The heat shock factor (HSF) is a pivotal transcriptional factor that regulates the expression of genes encoding heat shock proteins (HSPs) via heat shock elements (HSEs). nGAAnnTTCnnGAAn functions as the minimum consensus HSE (cHSE) in vivo. Here we show that the expression of Saccharomyces cerevisiae MDJ1 encoding a mitochondrial DnaJ homolog is regulated by HSF via a novel non-consensus HSE (ncHSEMDJ1), which consists of three separated pentameric nGAAn motifs, nTTCn-(11 bp)-nGAAn-(5 bp)-nGAAn. This is the first evidence to show that the immediate contact of nGAAn motifs is dispensable for regulation by HSF in vivo. ncHSEMDJ1 confers different heat shock responses versus cHSE and, unlike cHSE, definitively requires a carboxyl-terminal activation domain of HSF in the expression. ncHSEMDJ1-like elements are found in promoter regions of some other DnaJ-related genes. The highly conserved HSF/HSE system suggests that similar ncHSEs may be used for the expression of HSP genes in other eukaryotes including humans.

All organisms possess a highly conserved system that responds to elevated temperatures by transcriptionally inducing genes encoding heat shock proteins (HSPs)\(^1\) to deal with heat stress. The induction requires heat shock transcription factor (HSF) and cis-heat shock elements (HSEs)\(^1\). HSF has two conserved domains, a helix-turn-helix DNA binding domain and a coiled-coil hydrophobic repeat domain needed for trimers or higher order multimer formation \((2, 3)\). HSFs of the budding yeasts \((Saccharomyces cerevisiae\) and \(Kluyveromyces lactis\) \(^2\)) uniquely possess two activation domains in the amino terminus (AAD) and carboxyl terminus (CAD) \((4, 5)\), whereas those of fission yeast \((Drosophila melanogaster\)) and vertebrates have CADs alone. HSF is moderately phosphorylated under non-stress conditions and is further activated by hyperphosphorylation upon heat shock to induce the expression of HSPs \((6–8)\). In fission yeast, \(D. melanogaster\), and mammals, the binding activity of HSFs to HSEs is dramatically stimulated by heat shock as HSF monomers are converted to trimers \((9–11)\). In contrast, in the budding yeast, HSF constitutively binds to HSE, but the binding activity seems to increase after heat shock-induced hyperphosphorylation \((7, 12–16)\).

The HSE is composed of several contiguous inverted repeats of the 5-base pair sequence nGAAn (where n is any nucleotide) \((1, 17, 18)\). The number of the pentameric units in HSE varies, but at least three units are thought to be the minimum required for heat regulation in vivo. Namely, nGAAnnTTCnnGAAn is the minimum consensus HSE (chSE) \((19, 20)\). However, an HSE can tolerate and still function with a 5-bp insertion between two repeating units if the spacing and orientation of the pentameric elements are maintained, e.g. nGAAn-(5-bp)-nGAAn \((21)\). The most characterized endogenous non-consensus HSE (ncHSE) is that of \(CUP1\) in \(S. cerevisiae\), nTTCnnGAAn-(5-bp)-nGAAn denoted ncHSE\(_{CUP1}\) \((22–24)\). Similar elements regulate \(HSP82\) and \(HSC82\) \((12, 25–27)\). It is unclear whether the immediate contact of at least two pentameric motifs is needed for heat induction in vivo.

In higher organisms possessing multiple HSF genes, HSF isoforms appear to perform different biological functions and regulate distinct target genes via differential binding preferences for specific HSE architectures \((6, 28–36)\). By contrast, yeasts and \(D. melanogaster\) have a single essential HSF gene \((7, 37–41)\). Interestingly, the induction maximum of \(CUP1\) and chSE-driven \(SSA1\) is observed at different high temperatures \((22–24, 42)\). In addition, it is known that the binding preference of HSF for HSE variants changes under different physiological conditions \((23, 24, 43, 44)\). Thus, it is probable that the HSF-regulated gene expression is modulated by different HSE architectures in these organisms.

In \(S. cerevisiae\), there are various nuclear-encoded genes for mitochondrial HSPs (mtHSPs) as follows: DnaK homolog Ssc1, DnaJ homolog Mdj1, and chaperonin complex Hsp60/Hsp10. They are involved in protein import, protein folding and assembly, and proteolysis in mitochondria \((45–48)\). Although some of the mtHSP genes (HSP60 and HSP10) but not all (SSC1 and \(MDJ1\)) have obvious HSEs in their promoters, all of them are heat-inducible \((49–52)\). Here we show that \(MDJ1\) is regulated by HSF via a novel ncHSE.

