Cloning and Expression of Murine High Molecular Mass Heat Shock Proteins, HSP105*

(Received for publication, July 27, 1995, and in revised form, September 5, 1995)

Kunihiko Yasudaši, Akira Nakaiši, Takumi Hayatamaiši, and Kazuhiro Nagatasiši

From the 1Department of Biochemistry, Kyoto Pharmaceutical University, Yamashina-ku, Kyoto 607 and the 2Department of Cell Biology, Chest Disease Research Institute, Kyoto University, Sakyo-ku, Kyoto 606-01, Japan

We have shown that the 105-kDa heat shock protein (HSP105) and the 42 °C-specific heat shock protein (42 °C-HSP) constitute high molecular mass heat shock proteins. To elucidate the structure of these heat shock proteins, we have screened a cDNA library constructed with poly(A)* RNA derived from mouse FM3A cells preheated at 42 °C for 2 h using an antibody against murine HSP105. Two full-length cDNA clones were obtained: the pB105-1 insert encoded an 858-amino acid protein, and the pB105-2 insert encoded an 814-amino acid protein and lacked 44 amino acids found in pB105-1. The two clones contained the amino acid sequence found in the 17-kDa polypeptide fragments from HSP105 and 42 °C-HSP by lysylendopeptidase digestion. In vitro translation products of the RNA transcripts from pB105-1 and pB105-2 migrated to the same positions of HSP105 and 42 °C-HSP, respectively, on SDS-polyacrylamide gel electrophoresis. Northern blot analysis showed that the transcript was ~4 kilobases in murine FM3A cells and was strongly induced by heat shock and by treatment with arsinite or an amino acid analog. By reverse transcription-polymerase chain reaction analysis using primers by which deletion of 132 nucleotides in pB105-2 could be detected, the polymerase chain reaction product corresponding to pB105-2 was increased only after heat shock at 42 °C, whereas the product corresponding to pB105-1 was induced by heat shock at either 42 or 45 °C and also by other stresses. Thus, the cDNA clones pB105-1 and pB105-2 encode HSP105 and 42 °C-HSP, respectively, and HSP105 and 42 °C-HSP (a short form of HSP105) are suggested to be produced by alternative splicing. Here, HSP105 and 42 °C-HSP are renamed HSP105a and HSP105b, respectively. A protein sequence homology search revealed that HSP105 shares 54, 34, and 25% amino acid identity with human HSP70R, the sea urchin egg receptor for sperm, and murine inductible HSP70, respectively. Furthermore, by Northern blot analysis, HSP105 mRNA was revealed to be present in most murine tissues and to be highly expressed in the brain.

Upon exposure to heat shock, living cells from bacteria to humans synthesize a set of proteins called heat shock proteins (HSPs).1 Since HSPs are also induced by a variety of stresses, these proteins are also called stress proteins. However, HSPs are expressed in considerable amounts in nonstressed cells. HSPs are involved not only in cell protection and repair of cell damage caused by a variety of stresses, but also in normal cellular functions (for recent reviews, see Refs. 1–3). These proteins have been classified into several families according to their apparent molecular mass: high molecular mass HSP, HSP90, HSP70, HSP60, HSP47, and low molecular mass HSP. The families such as HSP70, HSP60, and HSP90 have been studied extensively. These proteins are found to interact with other proteins to mediate protein folding, unfolding, assembly, and disassembly of proteins as molecular chaperones.

The members of the mammalian high molecular mass HSP family are not well characterized, although yeast HSP104 has been cloned and sequenced (4, 5). Deletion of the HSP104 gene has little effect on growth at a normal temperature, and the cells die at the same rate as wild-type cells when exposed to a high temperature. However, the mutant cells do not acquire tolerance to heat and other forms of stress (4–6). Interestingly, HSP104 exhibits homology to bacterial ClpA/ClpB proteins, which appear to play a role in an ATP-dependent protein degradation process (7). Recently, ClpA was found to function as a molecular chaperone like DnaK (8).

