HspBP1, an Hsp70 Co-chaperone, Has Two Structural Domains and Is Capable of Altering the Conformation of the Hsp70 ATPase Domain*

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We present here the first structural information for HspBP1, an Hsp70 co-chaperone. Using circular dichroism, HspBP1 was determined to be 35% helical. Although HspBP1 is encoded by seven exons, limited proteolysis shows that it has only two structural domains. Domain I, amino acids 1–83, is largely unstructured. Domain II, amino acids 84–359, is predicted to be 43% helical using circular dichroism. Using limited proteolysis we have also shown that HspBP1 association changes the conformation of the ATPase domain of Hsp70. Only domain II of HspBP1 is required to bring about this conformational change. Truncation mutants of HspBP1 were tested for their ability to inhibit the renaturation of luciferase and bind to Hsp70 in reticulocyte lysate. A carboxyl terminal truncation mutant that was slightly longer than domain I was inactive in these assays, but domain II was sufficient to perform both functions. Domain II was less active than full-length HspBP1 in these assays, and addition of amino acids from domain I improved both functions. These studies show that HspBP1 domain II can bind Hsp70, change the conformation of the ATPase domain, and inhibit Hsp70-associated protein folding.

The Hsp70 family of proteins are key components for the response of the cell to stress, as well as for housekeeping functions such as protein folding and signal transduction (1–3). The proteins of this highly conserved family have two structural domains, an amino-terminal ATPase domain and a carboxyl-terminal substrate-binding domain. The two domains of Hsp70 proteins work in a coordinated fashion. The binding and hydrolysis of ATP by the ATPase domain affects the ability of the substrate-binding domain to bind to short hydrophobic sequences on client proteins. Similarly the binding of substrate by the substrate-binding domain stimulates the ATPase activity of the ATP binding domain. The ATPase domain is composed of two large subdomains of approximately equal size, designated I and II. Nucleotides are bound at the base of the large cleft between the subdomains. Each subdomain is further divided into two additional subdomains, designated A and B (4, 5).

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To adapt this mechanism of action to a specific function or cellular milieu, a number of accessory proteins modulate Hsp70 activity. The DnaJ protein family acts in a catalytic fashion to facilitate ATP hydrolysis coordinated to substrate binding. Another category of accessory proteins is nucleotide exchange factors. In prokaryotes, GrpE promotes the dissociation of ADP and the rebinding of ATP to DnaK (the prokaryotic homologue of Hsp70) (5). In eukaryotic cytosol the most extensively studied nucleotide exchange factor is Bag1. Comparison of cocrytsals of GrpE bound to the DnaK ATPase domain with cocrytsals of the Bag domain bound to the Hsc70 ATPase domain shows that, although GrpE and Bag1 have no structural similarity, they induce the same conformational switch in their Hsp70 family partner to promote nucleotide exchange (6). These nucleotide exchange factors promote an open conformation of the ATPase domain, which is characterized by a 14° rotation of subdomain IIB outward along with a reorientation of the amino terminus in subdomain IA. These two regions are thought to be critical for orienting the nucleotide moiety and act together like a clamp.

HspBP1 is a nucleotide exchange factor found in mammalian cytosol. HspBP1 has been shown to bind to the ATPase domain of Hsp70 and inhibit its ATPase activity as well as its ability to re-potent luciferase (7). Recently HspBP1 has also been shown to promote nucleotide dissociation from Hsc70 (8). There is growing evidence that HspBP1 is a member of a new class of nucleotide exchange factors. HspBP1 has been shown to be a homologue of the yeast nucleotide exchange factors fes1p and sls1p that are found in the cytosol and endoplasmic reticulum, respectively (8–10). HspBP1 also has significant similarity to Slilp, the human homologue of yeast sls1p (11). BAP, a protein found in mammalian endoplasmic reticulum, also acts as a nucleotide exchange factor and shares some sequence homology with HspBP1 (10, 12). There has been no structural information reported for HspBP1, though it has been predicted to have two regions that have similarity to armadillo repeats (12).

In this study, using limited proteolysis, we define two structural domains of HspBP1. We also show that interaction of HspBP1 or domain II (the proteolytically stable domain of HspBP1) with the Hsp70 ATPase domain is capable of changing the conformation of the ATPase domain. We further evaluate truncation mutants of HspBP1 for their ability to bind to Hsp70 and to inhibit luciferase renaturation. Domain II is sufficient for both these functions, but no mutant tested was as potent as full-length HspBP1.