**EXPERIMENTAL PROCEDURES**

**Culture Media, Strains, and Plasmids**—The media used include glucose-based rich medium (YPD) and synthetic medium (SD). YPGalR and SGAir are identical to YPD and SD with the exception that they contain 1% galactose and 1% raffinose instead of 2% glucose. The strains used are listed in Table I. The promoter regions of HSP genes were amplified by PCR using an Expand High Fidelity PCR system (Roche Diagnostics, Mannheim, Germany), \(MDJ1\)\((-403 to +3)\) (translation start site is +1), \(MDJ1\)\((-273 to +3)\), \(MDJ1\)\((-239 to +3)\), \(MDJ1\)\((-204 to +3)\), \(MDJ1\)\((-164 to +3)\), and \(MDJ1\)\((-126 to +3)\) (translation start site is +1).
TABLE I

| Strains (arias) | Genotype | Reference |
|----------------|----------|-----------|
| KMY1005        | Mata leu2 ura3 his3 trp1 lys2 | This study |
| shk7A (SCU298) | KMY1005 with shk7:His3 |         |
| ΔH5F1/92       | ade2 can1 his3 leu2 trp1 his3letion [LEU2/HSF1 TRP1 CEN] | 43        |
| hsf1Δ2900::ΔH5F1/96 | ΔH5F1/92 except [hsf1Δ2906 TRP1 CEN] | 43        |
| hsf1Δ2900::ΔH5F1/97 | ΔH5F1/92 except [hsf1Δ2903 TRP1 CEN] | 43        |
| ΔH5F1/92       | hsf1Δ2900::ΔH5F1/97 except [GAL1p-2HA-HSF1 URA3 CEN] | 80        |
| MATAΔhse65ΔΔΔΔΔΔ | Mata leu2 ura3 trp1 his3 ade2 can1 GAL SUC mao1 | 80        |
| MATAΔhse65ΔΔΔΔΔΔΔΔΔΔΔ | MATAΔhse65ΔΔΔΔΔΔΔΔΔΔΔ except [HIS3 msn4:TRP1 CEN] | 30        |
| Δmsn2−/4       | MATAΔhse65ΔΔΔΔΔΔΔΔΔΔΔ except [HIS3 msn4:TRP1 CEN] | 30        |
| KT1099         | Mata leu2 ura3 trp1 | 61        |
| gcl7−/− (KT1098) | gcl7−/−         | 61        |
| PS128          | ura3Δ–52 leu3 trp1 his3 ade2Δ–2 [hsf1Δ2::LEU2/HSF1 URA3 CEN] | 5        |
| HSFI (SCU335)  | PS128 except [HSF1 HIS3 CEN] | This study |
| hsf1ΔCAD (SCU336) | PS128 except [hsf1ΔCAD TRP1 CEN] | This study |

RESULTS

Promoters of MDJ1 and SSC1 Confer Heat Inducibility in a HSF-dependent Manner—This has been shown by Northern blot analysis that mRNA levels of MDJ1 is increased by heat shock (52) despite no obvious HSE. To see transcriptional heat inducibility of the MDJ1 promoter, the promoter activity was assayed using a construct MDJ1 promoter fused to lacZ (MDJ1-lacZ). This construct showed heat induction similar to SSC3-lacZ as the positive control, although it already had a higher basal expression at 23 and 30 °C than SSA3-lacZ (Fig. 1A). Ssc1 protein is also heat-inducible despite no obvious HSE (50). SSC1 was shown to be heat-inducible by SCS1-lacZ although to a lesser extent than that of MDJ1 promoter. The fold induction of MDJ1 and SSC1 was smaller compared with SSA3, which is well known to be remarkably heat-inducible. The apparent decreased induction is probably because of the relatively high basal expression levels of MDJ1 and SSC1, which presumably reflect their essentiality for mitochondrial functions even under non-stress conditions (52, 58).