The 110-kDa HSP (HSP110) from Chinese hamster ovary cells (9, 10) and 105-kDa HSP from murine cells (11, 12) have been reported to be the mammalian high molecular mass HSP. Chinese hamster HSP110 was found to be localized in the nuclei of nonstressed and heat-stressed murine cells by indirect immunofluorescence (9). Since HSP110 is associated with actively transcribed rRNA genes by immunoelectron microscopy, HSP110 was inferred to participate in ribosome assembly (10). On the other hand, by indirect immunofluorescence, we have shown that HSP105 is localized to the cytoplasm and nuclei under both nonstressed and stressed conditions in murine cells and has never been found in the nuclei, unlike HSP110 (11). Furthermore, we have found a specific 90-kDa HSP that is synthesized only when mammalian cells are heat-shocked continuously at 42 °C (42 °C-HSP) (13). An antibody to HSP105 serum reacts not only with HSP105, but also with 42 °C-HSP (11, 14). By amino acid sequence analysis, HSP105 and 42 °C-HSP were found to be similar proteins containing a particular sequence similar to an adenosine-binding domain of HSP70 family proteins and actin (12).

Herein, we report the cloning of HSP105 and 42 °C-HSP from murine cells and reveal that HSP105 and 42 °C-HSP are highly homologous to human HSP70R and moderately homologous to the sea urchin egg receptor for sperm and less so to...
murine inducible HSP70. The N-terminal half of these proteins contained an ATP-binding domain similar to that of HSP70 family members and was more conserved among these proteins. Northern blot analysis of various murine tissues revealed that HSP105 mRNA was present in most murine tissues and was highly expressed in the brain.

**EXPERIMENTAL PROCEDURES**

Construction and Screening of a cDNA Library—Mouse mammary carcinoma F3MA cells (supplied by the Japan Cancer Research Resource Bank) were maintained in Eagle’s minimal essential medium supplemented with 10% calf serum (Life Technologies, Inc.). Total RNA was isolated from F3MA cells pretreated at 42°C for 2 h by guanidine thiocyanate/phenol/chloroform method (18). Ten micrograms of poly(A) RNA derived from total RNA was used for in vitro transcription in a rabbit reticulocyte lysate system (Promega). The products were electrophoresed on a 3.5% polyacrylamide gel, and the gel was dried and autoradiographed at −80°C. Molecular size markers (100-base pair DNA ladder, Life Technologies, Inc.) were used for calibration of the sizes of the PCR products.

**RESULTS**

Cloning and Nucleotide Sequence of HSP105—The cDNA library was constructed with poly(A)+ RNA derived from mouse F3MA cells that were treated at 42°C for 2 h. The cDNA library was screened using an anti-mouse HSP105 antibody (11, 14). Among several reactive clones, one clone (ES105) with an −1.5-kb insert was isolated. The partial cDNA insert of ES105 was used to further screen the library, which resulted in two full-length cDNA clones. Nucleotide sequence analysis revealed that pB105-1 was 3345 nucleotides long and had a single open reading frame of 858 amino acids beginning with the ATG codon at nucleotide 55 and terminating with the in-frame TAG codon at nucleotide 2628 (Fig. 1). The other clone, pB105-2, was 3268 nucleotides long and had a single open reading frame of 814 amino acids beginning with the ATG codon at nucleotide 142 and terminating with the in-frame TAG codon at nucleotide 2584. pB105-2 lacked the region between nucleotides 1642 and 1773 of pB105-1 (boxed in Fig. 1). Both clones contained the predicted amino acid sequence (shaded in Fig. 1) found in the 17-kDa polypeptide fragments of HSP105 and 42°C-HSP generated by lysylendopeptidase digestion (12).