EXPERIMENTAL PROCEDURES

Construction of Mutants—To construct the HspBP1 mutants, new stop and start sites were engineered using PCR mutagenesis. One mutagenic primer (to add the ATG or stop codon) and an internal primer were used. The PCR product was then subcloned into pCR2.1
Circular Dichroism—Circular dichroism spectra of His$_4$-tagged recombinant HspBP1 and recombinant M84–359 were acquired using an AVIV 60DS (V4.1t) spectropolarimeter at 15 °C. The proteins were first exposed to 5 mM dithiothreitol in buffer A for 24 h and then dialyzed into 5 mM phosphate buffer, pH 7.5, over 48 h in the cold. The samples were then diluted to −10 μM. After analysis the concentration of the protein in the samples was determined more accurately using the absorbance at 280 nm and the molar extinction coefficients. The final concentrations were 13.7 μM for HspBP1 and 12.6 μM for M84–359. Spectra were acquired using 0.5-mm steps from 200 to 260 nm and a 1-mm path length. Each data set represents three repetitions. A separate spectrum was generated for the buffer alone, and this was subtracted from the protein spectra. The α-helical content was predicted using the mean residual molar ellipticities ([θ] in degrees cm$^2$ per dmole) and calculated according to the following standard equation (15): Fraction of α-helical content = (10θ222 + 23400)/30 000. The fraction was then multiplied by 100 and expressed as percent α-helical content.

Renaturation of Luciferase—Assays to measure renaturation of luciferase in rabbit reticulocyte lysate were done as previously described (7). The denatured luciferase was added to reticulocyte lysate in the presence of ATP on ice the resin was pelleted and the supernatant removed. To the resulting resin pellet, 50 μl of reticulocyte lysate containing an ATP regenerating system (16) was added. After a 90-min incubation on ice with frequent gentle mixing, the resin pellet was washed four times with ice-cold buffer C (10 mM Tris-HCl, pH 7.9, 1 mM MgCl$_2$, 50 mM KCl, 7.5 mM imidazole, and 0.2% Tween). After the last wash the pellet was taken to dryness, and SDS sample buffer was added and heated to 95 °C for 5 min. The samples were then Western blotted as described above.

**ATPase Binding to HspBP1 and Truncation Mutants**—Incubation of test proteins with the TALON resin, incubation conditions, and washing steps are all as described above for binding of full-length Hsp70. $^{35}$S-labeled ATPase domain was prepared using the TNT T7 quick-coupled transcription/translation system (Promega, Madison, WI) and $^{35}$Simethionine (Amersham Biosciences). The resulting $^{35}$S-labeled ATPase domain was diluted (1:2.67) with reticulocyte lysate containing an ATP regenerating system. $^{35}$S of this mixture was incubated with the TALON resin and test protein. The results were monitored using autoradiography.

**RESULTS**

The Genomic Sequence of HspBP1 Predicts Seven Protein Domains—As a starting point for our studies, we first constructed a model of HspBP1 using the exon boundaries as deduced from the genomic sequence to divide the protein sequence into domains. This type of model of HspBP1 (Fig. 1A, model a) was constructed from information on the human genomic sequence (National Center for Biotechnology, www.ncbi.nlm.nih.gov), revealing that the HspBP1 protein is made from seven exons.

**HspBP1 Has One Structured Domain as Defined by Limited Proteolysis**—To experimentally define the structural domains of HspBP1 and test the model that arose from the genomic sequence, we used limited proteolysis to probe HspBP1 higher order structure. Recombinant HspBP1 was subjected to proteolysis under mild conditions with three different proteases, trypsin, chymotrypsin, and proteinase K. All three proteases produced essentially the same results, so only the results for one, chymotrypsin, are shown (Fig. 1B). The proteases rapidly (−5 min) cleaved HspBP1 to a stable fragment that has an apparent mass of 10 kDa from the parent compound. This protected fragment was stable for up to an hour in the presence of chymotrypsin and trypsin and for 30 min when proteinase K was used. Further degradation produced no discernable fragments. Western blot analysis with an antibody recognizing the His$_4$ tag on the amino terminus showed that the amino termi-

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1. The abbreviations used are: MALDI/TOF, matrix-assisted laser desorption/time-of-flight; ESI-LC/MS, electrospray ionization-liquid chromatography/mass spectrometry.
2. P. R. Baker and K. R. Clausen, prospector.ucsf.edu.
that the stable proteolytic fragment encompasses amino acids digested for 60 min with chymotrypsin. These analyses indicate man degradation and LC/MS to analyze samples of HspBP1 (not shown).

that this epitope was preserved in the stable fragment (data raised against the most carboxyl third of HspBP1, indicated Western blot analysis with the HspBP1 antibody, a monoclonal nus was not preserved in the stable fragment (data not shown).