Electrophoretic Mobility Shift Assay—Protein extracts were pre pared from 100 ml of logarithmic phase cell cultures of strain SCU166 in the YPD medium. The cells harvested by centrifugation were washed once with distilled water and suspended in an equal volume of 10 mM Tris-HCl, pH 8.0, 10 mM MgCl₂, 10% (w/v) sorbitol in Msn2/4-dependent manners (59). However, heat shock, an aliquot of the cells cultivated at 37 °C were transferred to 39 °C. β-Galactosidase activity in the extract of chloroform/SDS-permeabilized cells was assayed and expressed in Miller units (55). Each value is expressed as the mean ± S.D. of duplicate determinations of three independent yeast transfections.

Identification of the Element Responsible for Heat Inducibility in the MDJ1 Promoter—The MDJ1 promoter region from −403 bp (translation start site is +1) used in Fig. 1 contains two stress response elements (STREs) (Fig. 2). STRE also confers heat inducibility to genes by redundant transcriptional factors Msn2 and Msn4. MDJ1 is induced by ethanol, NaCl, and sorbitol in Man2/4-dependent manners (59). However, heat induction still occurred even when the two STREs were deleted (Fig. 2, Δ1). Furthermore, a msn2Δ msn4Δ double mutant showed similar heat inducibility of MDJ1-lacZ (data not shown). Thus, these STREs were not needed for heat inducibility, but if involved, their contribution was marginal.

To dissect the function of the MDJ1 promoter, a series of progressive 5' deletion clones was made (Fig. 2). Both basal and heat-induced levels gradually decreased when the sequence upstream of −239 was deleted (clones Δ1–5). In contrast, further removal of the region from −239 to −203 increased both levels (clones Δ5–7). Further elimination of the region from factors in the promoter regions of genes, the sequences were searched using MatInspector V2.2 (transfac.ghb-braunschweig.de/cgi-bin/matSearch/matSearch.pl) (56). To find nchHSEΔ2903-like sequences in the yeast gene promoters, the yeast genomic DNA was searched using PatScan pattern matcher (www-unix.mcs.anl.gov/combio/PatScan/HTML/patscan.html) (57) with the query pattern TTCG...····GGAA...····ATG. 22141
The involvement of HSF in hsf1R206S or A putative third for1h (black bars) is referred to as a putative HSE. This assumption was tested by inserting an oligonucleotide corresponding to −174 to −144 of the MDJ1 promoter into a CYC1-lacZ reporter construct. As hypothesized, the basal expression level of CYC1-lacZ driven by this element was significantly (reproducibly but not remarkably) enhanced by hsf1R206S (Fig. 3A). Therefore, it was named ncHSEMDJ1. On the other hand, a loss of a two-component response regulator homolog Skn7, which has a DNA-binding domain similar to that of Hsf1 and induces some HSP genes in response to oxidative stress cooperatively with Hsf1 (60), showed no obvious effect on expression of ncHSEMDJ1-CYC1-lacZ (data not shown), indicating that its expression is Skn7-independent.

ncHSEMDJ1 also conferred heat inducibility to CYC1-lacZ (Fig. 3B). Thus, this element is necessary and sufficient for heat induction. The conversions of GAA of units B and C to non-functional mutations GAG are completely abolished heat inducibility (M2 and M3). In contrast, the conversion of TCC of unit A to CTC did not affect heat induction (M1). The lack of affect of this mutation is most probably attributed to the generation of a new TTC site 2 bp upstream TTTTC to TTTC, because an additional mutation introduced in the region TTTTC to CTTC completely abrogated heat inducibility (M4). These results demonstrate that all three pentamers are essential for heat inducibility and show that ncHSEMDJ1 functions as an HSE. Conversely, when the GAA-gap-2-GAA was altered to GAACTTCTTGGAAGA, the heat inducibility was reinforced (M5), indicating that this gap weakens heat inducibility. However, this gap length is very important for heat inducibility, because even if only one bp of this region was added or removed, the heat inducibility was completely abrogated (M6 and M7). By contrast, M1 shows that the gap1 length is flexible in terms of heat inducibility.

This novel ncHSE architecture ncHSEM1 is quite distinct even from ncHSECUP1 (Table II). Three pentameric motifs of ncHSEM1 are separated from each other (nTTCn(gap)-nGAAn(gap)-nGAAn) in a different manner than those of the HSEs previously described, cHSE (GAAnTTCTTGGAAGA), the heat inducibility was reinforced (M5), indicating that this gap weakens heat inducibility. However, this gap length is very important for heat inducibility, because even if only one bp of this region was added or removed, the heat inducibility was completely abrogated (M6 and M7). By contrast, M1 shows that the gap1 length is flexible in terms of heat inducibility.