Structural Features of HSP105—A homology search for protein sequence similarities revealed that murine HSP105 was highly homologous to human HSP70RY and the sea urchin egg receptor for sperm (Fig. 2). Among these proteins, two regions were highly identical: the N-terminal region between amino acids 1 and 500 and the region between amino acids 612 and 713. The N-terminal region of murine HSP105 contains a single ATP-binding site consisting of the conserved VVGLDVGVS (amino acids 3–10), EKLK (amino acids 271–274), IEIVG-GATRIPAVKE (amino acids 338–352), and D (amino acid 369) sequences (underlined in Fig. 1) (21). Since HSP105 and 42°C-HSP clearly contained an ATP-binding domain, we examined the ATP binding ability of HSP105 and 42°C-HSP by ATP-agarose column chromatography, but these proteins were found not to bind to the column under the conditions used for ATP binding of HSP70 (data not shown) (22).

Murine HSP105 was 54% identical to human HSP70RY (23, 24) and 34% to the sea urchin sperm receptor (25) and only 25% to murine inducible HSP70 (26). In general, HSP70 family members are conserved in the N-terminal ATP-binding domain and less so in the C-terminal putative peptide-binding domain (27–29). The N-terminal 500-amino acid sequence of HSP105 that contained an ATP-binding domain was more homologous to those of human HSP70RY (72% identity) and the sea urchin egg receptor (50%) and less so to that of human HSP70 (32%). Another region between positions 612 and 713 of HSP105 was also highly homologous to HSP70RY (amino acids 594–695; 66% identity) and the sea urchin sperm receptor (amino acids 649–750; 49% identity), but the homologous region was not found in murine inducible HSP70.

In Vitro Transcription and Translation of pB105—To examine protein products of pB105-1 and pB105-2, these cDNAs were transcribed in vitro with bacteriophage RNA polymerase and translated in rabbit reticulocyte lysate. The resulting [35S]methionine-labeled proteins were analyzed by SDS-PAGE (Fig. 3). The translation products of pB105-1 and pB105-2 were both separated to three protein bands, and the largest proteins of the translation products migrated to the same positions as

Tag DNA polymerase for 18 cycles (each cycle consisted of denaturation at 94°C for 30 s, annealing at 60°C for 30 s, and extension at 72°C for 1.5 min) in the presence of [α-32P]dCTP (3000 Ci/mmol; Amersham Corp.). The products were electrophoresed on a 3.5% polyacrylamide gel, and the gel was dried and autoradiographed at −80°C. Molecular size markers (100-base pair DNA ladder, Life Technologies, Inc.) were used for calibration of the sizes of the PCR products.
HSP105 and 42 °C-specific HSP, respectively, indicating that pB105-1 and pB105-2 encode HSP105 and 42°C-HSP, respectively.

Stress Inducibility of HSP105 mRNA—We next examined whether the level of HSP105 mRNA is affected by various stresses. First, mouse FM3A cells were incubated at 42 °C for 15–300 min or were incubated at 37°C after exposure to heat shock at 42°C for 60 min or at 45°C for 15 min, and total cellular RNA was analyzed by Northern blotting. Using pB105-2 as a probe, 4-kb RNA species were detected in both RNAs from nontreated and heat-treated FM3A cells (Fig. 4A). HSP105 mRNA began to increase after heating the cells for 60 min at 42°C and markedly increased after 5 h. When FM3A cells that were heated at 42°C for 60 min were incubated at 37°C, HSP105 mRNA decreased to basal levels after 3 h. The onset of the increase of HSP105 mRNA at 42°C was significantly delayed compared with that at 45°C. These results indicate that HSP105 mRNA is induced by heat shock at 42°C.

Fig. 1. Nucleotide sequence and predicted amino acid sequence of pB105-1. The top line shows the nucleotide sequence of the cDNA for HSP105, and the second line shows the predicted amino acid sequence. The shaded amino acids were determined by amino acid sequencing of HSP105 family members. The boxed nucleotides represent the spliced region of pB105-2. The underlined amino acids indicate the predicted ATP-binding domain of HSP70 family members. The nucleotides underlined by broken lines were used as primers for RT-PCR.
Fig. 2. Amino acid sequence homology between mouse HSP105 and human HSP70RY, sea urchin egg receptor, and mouse HSP70.

