We found a novel protein in the postmitochondria supernatant fraction of rat liver, which is soluble in 5% perchloric acid and strongly inhibits protein synthesis in a rabbit reticulocyte lysate system. The protein extracted from the supernatant fraction with 5% perchloric acid was purified by ammonium sulfate fractionation and CM-Sephadex chromatography. The protein was shown to consist of two identical subunits with a molecular mass of 14 kDa. By immunoscreening with the rabbit antiserum against the protein, a cDNA encoding the protein was cloned and sequenced. The cDNA contained an open reading frame of 411 base pairs encoding a 136-amino acid protein with a molecular mass of 14,149 Da. The deduced amino acid sequence was completely identical with that constructed from all of the above peptides. Interestingly, the perchloric-acid-soluble protein inhibited cell-free protein synthesis in the rabbit reticulocyte lysate system in a different manner from RNase A. The protein is likely to inhibit an initiation stage of cell-free protein synthesis. Among the rat tissues tested, the protein was located only in liver and kidney. These findings are the first report on a new inhibitor that may be involved in the regulation of protein synthesis in those tissues.

High mobility group (HMG) proteins are a family of nonhistone components in chromatin (1). There are four major HMG proteins (HMG1, -2, -14, and -17) in all the eukaryotic cells examined to date (2). Although the function of HMG proteins has not been identified unequivocally, HMG proteins have been implicated in transcription and in DNA replication (2). We showed that the HMG proteins play an important role in nutritional modulations of chick liver RNA synthesis (3) and isolated the HMG 2a cDNA from a zgt11 expression library of chick liver using polyclonal antibodies, which encodes a protein of 201 amino acids (4). In Northern blotting, 2.0- and 1.2-kb mRNAs for the protein were detected in the liver of newly hatched chick, but they were shown to decrease during post-hatched development (5).

In the course of the investigation on chicken and rat chromatin, we found in the livers of the animals a novel protein that was co-extracted with the H1 and HMG proteins by 5% perchloric acid. The perchloric-acid-soluble protein (PSP) showed inhibition of protein synthesis in a rabbit reticulocyte lysate system. Recently, Levy-Favatier et al. (6) reported the isolation of a 10-kDa perchloric-acid-soluble protein from rat liver and the cloning of a cDNA encoding the protein. They suggest that the half of the deduced polypeptide sequence presents 27% similarity with a region of the 83-90-kDa heat shock protein (hsp). However, they have never elucidated the function of the 10-kDa protein. It is of great interest to clarify whether our protein is identical with that reported by Levy-Favatier et al. (6) and to elucidate the physiological function of the PSP.

In this study, we describe the purification and characterization of the PSP from rat liver and the cloning and sequencing of a cDNA encoding the protein. Its function and distribution in rat tissues were also examined. Furthermore, a comparison of our protein with that detected by Levy-Favatier et al. (6) is described.

**EXPERIMENTAL PROCEDURES**

**Extraction of PSP**—Livers from male Wistar rats were immediately homogenized in two volumes of cold 0.25 m sucrose in buffer (50 mm Tris-HCl (pH 7.5), 25 mm KCl, and 10 mm MgCl2) with a Potter-Elvehjem type homogenizer. The homogenate was centrifuged at 10,000 g for 30 min. The supernatant was made 5% with respect to perchloric acid (PCA) by the addition of 60% PCA and centrifuged at 10,000 g for 15 min. The supernatant was then made 25% with respect to trichloroacetic acid, and the precipitate was collected by centrifugation. The precipitate was washed with acetone and dried under vacuum. The dried material was suspended in 0.9% acetic acid, and dialyzed extensively against 0.1 m sodium phosphate buffer (pH 7.5).

**CM-Sephadex C-25 Chromatography**—After clarification by a 10-min centrifugation at 10,000 × g, the proteins in the dialysate were fractionated with ammonium sulfate. The precipitate formed between 0 and 20% saturation was collected by centrifugation. The precipitate was suspended in 0.1 m sodium phosphate buffer (pH 7.5) and dialyzed against the same buffer. The dialysate was applied to a column (2.5 × 30 cm) of CM-Sephadex C-25. The flow-through fractions were collected. The protein in the flow-through fractions was used as the purified protein in this work.

**Preparation of Antiserum Against PSP**—New Zealand White rabbits were subcutaneously immunized with a emulsion of the PSP (100 μg) and complete Freund’s adjuvant. Two weeks later, the rabbits were injected with an emulsion of the PSP and incomplete Freund’s adjuvant as a booster. The animals were bled weekly after the booster injection.

**Immunoblotting**—The proteins in homogenate samples from various tissues were analyzed on polyacrylamide gels (15%) according to the method of Laemmli (7). After electrophoresis, the proteins on the gels were blotted onto nitrocellulose membranes (Schleicher & Schuell, Germany) and immunoblotted with the antiserum against the PSP as described previously (5).