To identify the stable proteolytic fragment we employed Edman degradation and LC/MS to analyze samples of HspBP1 digested for 60 min with chymotrypsin. These analyses indicate that the stable proteolytic fragment encompasses amino acids 84–359. Five rounds of Edman degradation on the digested fragment produced the amino acid sequence RGGRE, which corresponds to amino acids 84–88 of the HspBP1 sequence. This is at a predicted chymotrypsin cleavage site. ESI-LC/MS of this same time point predicts a mass of 30,918 Da, which corresponds to a fragment of amino acids 84–359 (predicted mass of 30,925 kDa). The spectrum for the proteolytic fragment is shown in Fig. 4B. Furthermore, a recombinant truncated version of HspBP1, M84–359, that had only the stable structured domain with an amino His6 tag, was expressed, purified, and subjected to proteolysis by chymotrypsin. After 60 min of exposure of M84–359 to chymotrypsin, the relative mobility was only slightly altered (Fig. 5C), possibly from removal of the His6 tag, further confirming that this part of HspBP1 is impervious to proteolysis.

From these analyses a new domain model of HspBP1 was constructed with two domains (Fig. 1A, model b). In this model the proteolytically stable structural domain is domain II, and the portion of HspBP1 removed by proteolysis is domain I. The region encoded by domain I in model b is similar to domain I in model a. From this point on, domain I will refer to amino acids 1–83, and domain II will refer to amino acids 84–359 as shown in model b.

The Structural Conformation of HspBP1 Is Predicted to be 35% Helical—Structural prediction programs suggest that HspBP1 has large helical regions. To test these predictions we have used circular dichroism to analyze the helical content of HspBP1 and the recombinant version of the stable proteolytic fragment, M84–359 (Fig. 2). Analysis of these data resulted in a predicted conformation (including amino-terminal His6 tags) for HspBP1 that is 35% helical and for the stable proteolytic fragment M84–359 that is 43% helical.

Association with HspBP1 Renders the Hsp70 ATPase Domain Susceptible to Limited Proteolysis—Limited proteolysis of HspBP1 and the Hsp70 ATPase domain was performed to assess the interaction of the two proteins. As previously reported, the Hsp70 ATPase domain alone was not digested by a 60-min treatment with chymotrypsin (Ref. 17 and data not shown). Preincubation of the Hsp70 ATPase domain with HspBP1, on the other hand, renders it susceptible to proteolysis (Fig. 3, A and B). It also appears to slow down the proteolysis of HspBP1 somewhat as shown in Fig. 3C but does not change the proteolytic sites. Preincubation on ice or at room temperature, as well as the addition of 3 mM MgCl2, 50 mM KCl, or 0.5 mM ATP (either to the ATPase domain prior to HspBP1 addition or at the time of addition) did not change the outcome of proteolysis as assessed by Coomassie Blue-stained gels of samples after 60 min of digestion (data not shown). The identity of the protein fragments was determined by mass spectrometry of limited proteolysis reactions after 5 and 60 min of proteolysis. Samples were analyzed using MALDI/TOF and ESI-LC/MS. Representative spectra are shown in Fig. 4. Fig. 4A shows the mid-mass range of a MALDI/TOF spectrum of a mixture of HspBP1 and the Hsp70 ATPase domain after 60 min of proteolysis. The full data from this analysis are given in Table I.

As shown in Table I, the areas of the ATPase domain that were susceptible to proteolysis after interacting with HspBP1 were determined to be concentrated mainly in subdomain I but also include the carboxyl terminus. The protected fragment left after proteolysis for 60 min (indicated by c in Fig. 3, A and B) was identified as amino acids 184–371 of the ATPase domain. This protected fragment encompasses the carboxyl terminal half of the ATPase domain with the exception of the last 11 amino acids. These results indicate that the association of the Hsp70 ATPase domain with HspBP1 dramatically changes the conformation of the ATPase domain.

Domain II Alone Is Capable of Rendering the Hsp70 ATPase Domain Susceptible to Limited Proteolysis—Limited proteolysis experiments were performed after preincubating M84–359 with the Hsp70 ATPase domain. The results, shown in Fig. 5, are similar to the results when full-length HspBP1 was employed (note the appearance of bands indicated by b and c in Figs. 3B and 5B). ESI-LC/MS of a 60-min sample produced a similar pattern of Hsp70 ATPase fragments, as shown in the last column of Table I. These results indicate that domain II is
sufficient to alter the conformation of the Hsp70 ATPase domain.