Region I of mouse HSP105, which contained an ATP-binding domain, is highly similar in these four proteins, whereas region III is similar in HSP105, HSP70RY, and the sperm receptor, and the identity of these regions is shown in the respective boxes. Region II represents the spliced-out region in pH105-2. The identity between HSP105 and other proteins is shown at the right. Numbers on the top indicate the positions of amino acid residues for each protein.

Fig. 3. Transcription and translation of pH105-1 and pH105-2.

A, 20 μg of cell extracts from control FM3A cells (lane 1) or cells heat shocked at 42 °C for 4 h (lane 2) was subjected to 8% SDS-PAGE and detected immunologically using anti-HSP105 antibody. B, linearized pH105-1 (lane 3) and pH105-2 (lane 2) were transcribed in vitro with T3 RNA polymerase, and the translation products were translated in rabbit reticulocyte lysate in the presence of [35S]methionine. Aliquots (3 μl) of translation mixture (25 μl) were subjected to 8% SDS-PAGE and visualized by autoradiography. Lane 1 represents the translation product without these plasmids. Molecular masses of marker proteins are shown at the left, and the upper and lower arrows indicate the positions of HSP105 and 42 °C-HSP, respectively.

37 °C, the mRNA increased significantly even after 15 min. By contrast, there is a significant increase in the amount of HSP70 mRNA after 15 min during continuous heating at 42 °C. As reported previously (30), the maximum induction of HSP70 mRNA was observed after 120 min of heat shock. When FM3A cells were incubated at 37 °C after heat shock at 45 °C for 15 min, HSP105 mRNA began to increase at 3 h, while HSP70 mRNA increased at 1 h (Fig. 4B). Furthermore, when FM3A cells were treated with 100 μM arsenite, HSP105 mRNA significantly increased at 1 h, and HSP70 mRNA also increased at 1 h after exposure to arsenite (Fig. 4C). Both HSP105 and HSP70 mRNAs increased at 3 h after exposure to 20 mM azetidine-2-carboxylic acid (Fig. 4D). Actin mRNA did not change significantly under these conditions.

RT-PCR Analysis—Since we had done two species of cDNAs (pB105-1 and pB105-2), we next performed RT-PCR analysis using primers by which the deletion of 132 nucleotides in pB105-2 could be detected. These primers were supposed to give PCR products of 410 and 298 nucleotides for the transcripts corresponding to pB105-1 and pB105-2, respectively. As shown in Fig. 5, two PCR products of approximately 410 (band a) and 280 (band b) nucleotides were detected in total RNAs prepared from cells treated at 42 °C. Bands a and b increased approximately 2- and 3-fold, respectively, after incubation at 42 °C for 5 h (Fig. 5, lanes 1-5), and the increase in the sum of both bands was similar to that of HSP105 mRNA analyzed by Northern blot analysis (Fig. 4). On the other hand, only one product (band a) was observed in the RNA prepared from cells treated at 45 °C (Fig. 5, lanes 6-10). Under these conditions, band a increased drastically as observed by Northern blot analysis. Treatment of the cells with arsenite or azetidine-2-carboxylic acid did not induce band b, while it induced a dramatic increase in band a (Fig. 5, lanes 11 and 12, respectively).

Since mRNA from pB105-2 was only induced by heat shock at 42 °C, pB105-2 was supposed to encode 42 °C-HSP.

Tissue Specificity of HSP105 Expression—To determine whether HSP105 mRNA is expressed in murine tissues, total cellular RNAs from various murine tissues were analyzed by Northern blotting using pB105-2 as a probe (Fig. 6). Four-kilobase species, as observed in FM3A cells, hybridized to the probe in RNA from almost all tissues. HSP105 mRNA was highly expressed in the brain and moderately expressed in the lungs, heart, thymus, spleen, liver, and small intestine. These findings were also confirmed by Western blot analysis.2

DISCUSSION

We have previously reported that HSP105 and 42 °C-HSP, which are serologically related to each other, have an ATP-binding domain similar to that of the HSP70 family and actin (11–14). Here, we describe the cloning of the cDNAs encoding HSP105 and 42 °C-HSP from mice.

