma Hepa-1 cells (provided by Dr. Y. Fuji-i-Kuriyama, Tohoku University) was cultured in Dulbecco’s modified Eagle’s medium containing 10% fetal calf serum (GBHC).

**Proteins**—Calmodulin was purified from porcine brains as described (Nishida et al., 1979). Rabbit skeletal muscle G-actin was prepared by the method of Spudich and Watt (1971) and further purified by gel filtration on a Superdex-200 HiLoad column equilibrated with a buffer solution containing 0.1 mM CaCl<sub>2</sub>, 0.2 mM ATP, 0.1 mM dithiothreitol, 0.01% NaN<sub>3</sub>, and 2 mM Hepes, pH 7.8. HSP90
was purified from mouse lymphoma L5178Y cells as previously described (Koyasu et al., 1986). Tropomysin from rabbit skeletal muscle was a generous gift from Dr. E. Nishida (University of Tokyo). Rabbit antibodies raised against purified mouse HSP90 was prepared as described (Yonezawa et al., 1988).

**Ligand Binding Assay**—A hydroxylapatite adsorption assay was used to quantitate the receptors (Nemoto et al., 1990). An aliquot of samples was incubated with 20 nM [3H]TAA in the presence or absence of a 500-fold molar excess of radiolabeled TAA. After the incubation for 3 h on ice, the sample was mixed with an equal volume of hydroxylapatite suspension (50% v/v in HEDG + Mo buffer) for 20 min with occasional mixing. Hydroxylapatite was precipitated by a brief centrifugation, the pellet material was washed with 0.5 ml of HEDG + Mo buffer three times, and bound [3H]TAA-GCR complexes were extracted with 0.2 ml of 0.4 M potassium phosphate, pH 7.4, for 15 min on ice with occasional vortexing for the counting of radioactivity.

**Preparation of Actin-free Glucocorticoid Receptors**—Actin-free and ligand-free GCRs were prepared according to the methods described by Nemoto et al. (1990) with modifications. All procedures were carried out at 0–4 °C. Briefly, rat livers were perfused with ice-cold phosphate-buffered saline, pH 7.4, washed with HEDG + Mo buffer, ground, homogenized in 2 volumes of HEDG + Mo buffer containing protease inhibitors (1 mM phenylmethylsulfonyl fluoride, 2 μg/ml leupeptin, 1 μg/ml pepstatin A, 2 μg/ml antipain, and 10 μg/ml aprotinin), and clarified by centrifugation (75,000 × g, 40 min).

The lipid layer was discarded, and the supernatant fluid was mixed with 1 ml of DEAE-Sepharose Fast Flow column equilibrated with HEDG + Mo buffer containing 80 mM NaCl. Proteins were retained on the DEAE-Sepharose Fast Flow column (16 mm × 20 cm) equilibrated with HEDG + Mo buffer and gently agitated for 1 h. The unbound materials were collected by centrifugation (3,000 × g, 10 min) and applied on a DEAE-Sepharose Fast Flow column (16 mm × 20 cm) equilibrated with HEDG + Mo buffer containing 80 mM NaCl, and the column was washed extensively with the same buffer until the absorbance at 280 nm reached a stable baseline value. Proteins retained on the DEAE-Sepharose column were eluted with 0.2 M NaCl in HEDG + Mo buffer containing protease inhibitors (1 mM phenylmethylsulfonyl fluoride, 2 μg/ml leupeptin, 1 μg/ml pepstatin A, 2 μg/ml antipain, and 10 μg/ml aprotinin), and clarified by centrifugation (75,000 × g, 40 min). The lipid layer was discarded, and the supernatant fluid was mixed with a 1 ml of DEAE-Sepharose Fast Flow column equilibrated with HEDG + Mo buffer and gently agitated for 1 h. The unbound materials were collected by centrifugation (3,000 × g, 10 min) and applied on a DEAE-Sepharose Fast Flow column (16 mm × 20 cm) equilibrated with HEDG + Mo buffer containing 80 mM NaCl, and the column was washed extensively with the same buffer until the absorbance at 280 nm reached a stable baseline value. Proteins retained on the DEAE-Sepharose column were eluted with 0.2 M NaCl in HEDG + Mo buffer and subsequently separated by gel filtration chromatography on a Superdex-200 HiLoad column (16 mm × 60 cm) which had been equilibrated with HEDG + Mo buffer without NaCl. Proteins were retained on the column by the absorbance at 280 nm, and the glucocorticoid binding was assayed as described above. HSP90 is one of the major proteins in the preparation of partially purified 8 S GCR. The glucocorticoid binding activity was eluted as a single peak from the gel filtration column. The stannous chloride assay (Strelow and Borthors, 1967) showed that the GCR fractions contained molybdate. An addition of 500-fold nonradioactive TAA to the peak fraction completely abolished the binding of [3H]TAA indicating that the observed [3H]TAA binding peak contains specific GCR. The fractions contained −2 mg/ml proteins and were free of actin as shown by SDS-PAGE (data not shown).