Neither Domain II nor Other HspBP1 Mutants Are Capable of Fully Recapitulating the Inhibition of Luciferase Refolding of HspBP1—Next we sought to evaluate whether domain II or other HspBP1 truncation mutants were sufficient for other HspBP1 activities. The initial characterization of HspBP1 showed that it was a potent inhibitor of luciferase renaturation in reticulocyte lysate (7). To determine which part of HspBP1 was necessary for this function, we tested the ability of five mutants to inhibit luciferase renaturation in reticulocyte lysate. For these experiments cDNAs for truncation mutants of HspBP1 were constructed, expressed, and purified. One of these mutants, M1–138, was truncated at the carboxy terminus of HspBP1 at amino acid 138. This mutant fully encompasses domain I (amino acids 1–83). The other four mutants are truncated from the amino terminus and are as follows: M51–359 starting at amino acid 51; M70–359 starting at amino acid 70, which begins with the first amino acid encoded by exon 1; the aforementioned M84–359, which is domain II only; and M301–359, which starts at amino acid 301. The results of these assays are shown in Fig. 6. Although the amino truncation mutants M51–359, M70–359, and M84–359 demonstrated the ability to inhibit luciferase renaturation, none of the mutants was as potent as HspBP1. One explanation for these findings could be that the placement of the His6 tag on the amino terminus of the mutants was affecting their function. The removal of the His6 tag from the stable proteolytic domain M84–359 produced no change in its activity in these assays (data not shown). To determine whether the ability to inhibit renaturation was in the amino portion of HspBP1 that was being removed in these mutants, M1–138 was tested. It had no activity, indicating that this part of HspBP1 alone was not capable of inhibition of luciferase renaturation. The other mutant, M301–359, had no activity as well.

Ability to Inhibit Luciferase Refolding Correlates with the Ability to Bind Hsp70—The same panel of mutants was then tested for their ability to bind endogenous Hsp70 in reticulocyte lysate as shown in Fig. 7A. The mutants were immobilized by means of their His6 tags on TALON resin and incubated with reticulocyte lysate containing an ATP regenerating system. The results of these assays correlate well with the results of the luciferase renaturation assay. M301–359, which showed no activity in the luciferase renaturation assay, showed no binding of Hsp70. M1–138, which also had no activity in the luciferase renaturation assay, had weak binding of Hsp70. M301–359 of HspBP1, as the amino terminus was truncated, the binding of Hsp70 fell dramatically. To rule out their binding as substrates, the mutants were also tested to see whether they were binding to the ATPase domain of Hsp70 even though this should have been prevented by ample amounts of ATP and an ATP regenerating system in the lysate. For these assays the ATPase domain was transcribed and translated in reticulocyte lysate using [35S]-labeled methionine. As shown in Fig. 7B, with the exception of M1–138 the mutants that demonstrated Hsp70 binding also bind the ATPase domain. M1–138 showed weak binding of full-length Hsp70 and no binding of the ATPase domain. M301–359, which showed no ability to bind Hsp70, also does not bind the ATPase domain. This assay precludes quantification because the [35S]-ATPase domain is competing with endogenous Hsp70 for binding to HspBP1 or the trun-
HspBP1 Structure/Function and Hsp70 Conformational Change

Table I

| Assignment | MALDI/TOF | ESI-LCMS | Other samples containing fragment |
|------------|-----------|-----------|----------------------------------|
|            | M_{obs}   | z         | % Error  | M_{obs}                     | % Error  | A       | B       | C       | AC       |
| A 108-183  | 8234      | 1         | 0        | 8236                      | 0.02     |         | x       |         |         |
| A 93-183   | 9905      | 1         | -0.06    | 11551                     | -0.04    |         |         |         |         |
| A 79-183   | 11534     | 1         | -0.02    | 12134                     | -0.03    |         |         |         |         |
| A 74-183   | 12133     | 1         | -0.04    | 12716                     | -0.05    |         |         |         |         |
| A 69-183   | 12718     | 1         | -0.03    | 21144                     | -0.02    |         |         |         |         |
| A 184-371  | 21147     | 1         | -0.01    | 30918                     | -0.02    | x       | x       | x       |         |
| A 184-371  | 21140     | 2         | -0.04    | 30918                     | -0.02    |         |         |         |         |
| B 84-359   | 30879     | 1         | -0.12    | 37906                     | -0.03    |         |         |         |         |
| B 74-359   | 31735     | 1         | -0.08    | 41770                     | -0.01    | x       | x       |         |         |
| B 69-371   | 33745     | 1         | -0.37    |                         |         |         |         |         |         |
| B 14-359   | 37787     | 1         | -0.34    |                         |         |         |         |         |         |
| A 2-382    | 41770     | 1         | -0.27    |                         |         |         |         |         |         |