We have screened a cDNA library constructed with mRNA from heat-shocked mouse FM3A cells using an anti-HSP105 antibody and have obtained two full-length cDNA clones, pB105-1 consists of 858 amino acids, and pB105-2 encodes 814 amino acids. Both clones were exactly the same except that pB105-1 lacked 44 amino acids in the region between positions 530 and 573 of pB105-1, and both contained the amino acid sequence found in the 17-kDa polypeptide fragments from HSP105 and 42 °C-HSP digested by lysylendopeptidase (12). From in vitro translation experiments and RT-PCR analysis, the cDNA clones pB105-1 and pB105-2 were suggested to encode HSP105 and 42 °C-HSP, respectively. HSP105 mRNA was strongly induced by heat shock at 42 or 45 °C and by treatment with arsenite or azetidine-2-carboxylic acid (Fig. 4). Furthermore, when FM3A cells were treated with 100 μM arsenite, HSP105 mRNA significantly increased at 1 h, and HSP70 mRNA also increased at 1 h after exposure to arsenite (Fig. 4C). Both HSP105 and HSP70 mRNAs increased at 3 h after exposure to 20 mM azetidine-2-carboxylic acid (Fig. 4D). Actin mRNA did not change significantly under these conditions.

RT-PCR Analysis—Since we had done two species of cDNAs (pB105-1 and pB105-2), we next performed RT-PCR analysis using primers by which the deletion of 132 nucleotides in pB105-2 could be detected. These primers were supposed to give PCR products of 410 and 298 nucleotides for the transcripts corresponding to pB105-1 and pB105-2, respectively. As shown in Fig. 5, two PCR products of approximately 410 (band a) and 280 (band b) nucleotides were detected in total RNAs prepared from cells treated at 42 °C. Bands a and b increased approximately 2- and 3-fold, respectively, after incubation at 42 °C for 5 h (Fig. 5, lanes 1-5), and the increase in the sum of both bands was similar to that of HSP105 mRNA analyzed by Northern blot analysis (Fig. 4). On the other hand, only one product (band a) was observed in the RNA prepared from cells treated at 45 °C (Fig. 5, lanes 6-10). Under these conditions, band a increased drastically as observed by Northern blot analysis. Treatment of the cells with arsenite or azetidine-2-carboxylic acid did not induce band b, while it induced a dramatic increase in band a (Fig. 5, lanes 11 and 12, respectively).

Since mRNA from pB105-2 was only induced by heat shock at 42 °C, pB105-2 was supposed to encode 42 °C-HSP.

Tissue Specificity of HSP105 Expression—To determine whether HSP105 mRNA is expressed in murine tissues, total cellular RNAs from various murine tissues were analyzed by Northern blotting using pB105-2 as a probe (Fig. 6). Four-kilobase species, as observed in FM3A cells, hybridized to the probe in RNA from almost all tissues. HSP105 mRNA was highly expressed in the brain and moderately expressed in the lungs, heart, thymus, spleen, liver, and small intestine. These findings were also confirmed by Western blot analysis.2

DISCUSSION

We have previously reported that HSP105 and 42 °C-HSP, which are serologically related to each other, have an ATP-binding domain similar to that of the HSP70 family and actin (11–14). Here, we describe the cloning of the cDNAs encoding HSP105 and 42 °C-HSP from mice.