Fig. 2. Deletion analysis of the MDJ1 promoter. lacZ was expressed under the control of the sequentially deleted constructs of the MDJ1 upstream region. Wild-type strains (KMY1005) harboring these constructs were transferred from 23 to 37 °C for 1 h. Translation start site is +1.

A Novel Non-canonical HSE of MDJ1, ncHSEM1—The involvement of HSF in MDJ1 expression suggests that −174TCC−171 abolish basal level (clone Δ10). A further deletion of the region containing −156GAA−154 entirely abrogated heat induction (clone Δ11). These results indicate that these regions are necessary for heat inducibility.

A novel binding of HSF to ncHSEM1 in vitro—To assess the binding ability of HSF to ncHSEM1, Electrophoretic mobility shift assay was carried out with ncHSEM1 as a probe and with protein extracts from yeast cells expressing 2HA-tagged Hsf1 (2HA-Hsf1). Similar to cHSE, a specific DNA-protein complex was found using ncHSEM1 (Fig. 4, lanes 2 and 8). These complexes were converted to slower migrating forms (super-shift) when anti-HA antibody was co-incubated prior to electrophoresis (data not shown), indicating that these complexes contain HSF. However, ncHSEM1 was a less effective competitor than cHSE to cHSE (lanes 3–6). cHSE to ncH-
ncHSE MDJ1 can bind to HSF in vitro although with weaker affinity than cHSE.

ncHSE MDJ1 and cHSE Confer Different Heat Responses—The different architecture between ncHSE MDJ1 and cHSE suggests that they may confer different heat responses to gene expression.

This appears to be the case. Whereas they showed maximum induction at 39 °C, even at 41 °C, ncHSE MDJ1 still maintained heat inducibility (40% heat induction at 39 °C) compared with cHSE (22%) (Fig. 5A). Additionally, heat response conferred by cHSE was transient, whereas the heat response mediated by ncHSE MDJ1 was rather sustained (Fig. 5B).

Hsf1 CAD Is Essential for ncHSE MDJ1-driven Gene Expression—AAD, but not CAD, of Hsf1 is essential for viability and for basal expression of HSP genes (5) (see also Fig. 6A). However, CAD is critical for heat induction of CUP1, HSP82, and HSC82 but not of cHSE-driven HSP genes (22–24, 42), suggesting that CAD is specifically required for ncHSEs with gaps. This is also true for ncHSE MDJ1. The heat induction of cHSE-driven reporter still occurred in the strain lacking CAD of Hsf1 (hsf1ΔCAD), albeit to reduced levels (Fig. 6B). On the contrary, both basal and heat-induced expression of ncHSE MDJ1-driven reporter was drastically reduced by the loss of CAD. These results demonstrate that CAD is essential for ncHSE MDJ1-mediated gene expression. This finding is consistent with the observations that CAD is required for sustained responses to heat shock (5) and that ncHSE MDJ1 conferred sustained heat response (Fig. 6B).

Glc7 Differentially Regulates cHSE-directed and ncHSE MDJ1-directed Gene Expression—HSF activity is regulated by its phosphorylation status. Loss-of-function mutations of a type 1 serine/threonine protein phosphatase complex Glc7-Gac1 (Glc7 and Gac1 are catalytic and regulatory subunits, respectively) such as glc7-1 (61) attenuate heat induc-

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**TABLE II**

Comparison between architecture of HSEs and HSE-like elements found in promoters of various HSP genes

| HSE(-like) element | Gene, position | Confers heat induction? |
|--------------------|---------------|-------------------------|
| gGAAttTTTcaGAAa | HSP60, −168 | Yes<sup>a</sup> |
| gGAAttTTTcaGAAa | HSF10, −164 | Yes<sup>a</sup> |
| cTTc-gaGAcacaagaGACa | CUP1, −211 | Yes |
| tTT-c-gaGAcgccgtqGAag | HSP62, −235 | Yes |
| tTT-c-tacatcctgtaGAA-cctatqGAAa | MDJ1, −174 | Yes (this study) |
| tTT-c-tacatcctgtaGAA-cctatqGAAa | YDJ1, −391 | ? |
| gTTccacaaqttatqGAA-atcqttqGAac | YNL099w, −212 | No (this study) |
| gGAAttttggtTTTTtqTTTccacgttqGAAa | SSC1, −177 | No (this study) |

<sup>a</sup>The position of A of the initial ATG is +1.