**Preparation of Cytosol**—Hepa-1 cells were scraped and homogenized in HEDG + Mo buffer containing protease inhibitors (1 mM phenylmethylsulfonyl fluoride, 2 μg/ml leupeptin, 1 μg/ml pepstatin A, 2 μg/ml antipain, and 10 μg/ml aprotinin). The homogenate was then centrifuged at 22,000 × g for 20 min at 2 °C, the supernatant was further centrifuged at 100,000 × g for 60 min at 2 °C, and the resulting supernatant was used as Hepa-1 cytosol.

**Labeling the Glucocorticoid Receptors with [3H]TAA**—Samples containing the receptors were incubated with 20 nM [3H]TAA for 4 h on ice. The unbound ligands were adsorbed to a 1/5 volume of a dextran charcoal suspension (100 mg/ml charcoal and 10 mg/ml dextran in HEDG + Mo buffer) at 4 °C for 4 h. The charcoal was removed by centrifugation at 4000 × g for 10 min and by filtering the supernatant through a 0.22-μm filter. When indicated, the [3H]TAA-labeled samples were incubated with anti-HSP90 IgG on ice for 3 h.

**Sucrose Gradient Centrifugation Analysis**—200-μl aliquots of [3H]TAA-labeled samples were layered onto 5.2-ml linear sucrose density gradients (5–20%) prepared in HEDG + Mo buffer. Gradients were centrifuged at 45,000 rpm for 16 h at 2 °C in a Beckman SW 50.1 rotor. 150-μl fractions were collected, and the radioactivity in each fraction was determined.

**Assay of Binding of Steroid Hormone Receptors to Actin Filaments**—Hepa-1 cytosols or partially purified GCRs were labeled with [3H]TAA as described above. Various concentrations of actin were polymerized by incubating in a buffer containing 20 mM Hepes, 100 mM KCl, 2.5 mM MgCl2, 0.03 mM ATP, 1 mM 2-mercaptoethanol, 0.02 mM dithiothreitol, and 0.02 mM imidazole, pH 7.5, in the presence of 2 mM Ca2+ or 2 mM EGTA for 60 min at room temperature, and a further 20 min on ice, and resulting actin filaments were incubated with samples containing labeled receptors on ice for 90 min. When indicated, various concentrations of purified calmodulin, tropomyosin, or HSP90 were added in the mixture before the addition of the receptors. The mixture was then centrifuged at 100,000 × g for 90 min at 2 °C so that polymerized actin and associated proteins were precipitated, and the radioactivities in the pellet fractions solved in 1% SDS were counted.

**RESULTS**

**Co-precipitation of Labeled GCRs in Crude Cell Extracts with Endogenous or Exogenous Actin Filaments**—GCRs in Hepa-1 cytosols were labeled with [3H]TAA, mixed with or without exogenous actin filaments, and centrifuged so as to pellet down the actin filaments together with bound proteins. The binding was determined at various concentrations of cytosol fractions in the presence or absence of exogenously added actin. All the experimental results were summarized in Fig. 1, indicating that exogenously added actin filaments significantly increased the precipitated radioactivity in all preparations. We have confirmed that the [3H]TAA-labeled Hepa-1 cytosol used for these binding experiments contained exclusively the 8 S form of GCR (data not shown). These results suggest that [3H]TAA-bound 8 S GCR was co-precipitated with actin filaments. The cytosolic fraction contains high concentrations of actin. This explains the fact that significant amounts of [3H]TAA-bound receptors were precipitated in the absence of the exogenous actin. We have observed that calmodulin reduced the amount of [3H]TAA-bound 8 S GCR co-precipitated with actin filaments in a Ca2+-dependent manner (see below).