We have screened a cDNA library constructed with mRNA from heat-shocked mouse FM3A cells using an anti-HSP105 antibody and have obtained two full-length cDNA clones, pB105-1 consists of 858 amino acids, and pB105-2 encodes 814 amino acids. Both clones were exactly the same except that pB105-1 lacked 44 amino acids in the region between positions 530 and 573 of pB105-1, and both contained the amino acid sequence found in the 17-kDa polypeptide fragments from HSP105 and 42 °C-HSP digested by lysylendopeptidase (12). From in vitro translation experiments and RT-PCR analysis, the cDNA clones pB105-1 and pB105-2 were suggested to encode HSP105 and 42 °C-HSP, respectively. HSP105 mRNA was strongly induced by heat shock at 42 or 45 °C and by treatment
with arsenite or an amino acid analog, whereas 42 °C-HSP mRNA was only induced by heat shock at 42 °C.

The 42 °C-specific HSP consists of at least two polypeptides (basic and acidic) with molecular masses of ~90,000 (31). Pulse-chase experiments suggested that the acidic protein originated from the basic protein by post-translational modification. HSP105 is induced by heat shock either at 42 or 45 °C, whereas 42 °C-HSP is synthesized only when heated continuously at 42 °C (13, 14). Since the amino acid sequences of HSP105 and 42 °C-HSP deduced from the cDNA sequences were the same except that 42 °C-HSP lacked 44 amino acids in the region between positions 530 and 573 of HSP105, 42 °C-HSP is synthesized only when heated continuously at 42 °C. HSP105 is induced by heat shock either at 42 or 45 °C, whereas 42 °C-HSP is synthesized only when heated continuously at 42 °C. HSP105 is induced by heat shock either at 42 or 45 °C, whereas 42 °C-HSP is synthesized only when heated continuously at 42 °C.

Previously, we reported that heat shock induces an alternative splicing in the 5'-noncoding region of the collagen-specific stress protein, HSP47 (32). Similarly to the observation for HSP105 (Fig. 5), the alternative splicing of HSP47 was not observed by treatment with other stress inducers like arsenite (basic and acidic) with molecular masses of ~90,000 (31). Pulse-chase experiments suggested that the acidic protein originated from the basic protein by post-translational modification. HSP105 is induced by heat shock either at 42 or 45 °C, whereas 42 °C-HSP is synthesized only when heated continuously at 42 °C (13, 14). Since the amino acid sequences of HSP105 and 42 °C-HSP deduced from the cDNA sequences were the same except that 42 °C-HSP lacked 44 amino acids in the region between positions 530 and 573 of HSP105, 42 °C-HSP is synthesized only when heated continuously at 42 °C. HSP105 is induced by heat shock either at 42 or 45 °C, whereas 42 °C-HSP is synthesized only when heated continuously at 42 °C. HSP105 is induced by heat shock either at 42 or 45 °C, whereas 42 °C-HSP is synthesized only when heated continuously at 42 °C.
some 6, 14, and 21 (24). The function of HSP70RY is not clear, but it may have distinctive functions in antigen processing and presenting of B lymphocytes. On the other hand, the sea urchin egg receptor for sperm is a transmembrane glycoprotein of 1184 amino acids with a short cytoplasmic domain (25). The extracellular signal-binding domain of the receptor shows sequence similarity to HSP70 family members. The extracellular portion of the receptor binds to the sperm protein, bidein, and also inhibits fertilization in a species-specific manner. In addition, the region between amino acids 612 and 713 of HSP110 was also highly similar to HSP70RY and the sea urchin sperm receptor between amino acids 594 and 695 and between amino acids 649 and 750, respectively, but not to murine inducible receptor between amino acids 594 and 695 and between amino acids 612 and 713 of HSP105. In the sea urchin sperm receptor, the second homologous region between amino acids 594 and 695 and between amino acids 612 and 713 of HSP105 was expressed in most murine tissues and was significantly induced by heat shock. Thus, the APG protein may be a testis-specific member of the HSP105 family in mice, as is testis-specific HSP70 (HSP70T) in the HSP70 family (33).