<sup>b</sup>T. Tachibana, S. Astuni, R. Shioda, M. Ueno, M. Uritani, and T. Ushimaru, unpublished data.

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**FIG. 4.** HSF binds to ncHSE MDJ1<sup>−</sup> in vitro. Protein extract from cells expressing HA2-HSF1 was incubated with labeled cHSE or ncHSE MDJ1. Lanes 1–6, labeled cHSE; lanes 7–12, labeled ncHSE MDJ1; lanes 3 and 9, competed with 100× excess of cHSE; lanes 4 and 10, competed with 100× excess of ncHSE MDJ1; lanes 5 and 11, competed with 10× excess of ncHSE MDJ1; lanes 6 and 12, competed with 100× excess of ncHSE MDJ1. For all lanes with the exception of lanes 1 and 7, protein extracts were added.

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**FIG. 5.** ncHSE MDJ1 confers heat response in a HSF-dependent manner. A, the oligonucleotide corresponding to sites from −174 to −144 of the MDJ1 promoter region (ncHSE MDJ1) was fused to a CYC1-lacZ reporter. Plasmid pncHSE MDJ1-CYC1-lacZ was transformed in strains harboring HSF1Δ(HSF1)92 and hsf1ΔCADΔ(HSF1)96. B, substituted (M1–M5), deleted (M6), and inserted (M7) mutations were tested. Wild-type strain (KMY1005) with each construct was heat-treated. *: substituted site; -: deleted site. The underlined nucleotides represent the conserved GAA motifs.

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**FIG. 3.** ncHSE MDJ1 confers heat response in a HSF-dependent manner. A, the oligonucleotide corresponding to sites from −174 to −144 of the MDJ1 promoter region (ncHSE MDJ1) was fused to a CYC1-lacZ reporter. Plasmid pncHSE MDJ1-CYC1-lacZ was transformed in strains harboring HSF1Δ(HSF1)92 and hsf1ΔCADΔ(HSF1)96. B, substituted (M1–M5), deleted (M6), and inserted (M7) mutations were tested. Wild-type strain (KMY1005) with each construct was heat-treated. *: substituted site; -: deleted site.
different HSE architectures. However, we cannot formally exclude the possibility that another transcriptional factor, which is also regulated by Glc7-Gac1, may bind to the long gap of ncHSEMDJ1 element and influence reporter expression.

cmpHSEMDJ1-like Elements Are Found in Promoter Regions of Other DnaJ Homolog Genes—Computer-assisted analysis revealed that there are ncHSEMDJ1-like elements in promoter regions of many genes. Most interestingly, ncHSEMDJ1-like elements were found in the promoter regions of two genes for DnaJ homologs, cytosolic Ydj1, and as-yet-uncharacterized Ynl077w (Table II). Northern analysis showed that Ydj1 is heat-induced (63). We also found that Ydj1-lacZ and Ynl077w-lacZ were also transcriptionally heat-inducible (data not shown). From similarities in the DNA architecture, it is most probable that these ncHSEMDJ1-like elements generally confer heat inducibility. In contrast, DnaJ-related Sis1 is heat-induced via a chSE (64). Other DnaJ-related genes, Scj1, Xdj1, Jem1, Sec63, Ynl227c, Hljl1, Caij1, Zouj1, Djp1, Yjl162c, Ypr041c, and Yjr097w possess no obvious HSE-like elements. Thus, some but not all DnaJ-related genes appear to be driven via ncHSEMDJ1-like elements. On the other hand, a region containing several nGAA motifs was found in the SSC1 promoter (Table II), but this architecture is different from those of cHSE or ncHSE. Similar heat induction was observed in SSC1(-178 to -3)-lacZ and SSC1-lacZ (data not shown), indicating that the element responsible for heat induction is contained within -145 to -1.