To compare the binding activity to actin filaments of HSP90-containing 8 S GCR and that of HSP90-free 4 S receptor, the cytosolic fraction containing HSP90-free 4 S receptor was prepared by extracting the cells in the absence of HEDG + Mo buffer containing protease inhibitors (1 mM phenylmethylsulfonyl fluoride, 2 μg/ml leupeptin, 1 μg/ml pepstatin A, 2 μg/ml antipain, and 10 μg/ml aprotinin). The homogenate was then centrifuged at 22,000 × g for 20 min at 2 °C, the supernatant was further centrifuged at 100,000 × g for 60 min at 2 °C, and the resulting supernatant was used as Hepa-1 cytosol. For the preparation of cytosol containing 4 S receptors, sodium molybdate was omitted during the processes, and 0.4 M KCl was added to the buffer.

**Density Gradient Centrifugation Analysis**—200-μl aliquots of [3H]TAA-labeled samples were layered onto 5.2-ml linear sucrose density gradients (5–20%) prepared in HEDG + Mo buffer. Gradients were centrifuged at 45,000 rpm for 16 h at 2 °C in a Beckman SW 50.1 rotor. 150-μl fractions were collected, and the radioactivity in each fraction was determined.

**FIG. 1.** Association between GCR-HSP90 complexes and actin filaments in Hepa-1 cytosols. Various amounts of [3H]TAA-labeled Hepa-1 cytosols were incubated with (+ Exogenous Actin) or without (− Exogenous Actin) various concentrations of exogenous actin filaments, and the polymerized actin and the associated proteins were precipitated as described under "Experimental Procedures." The radioactivities in resulting pellet fractions were counted. Each point represents the co-precipitated GCR in the absence (corresponding to the horizontal axis) or presence (the vertical axis) of the exogenous actin.
of molybdate, followed by treatment in 0.4 M KCl. We have confirmed by sucrose density gradient centrifugation analysis that the cytosol fraction prepared under these conditions contained 8 S GCR (data not shown). The cytosol fraction containing 8 S receptor and that containing 4 S receptor were separately incubated with polymerized actin in the same buffer condition, and co-precipitation of receptors was determined (Fig. 2). The results clearly indicated that the HSP90-containing 8 S GCR was co-precipitated with both endogenous and exogenous actin filaments, whereas the HSP90-free 4 S GCR was not co-precipitated at all.

**Purified GCR Binds to Polymerized Actin Filaments**—To elucidate the binding of 8 S GCR with actin filaments more precisely, we partially purified the 8 S GCR from rat livers to form an actin-free preparation. The purification was performed using phosphocellulose and DEAE-Sepharose followed by gel filtration according to the methods described by Nemoto et al. (1990) with modifications. Partially purified 8 S GCR was tested for its actin binding activity. Aliquots of partially purified 8 S GCR were incubated with 20 nM [3H]TAA, and the unbound ligand was adsorbed to dextran-charcoal. About one-fourth of the unadsorbed radioactivity was considered to be free from GCR because this radioactivity did not bind to hydroxylapatite. The results clearly showed that purified 8 S GCR bound to filamentous actin in an actin dose-dependent manner (Fig. 3, ○). The binding of 8 S GCR was saturable as the concentration of actin increased. The amount of 8 S GCR bound to actin filaments was correlated with that of the bulk of HSP90 co-precipitated with actin filaments as revealed by SDS-PAGE of the precipitated proteins (Fig. 3, O). 40–50% of the total [3H]TAA was co-precipitated with an excess amount of polymerized actin. These results suggest that 53–67% of the total GCR bound actin filaments. It is not clear, however, why the remaining GCR did not bind actin filaments.

**Effects of Purified HSP90, Tropomyosin, and CaCl2-Calmodulin on the Binding of Partially Purified GCR with Actin Filaments**—We tested whether or not the binding to actin filaments of partially purified 8 S GCR is similar to that of HSP90. First, we examined the effect of purified HSP90 on the binding of 8 S GCR to filamentous actin. Purified HSP90 inhibited the binding in a dose-dependent manner (Fig. 4(a)). Secondly, we tested the effect of purified tropomyosin on the binding for tropomyosin is known to inhibit the binding of purified HSP90 to filamentous actin in vitro (Nishida et al., 1986). The results clearly showed that tropomyosin inhibits the binding (Fig. 4(b)). The inhibition by 50% was observed with about 0.25 mg/ml tropomyosin, which is comparable to the inhibitory concentration of tropomyosin on the binding of purified HSP90 to actin filaments in vitro (Nishida et al., 1986). Finally, we have found that calmodulin inhibits the binding in the presence of CaCl2 (Fig. 5), which is consistent with the results obtained with purified HSP90 and actin. Neither CaCl2 without calmodulin nor calmodulin without CaCl2 affected the binding at all. These results again strongly suggest that 8 S GCR binds to filamentous actin via its HSP90 moiety.