![Fig. 5. Limited proteolysis of HspBP1 domain II and the Hsp70 ATPase domain.](image)

![Fig. 6. Inhibition of luciferase renaturation by full-length and truncation mutants of HspBP1.](image)

![Fig. 7. Binding of HspBP1 to Hsp70 and the Hsp70 ATPase domain.](image)

**DISCUSSION**

HspBP1 is a member of an emerging family of proteins that modulate Hsp70 activity. To date, nothing has been known about its protein structure. The work reported here shows that HspBP1 has two structural domains as defined by limited proteolysis. In regard to the previously reported activities of HspBP1, inhibition of Hsp70 renaturation of luciferase and Hsp70 binding, the full-length protein is required for full activity, but domain II is sufficient for activity in both assays. We also present here a novel activity for HspBP1, which is the ability to change the conformation of the ATPase domain of Hsp70. For this activity HspBP1 is modular and domain II is sufficient.

As a starting point for these studies we constructed a model of HspBP1 using human genomic information. Seven exons make up the mRNA for the coding region of HspBP1. The model that we have developed using limited proteolysis shows that HspBP1 has two structural domains. Domain I encompasses the first 83 amino acids (~10 kDa) and domain II, amino acids 84–359 (~30kDa). Domain I is susceptible to limited proteolysis and thus predicted to consist mainly of random coil. Domain II, with an amino-terminal His6 tag, is predicted by circular dichroism to be 43% helical and is impervious to proteolysis under native conditions.

[1] A 93-183 9905 1
[2] A 79-183 11534 1
[3] A 74-183 12133 1
[4] A 69-183 12718 1
[5] A 184-371 21147 1
[6] A 184-371 21140 2
[7] B 84-359 30879 1
[8] B 74-359 31735 1
[9] B 69-371 33745 1
[10] B 14-359 37787 1
[11] A 2-382 41770 1

**TABLE I**

Listed are the observed fragments from MALDI/TOF analysis of a sample after 60 min of digestion. In the first column the assignment of the fragment is given, with A indicating the Hsp70 ATPase domain, B indicating HspBP1, followed by the corresponding amino acid numbers. M_{obs} is the observed mass in kDa, z is the charge of the observed ion, and % error is the difference between M_{act} and the actual mass expressed as a percent of the actual mass. The results of ESI-LCMS analysis performed on the same sample are given to provide confirmation and higher accuracy determination of larger masses. The columns to the right indicate which other 60-min samples contained the same proteolytic fragment. A is the HSP70 ATPase domain alone; B is HspBP1 alone, and C is M84–359 alone. AC is the Hsp70 ATPase domain preincubated with M84–359.
We have also used limited proteolysis to show that the ATPase domain of Hsp70 changes its conformation in response to HspBP1 exposure. The factors that make a site susceptible to limited proteolysis are thought to be flexibility and accessibility (18). After exposure to HspBP1, the Hsp70 ATPase domain becomes susceptible to proteolysis at five sites in subdomain IB, one site in subdomain IA, and at the carboxyl terminus. This leaves a protected fragment of the ATPase domain after 60 min of proteolysis that consists of almost the entire subdomain II. The method that we have employed here does not exclude the possibility that other conformational changes in the ATPase domain take place. For example, the physical association of HspBP1 with regions undergoing a conformational change could protect regions of the ATPase domain from proteolysis. It will be interesting to see whether the conformational changes brought about by HspBP1 association are different from the open conformation seen with Bag1 and GrpE binding. We must wait for more definitive structural information before that can be determined.

Domain II of HspBP1 is sufficient to carry out all the activities of HspBP1 that we tested for, namely, inhibition of renaturation of luciferase, binding to Hsp70 in reticulocyte lysate, and alteration of the conformation of the Hsp70 ATPase domain. A carboxyl terminal truncation mutant containing all of domain I and 65 additional amino acids from domain II shows no ability to inhibit luciferase renaturation and only weakly binds to Hsp70, suggesting that domain I is not sufficient for these activities. Domain II is less active than full-length HspBP1 in the inhibition of renaturation of luciferase and binding to Hsp70. This suggests that domain I promotes a tighter association of Hsp70 with HspBP1. We tested two other amino-terminal truncations that contained domain II and additional amino acids from domain I. The assays performed with these mutants show that each successive addition of amino acids from domain I to domain II improves the activity of the protein. The hypothesis most consistent with our data is that domain I binds to Hsp70 with a low affinity, which increases the affinity of HspBP1 for the ATPase domain of Hsp70. Further structural studies are needed to understand these issues fully.

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