HSP104 is a high molecular mass heat shock protein in yeast, and the antibody against yeast HSP104 cross-reacts with the high molecular mass heat shock protein from human cells and Chinese hamster cells (5). Yeast HSP104 is a protein of 908 amino acids, and the deduced amino acid sequence revealed the presence of two nucleotide-binding sites in HSP104. Deletion of the HSP104 gene in yeast cells has little effect on growth at a normal temperature, but has an effect on the acquisition of resistance to heat and other various stresses (4–6). The two nucleotide-binding domains of HSP104 are essential for thermotolerance (5). Recently, HSP104 was found to function to resolubilize heat-aggregated proteins (34). Furthermore, yeast HSP104 exhibits homology to bacterial ClpA/ClpB proteins, which appear to play a role in an ATP-dependent protein degradation process (7). In addition, ClpA functions as a molecular chaperone like DnaK (8). However, murine HSP105 did not show any identity to yeast HSP104 or bacterial ClpA/ClpB proteins.

When preparing this report, the cDNA sequence of Chinese hamster HSP105 was found in the GenBank™ protein sequence data base and has recently been reported (35). The cDNA of Chinese hamster HSP105 encodes 858 amino acids, and the predicted amino acid sequence shared 96% identity with murine HSP105c. Chinese hamster HSP110 is a high molecular mass heat shock protein that was first reported by Subjeck et al. (9). This heat shock protein is constitutively expressed at low levels and appears to increase with heat shock. When visualized by indirect immunofluorescence, HSP110 is present in nuclei and essentially in nucleoli. Since HSP110 is released from the nucleoli by RNase treatment, it is probably complexed to the RNA component of the nucleoli and possibly participates in ribosome assembly (9, 10). However, when we carefully examined the cellular distribution of HSP105 by immunofluorescence using the anti-HSP105 antibody, we found that HSP105 is mainly localized in the cytoplasm and nuclei and is never found in the nucleoli in either nonstressed or stressed cells (11). It would also be interesting to clarify the differences between our murine HSP105 and Chinese hamster HSP110.

Acknowledgments—We thank Drs. J. Fujita and Y. Kaneko for providing unpublished data.