**DISCUSSION**

The results presented in this paper argue strongly that 8 S GCR binds to actin filaments but that 4 S GCR does not. Purified HSP90 or tropomyosin completely abolished the binding and calmodulin inhibited the binding only in the
presence of Ca++. Taken together with our previous findings on the in vitro binding of HSP90 to actin (Koyasu et al., 1986; Nishida et al., 1986), we conclude that the binding is specifically and exclusively mediated by the HSP90 moiety.

A body of evidence indicates that 8 S GCR is present in the cytoplasm as the non-DNA binding form and that binding of glucocorticoids induces dissociation of the GCR-HSP90 complex and confers on the receptor the ability to bind DNA (for review, see Pratt et al., 1989; Denis and Gustafsson, 1989). During this activation process of GCR, the receptor is translocated into the nucleus (Wikström et al., 1987). The GCR polypeptide has been shown to contain a nuclear location signal sequence and, therefore, to be intrinsically located in the nucleus (Picard and Yamamoto, 1987). Nevertheless, 8 S GCR is present in the cytoplasm. One explanation for this fact is that the nuclear location signal of GCR becomes cryptic when it associates with HSP90. Another plausible explanation was raised in this study; that is, 8 S GCR anchors on actin filaments and, therefore, is restricted to translocate into the nucleus although the nuclear location signal is exposed and functional.

Receptors for androgen (Joab et al., 1984), estrogen (Reudeilh et al., 1987), progesterone (Kost et al., 1989), mineralocorticoid (Rafestin-Oblin et al., 1989), and dioxin (Peredew, 1988; Denis et al., 1988) are similar to GCR in their complex formation with HSP90. All of these receptor complexes might be reasonably expected to interact with actin filaments through their HSP90 moieties. The cytosolic and nuclear distribution of these receptor complexes are somewhat controversial, however. Recently, it was shown that the progesterone receptor-HSP90 complex is predominantly distributed in the nucleus (Renoir et al., 1990) suggesting that the nuclear location signal is exposed in this complex. As we have previously described, the interaction between HSP90 and actin filaments is relatively weak (Kd = ~10^-4 M) (Nishida et al., 1986). In addition, the interaction was regulated by Ca++ and calmodulin (Nishida et al., 1986). All these properties of the interaction have been shown in this study for that between 8 S GCR and actin filaments. Some steroid hormone receptors or a portion of them may be free from the anchoring system postulated herein.

Other members belonging to the steroid hormone receptor superfamily or the nuclear receptor superfamily (Evans, 1988), including the receptors for thyroid hormone (Pascual et al., 1982) and retinoids (Petkovich et al., 1987; Giguere et al., 1987), exist as molecular forms free from HSP90 and they reside exclusively in the nucleus even in the absence of the corresponding ligands. The receptors of this type may be translocated to the nucleus soon after they are synthesized possibly because they do not form complexes with HSP90. In fact, newly translated thyroid receptors do not form complexes with HSP90 in an in vitro system, whereas newly translated GCR does (Dalman et al., 1990). The intracellular distribution of the steroid hormone receptor superfamily proteins appears to be determined by the association with HSP90 and by the ability to interact with the cytoskeletal system through the HSP90 moieties.

Several physiologically important proteins other than the cytosolic steroid hormone receptors and the dioxin receptors have also been shown to be associated with HSP90. They include tyrosine kinase oncogene products such as pp60^src (Brugge et al., 1981; Opperman et al., 1981; Schuh et al., 1985), and eIF2a kinase (Rose et al., 1987; Matts and Hurst, 1989), a serine/threonine-specific protein kinase. These facts suggest that HSP90 serves as a common carrier protein for biologically key functional molecules. It would be of interest to examine whether the complexes of the above kinases with HSP90 interact with actin filaments.

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