**DISCUSSION**

A single HSF trimer binds to the one copy of the HSE element (17, 65), whereas a pair of trimers interacts with the ncHSE cooperatively in the presence of more than three GAA motifs (66). Although there is no direct evidence, we assume that a single HSF trimer binds to ncHSEMDJ1, because the mobility of the HSF-ncHSEMDJ1 complex was nearly the same as that of HSF-chSE complex (Fig. 4). The 73 amino acids between the DNA-binding and the trimerization domain in Hsf1 are suggested to be a flexible linker (67). We believe that this flexible linker would be long enough to allow each trimerization domain to come close and form a trimeric coiled-coil structure. The hsflCAD strain almost completely lost heat induction of ncHSEMDJ1-directed but not chSE-directed reporter, indicating that CAD function depends on cis-element

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T. Tachibana, S. Astumi, R. Shioda, M. Uritani, and T. Ushimaru, unpublished data.
architecture. We propose the following model to explain these observations. The AADs of three HSF molecules can associate with each other in the HSF/chSE complex but not in the HSF/ncHSE complex. In contrast, three CADs probably are able to associate with each other in both complexes (Fig. 8). At least either clustered AADs or clustered CADs are required for HSF-mediated gene expression. Therefore, Hsfl/CAD can still transactivate gene expression via chSE but not ncHSE/MDJ1.

We found that the expression of mtHSP genes examined (MDJ1, SSC1, HSP10, and HSP60) were all regulated by HSF, this study and Ushimaru et al., unpublished data). Mitochondria are thought to have evolved from a free-living eubacterium endosymbiont. At present, however, all of these mtHSP genes have transferred from the mitochondrial genome to the nuclear genome during evolution. Subsequently, mtHSP genes have been put under the HSF/HSE system of the host cell. However, the adoption of this system is not self-evident, because S. cerevisiae possesses other distinct heat response systems, i.e. Ras-protein kinase A, calcineurin, and protein kinase C pathways. The calcineurin and PKC pathways appear to play restricted roles in cation homeostasis and reinforcement of membrane rigidity (68–73). In contrast, the HSF/HSE system performs general roles in the protection against heat stress. Most HSP genes are regulated by the HSF/HSE system, which is activated by the accumulation of unfolded proteins in the cytosol, e.g. after heat shock (74). Because upon heat shock, unfolded proteins are accumulated in all intracellular compartments including in mitochondria, this simple HSF/HSE heat-responsible system might be sufficient for the regulation of mtHSP genes. Some genes regulated by HSF are also controlled by Msn2/4 (75). These two systems are differentially used under various stress conditions (59, 75). In the case of MDJ1, the HSF/HSE and the Msn2/4/STRE systems are responsible for induction by heat (this study), and by ethanol, NaCl, and sorbitol (59), respectively.

Mdj1 is a partner of Ssc1 in the mitochondrial matrix. Jac1 is another J-type chaperone working together with another HSP70 protein Ssq1 in the mitochondrial matrix (76–79). However, neither Ssq1 nor Jac1 has any obvious HSEs. On the other hand, many of the ncHSE/MDJ1-like elements are found in the promoters of other genes, indicating that they have general roles. From the conservation of the HSF/HSE system among euakaryotes, this study suggests that ncHSE/MDJ1-like elements may be involved in the expression of HSP genes in other organisms including humans. For example, one candidate is human HSP90 (GenBank™ accession number J04988), which has two ncHSE/MDJ1-like elements in its promoter region, 1062(TC/24 bp)-GAATGTTGTCGAAA-1033-646GAAGCTGGAAGACA-32 bp)-TTC-919 (transcription start site is +1), although their gap1 length is rather longer.