REFERENCES

1. Weich, W. J. (1992) Physiol. Rev. 72, 1063–1081
2. Hentrick, J. P., and Hartl, F.-U. (1993) Annu. Rev. Biochem. 62, 349–384
3. Craig, E. A., Weissmann, J. S., and Horwich, A. L. (1994) Cell 78, 365–372
4. Sanchez, Y., and Lindquist, S. L. (1990) Science 248, 1112–1115
5. Parsell, D. A., Sanchez, Y., Stitzel, J. D., and Lindquist, S. (1993) Nature 363, 270–273
6. Sanchez, Y., Taulien, J., Borkovich, K. A., and Lindquist, S. (1992) EMBO J. 11, 2357–2364
7. Gottessman, S., Squires, C., Pichersky, E., Carrington, M., Hobbs, M., Mattick, J. S., Dalrymple, B., Kurumitsu, H., Shirouzu, M., Foster, T.,Clark, W. P., Ross, B., Squires, C. L., and Maurizi, M. R. (1990) Proc. Natl. Acad. Sci. U.S.A. 87, 3513–3517
8. Wickner, S., Gottessman, S., Skowrya, D., Hoskins, J., Mcrkenney, K., and Maurizi, M. R. (1994) Proc. Natl. Acad. Sci. U.S.A. 91, 12218–12222
9. Subjeck, J. R., Shyy, T., Shen, J., and Johnson, R. J. (1983) J. Cell Biol. 97, 1389–1395
10. Shyy, T.-T., Subjeck, J. R., Heinaman, R., and Anderson, G. (1986) Cancer Res. 46, 4738–4745
11. Hatayama, T., Nishiyama, E., and Yasuda, K. (1994) Biochem. Biophys. Res. Commun. 200, 1367–1373
12. Hatayama, T., Yasuda, K., and Nishiyama, E. (1994) Biochem. Biophys. Res. Commun. 204, 357–365
13. Hatayama, T., Honda, K., and Yukioka, M. (1986) Biochem. Biophys. Res. Commun. 137, 957–963
14. Honda, K., Hatayama, T., and Yukioka, M. (1989) Biochem. Biophys. Res. Commun. 160, 60–66
15. Nakano, N. (1966) Tohoku J. Exp. Med. 88, 69–84
16. Chirgwin, J. M., Przybyla, A. E., MacDonald, R. J., and Rutter, W. J. (1979) Biochemistry 18, 5294–5299
17. Nakai, A., and Morimoto, R. I. (1993) Molec. Cell. Biol. 13, 1983–1997
18. Chomczynski, P., and Sacchi, N. (1987) Anal. Biochem. 162, 156–159
19. Hunt, C., and Morimoto, R. I. (1985) Proc. Natl. Acad. Sci. U.S.A. 82, 6455–6450
20. Gunning, P., Ponte, P., Okayama, H., Engel, J., Blau, H., and Kedes, L. (1983) Mol. Cell. Biol. 3, 787–795
21. Flaherty, K. M., McKay, D. B., Kabach, W., and Hidmes, K. C. (1993) Proc. Natl. Acad. Sci. U.S.A. 90, 5041–5045
22. Welch, W. J., and Feramisco, J. R. (1985) Molec. Cell. Biol. 5, 1229–1237
23. Fathallah, D. M., Cherif, D., Delafi, K., and Arnault, M. A. (1993) J. Immunol. 151, 810–813
24. Harrison, G. S., Drakkin, H. A., Kao, F.-T., Hartz, J., Hart, I. M., Chu, H. Y., Wu, B. J., and Morimoto, R. I. (1987) Somatic Cell Mol. Genet. 13, 119–130
25. Foltz, K. R., Partin, J. S., and Lennart, W. J. (1993) Science 259, 1421–1425
26. Lowe, D. G., and Moran, L. A. (1986) J. Biol. Chem. 261, 2102–2112
27. Bork, P., Sander, C., and Valencia, A. (1992) Proc. Natl. Acad. Sci. U.S.A. 89, 7290–7294
28. Chappell, T. G., Konforti, B. B., Schmid, S. L., and Rothman, J. E. (1987) J. Biol. Chem. 262, 746–753
29. Rippmann, F., Taylor, W. R., Rothbard, J. B., and Green, N. M. (1991) EMBO J. 10, 1053–1059
30. Hatayama, T., Tsujio, K., Wakatsuki, T., Kitamura, T., and Imahara, H. (1992) J. Biochem. (Tokyo) 113, 484–490
31. Honda, K., Hatayama, T., and Yukioka, M. (1988) J. Biochem. (Tokyo) 103, 81–89
32. Takuchi, K., Hosokawa, N., Hirayoshi, K., and Nagata, K. (1994) Mol. Cell. Biol. 14, 567–575
33. Matsumoto, M., and Fujimoto, H. (1990) Biochem. Biophys. Res. Commun. 166, 43–49
34. Parsell, D. A., Kowal, A. S., Singer, M. A., and Lindquist, S. (1994) Nature 372, 475–478
35. Lee-Yoon, D., Easton, D., Murawski, M., Burd, R., and Subjeck, J. R. (1995) J. Biol. Chem. 270, 15725–15733

3 Y. Kaneko and J. Fujita, personal communication.
Cloning and Expression of Murine High Molecular Mass Heat Shock Proteins, HSP105
Kunihiko Yasuda, Akira Nakai, Takumi Hatayama and Kazuhiro Nagata

J. Biol. Chem. 1995, 270:29718-29723.
doi: 10.1074/jbc.270.50.29718

Access the most updated version of this article at http://www.jbc.org/content/270/50/29718

Alerts:
- When this article is cited
- When a correction for this article is posted

Click here to choose from all of JBC's e-mail alerts

This article cites 35 references, 17 of which can be accessed free at http://www.jbc.org/content/270/50/29718.full.html#ref-list-1