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REFERENCES

1. Morimoto, R. I. (1993) Science 259, 1409–1410
2. Hatano, K., Ushimaru, H., Weinfeld, R. A., and Wakisaka, N. (1994) Science 263, 224–227
3. Rabindran, S. K., Haroun, R. I., Clos, L. Nauwleski, J., and Wu, C. (1993) Science 259, 230–234
4. Nieto-Sotelo, J., Wiederricht, G., Okuda, A., and Parker, C. S. (1990) Cell 62, 807–817
5. Sorger, P. K. (1990) Cell 62, 793–805
6. Liu, X. D., Liu, P. C., Santoro, N., and Thiele, D. J. (1997) EMBO J. 16, 6469–6477
7. Sorger, P. K., and Pelham, H. R. (1988) Cell 54, 855–864
8. Xia, W., and Voellmy, R. (1997) J. Biol. Chem. 272, 4094–4102
9. Kington, R. E., Schuetz, T. J., and Larin, Z. (1987) Mol. Cell. Biol. 7, 1530–1534
10. Wu, C. (1984) Nature 310, 229–234
11. Zimmaro, V., and Wu, C. (1987) Nature 327, 727–730
12. Santoro, N., Johansson, N. C., Collins, K. W., and Lee, S. W. (1990) Mol. Cell. Biol. 10, 611–613
13. Jakobsen, B. K., and Pelham, H. R. (1988) Mol. Cell. Biol. 8, 5408–5420
14. Mager, W. H., and Ferreira, P. M. (1993) Biochem. J. 290, 1–13
15. Sorger, P. K., Lewis, M. J., and Pelham, H. R. (1987) Nature 329, 81–84
16. Becker, J. J., Ballou, C., and Fackenthal, D. L. (1994) Mol. Cell. Biol. 14, 501–508
17. Xiao, H., Pericis, O., and Liu, J. T. (1991) Cell 64, 585–593
18. Fernandez, M. X., Xiao, H., and Liu, J. T. (1994) Nucleic Acids Res. 22, 167–173
19. Slater, M. R., and Craig, E. A. (1987) Mol. Cell. Biol. 7, 1906–1916
20. Amin, J., Ananthan, J., and Voellmy, R. (1988) Mol. Cell. Biol. 8, 3761–3769
21. Liu, X. D., and Thiele, D. J. (1996) Genes Dev. 10, 592–603
22. Santoro, N., Johansson, N. C., and Thiele, D. J. (1998) Mol. Cell. Biol. 18, 6340–6352
23. Tamai, K. T., Liu, X., Silar, P., Sosinskiou, T., and Thiele, D. J. (1994) Mol. Cell. Biol. 14, 8155–8165
24. Baler, R., Dahl, G., and Voellmy, R. (1993) Mol. Biol. Cell 4, 1392–1407
25. Sistonen, L., Sarge, K. D., Phillips, B., Abravaya, K., and Morimoto, R. I. (1993) Mol. Cell. Biol. 13, 3291–3297
26. Sarge, K. D., Murphy, S. P., and Morimoto, R. I. (1993) Mol. Cell. Biol. 13, 2392–2397
27. Nakai, A., Tanabe, M., Kawase, Y., Inazawa, J., Morimoto, R. I., and Nagata, K. (1997) J. Biol. Chem. 272, 15389–15395
28. Nakai, A., Kagawa, Y., and Nagata, K. (1997) J. Biol. Chem. 272, 15389–15395
29. Clot, J., Westwood, J. T., Becker, P. B., Wilson, S., Lambert, K., and Wu, C. (1993) Mol. Cell. Biol. 13, 749–761
30. Jakobsen, B. K., and Pelham, H. R. (1993) EMBO J. 12, 369–375
31. Li, L., Misono, M., and Wu, C. (1997) EMBO J. 16, 2452–2462
32. Wiederricht, G., Seto, D., and Parker, C. S. (1988) Cell 54, 841–853
33. Young, M. R., and Craig, E. A. (1993) Mol. Cell. Biol. 13, 5657–5664
34. Sewell, A. K., Yokoya, F., Wu, Y., Miyagawa, T., Murayama, T., and Wing, W.
44. Silar, P., Butler, G., and Thiele, D. J. (1991) Mol. Cell. Biol. 11, 1232–1238
45. Craig, E. A., Gamblin, B. D., and Nelson, R. J. (1993) Microbiol. Rev. 57, 492–414
46. Langer, T., and Neupert, W. (1995) in The Biology of Heat Shock Proteins and Molecular Chaperones (Morimoto, R., Tessieres, A., and Georgopoulos, C., eds) pp. 53–83, Cold Spring Harbor Laboratory, Cold Spring Harbor, NY
47. Westermann, B., Prip-Buus, C., Neupert, W., and Schwarz, E. (1995) EMBO J. 14, 3452–3460
48. Westermann, B., Gaume, B., Herrmann, J. M., Neupert, W., and Schwarz, E. (1996) Mol. Cell. Biol. 16, 7063–7071
49. Hahfeld, J., and Hartl, F. U. (1994) J. Cell Biol. 126, 305–315
50. Kawakami, K., Shafer, B. K., Garfinkel, D. J., Strathern, J. N., and Nakamura, Y. (1992) Genetics 131, 821–832
51. Laloraya, S., Gamblin, B. D., and Craig, E. A. (1994) Proc. Natl. Acad. Sci. U. S. A. 91, 6481–6485
52. Bowlley, N., Prip-Buus, C., Westermann, B., Brown, C., Schwarz, E., Barrell, B., and Neupert, W. (1994) Cell 77, 249–259
53. Mori, K., Kawahara, T., Yoshida, H., Yanag, H., and Yura, T. (1996) Genes Cells 1, 803–817
54. Brachmann, C. B., Davies, A., Cost, G. J., Caputo, E., Li, J., Hieter, P., and Boeke, J. D. (1998) Yeast 14, 115–132
55. Mori, K., Ma, W., Gething, M. J., and Sambrook, J. (1993) Cell 74, 743–756
56. Quandt, K., Prech, K., Karas, H., Wingender, E., and Werner, T. (1995) Nucleic Acids Res. 23, 4878–4884
57. Desouza, M., Larren, N., and Overbeek, R. (1997) Trends Genet. 13, 487–498
58. Mochida, K., Nakagawa, K., Yamanoto, E., and Shibata, T. (1990) J. Biol. Chem. 265, 15189–15197
59. Moskvina, E., Schuller, C., Maurer, C. T., Maggs, W. H., and Ruis, H. (1998) Yeast 14, 1041–1050
60. Stas, D. C., Johnson, C. A., Erkine, A. M., Makino, K., Morgan, B., Gross, D. S., and Johnston, L. H. (2000) Mol. Cell. Biol. 11, 2335–2347
61. Stuart, J. S., Frederick, D. L., Varner, C. M., and Tatchell, K. (1994) Mol. Cell. Biol. 14, 896–905
62. Lin, J. T., and Lis, J. T. (1999) Mol. Cell. Biol. 19, 3237–3245
63. Aten, D. P., and Yaffe, M. P. (1992) Mol. Cell. Biol. 12, 293–291
64. Zhong, T., Luke, M. M., and Arndt, K. T. (1996) J. Biol. Chem. 271, 1349–1356
65. Perisic, O., Xiao, H., and Lis, J. T. (1999) Cell 99, 797–806
66. Erkine, A. M., Magrovan, S. F., Sekinger, E. A., and Gross, D. S. (1999) Mol. Cell. Biol. 19, 1627–1639
67. Flick, K. E., Gonzalez, L., Jr., Harrison, C. J., and Nelson, H. C. (1994) J. Biol. Chem. 269, 12475–12481
68. Kudo, Y., Jung, U. S., Potrowski, J., and Levin, D. E. (1995) Genes Dev. 9, 1559–1571
69. Mathews, D. P., Kingsbury, T. J., Ahsan, U. S., and Cunningham, K. W. (1997) Genes Dev. 11, 3445–3458
70. Stathopoulos, A. M., and Cyert, M. S. (1997) Genes Dev. 11, 3432–3444
71. Watanabe, Y., Irie, K., and Matsumoto, K. (1995) Mol. Cell. Biol. 15, 5740–5749
72. Westermann, B., Irie, K., Printen, J. A., Stevenson, B. J., Sprague, G. F., Jr., Matsumoto, K., and Errede, B. (1995) Mol. Cell. Biol. 15, 6545–6553
73. Zhao, C., Jung, U. S., Garrett-Engele, P., Roe, T., Cyert, M. S., and Levin, D. E. (1998) Mol. Cell. Biol. 18, 1033–1022
74. Craig, E. A., and Gross, C. A. (1991) Trends Biochem. Sci 16, 135–140
75. Treger, J. M., Schmitt, A. P., Simon, J. R., and McIntee, K. (1998) J. Biol. Chem. 273, 26875–26879
76. Strain, J., Lorenz, C. R., Bode, J., Garland, S., Smolen, G. A., Ts, D. T., Vickery, L. E., and Culotta, V. C. (1998) J. Biol. Chem. 273, 31138–31144
77. Voisine, C., Cheng, Y. C., Ohlson, M., Schilke, B., Hoff, K., Beintnt, H., Marszalek, J., and Craig, E. A. (2001) Proc. Natl. Acad. Sci. U. S. A. 98, 1483–1488
78. Liu, T., Westermann, B., Neupert, W., and Herrmann, J. M. (2001) J. Mol. Biol. 307, 815–825
79. Kim, K., Saxe, S., Gordon, D. M., Pain, D., and Dancis, A. (2001) J. Biol. Chem. 276, 17524–17532
80. Siderius, M., Ruts, E., and Mager, W. H. (1997) Microbiology 143, 3241–3250
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