**Isolation of the N- and C-terminal Peptides of PSP**—The N- and C-terminal peptides of PSP were isolated using an anhydrotrypsinagarose column (Takara Shuzo, Kyoto, Japan). The column strongly...
adsorbs peptides, the C-terminal amino acid residue of which is an Arg or Lys residue. The protein solution (300 μg), which had been dialyzed against 50 mM Tris-HCl buffer (pH 8.0) including 0.01% SDS, was digested at 37 °C overnight with TPCK-treated trypsin at a 1:100 ratio of trypsin to the protein. The digest was applied on the anhydrotrypsinagarose column (0.7 × 2.5 cm) equilibrated with 50 mM sodium citrate (pH 5.0). The column was washed with the same buffer. The peptides adsorbed on the column were eluted with 0.1 M formic acid. For the isolation of the N-terminal peptide, the peptides eluted from the above agarose column were further digested at 37 °C overnight in 0.1 M sodium phosphate buffer (pH 7.0) with aminopeptidase M at a 1:100 ratio of aminopeptidase M to the protein. The protein solution (300 μg) was digested at 37 °C overnight with lysyl endopeptidase (the protein/lysyl endopeptidase, 1:100:1 (w/w)) in 50 mM Tris-HCl buffer (pH 8.0) by reversed-phase high performance liquid chromatography (HPLC). HPLC was performed on a C4 column by elution with a linear gradient of acetonitrile (0–60%) in 0.05% trifluoroacetic acid (TFA) for 120 min at a flow rate of 0.5 ml/min. The peptide of interest was eluted at a retention time of 16.78 min, collected, and subjected to electrospray ionization (ESI) mass spectrometry and amino acid analysis. An aliquot of the peptide collected was further hydrolyzed at 37 °C overnight in 1.2 M ammonium bicarbonate (pH 7.7), including 1 mM mercaptoethanol and 1 mM EDTA with the acyl amino acid-releasing enzyme (Boehringer Mannheim) (5 μg). The product obtained with the acyl amino acid-releasing enzyme was purified by the above reversed-phase HPLC. The purified peptide was eluted at a retention time of 6.90 min, collected, and subjected to ES mass spectrometry and automated Edman degradation. For the isolation of the C-terminal peptide, the flow-through fractions obtained from the above-mentioned agarose column were dried with a centrifugal evaporator. The peptide(s) in the residue were fractionated by the above reversed-phase HPLC. The peptide of interest was eluted at a retention time of 64.30 min, collected, and subjected to ES mass spectrometry and automated Edman degradation. ESI mass spectra were obtained using a Finnigan TSQ-700 quadrupole mass spectrometer with a UltiTraq-32 operation system (electrospray voltage, 4.5 kV; ion multivoltage, 1.0 kV; flow rate of analyte solution, 5.0 μl/min; concentration of analyte solution, 1.0 μg/ml). After acid hydrolysis, the amino acid compositions of the peptide were analyzed by a Tosoh amino acid analysis system equipped with a FS-8010 fluorescence detector and a MCI gel CK-10U column. The peptides were used for their sequencing as described above.

**Proteolysis of the PSP and Sequencing Analysis of the Peptide—**The purified PSP was proteolyzed with lysyl endopeptidase for the analysis of its amino acid sequence. The PSP (100 μg) was incubated at 37 °C overnight with lysyl endopeptidase (the protein/lysyl endopeptidase, 100:1 (w/v)) in 50 mM Tris-HCl buffer (pH 8.0). The proteolytic products were separated by reversed-phase HPLC. The peptides were applied on a Vydac C18 column. After washing for the first 5 min with 0.1% trifluoroacetic acid, the peptides on the column were eluted with a linear gradient of 0–60% acetonitrile in 0.1% trifluoroacetic acid for the second 60 min at a flow rate of 1 ml/min. The peptides eluted were detected by following the absorbance at 206 nm and 280 nm. Finally, nine peptides (I–IX) were isolated and pooled. After drying in vacuo, the peptides were used for their sequencing as described above.

**Effect of the PSP on Cell-free Protein Synthesis—**Effects of the PSP and RNase A on cell-free protein synthesis were examined. The cell-free protein synthesis was carried out in the presence or absence of the PSP and RNase A with a rabbit reticulocyte lysate assay system (Amersham Corp.) using a tabacco mosaic virus mRNA (Boehringer Mannheim) as recommended by the manufacturer.

**Enzyme Assay and Staining for Ribonuclease Activity—**Ribonuclease activity was assayed according to the method reported by Reddi (8) with some modifications. The enzyme solution (100 μg) including the PSP or RNase A was added to 2 ml of RNA solution containing 50 mM Tris-HCl (pH 7.5), 5 mg of yeast RNA, and the reaction mixture was incubated at 37 °C for 10 min. The reaction was stopped by the addition of 1 ml of an ice-cold solution (2 mM La(NO3)3, 15% PCA). The acidified reaction mixture was centrifuged for 10 min. The supernatant (0.3 ml) was diluted 10 times with 2 ml of distilled water, and the absorbancy was measured at 260 nm. A 15% polyacrylamide gel containing 0.3 mg/ml of total RNA from rat liver was prepared, and staining for ribonuclease activity was done by the methods of Blank et al. (9).

**Immunohistochemical Staining—**Immunohistochemical staining of rat liver and kidney was performed after perfusion of the tissues with fixing solution as described previously (10). Animals were anesthetized by intraperitoneal injection of sodium pentobarbital, and 200 units of heparin in 0.2 ml of saline were injected into the inferior vena cava. Then, a cannula was inserted through the inferior vena cava to the right atrium, which was ligated in place. Tissues were perfused through the cannula with Ca2+- and Mg2+-free Dulbecco’s phosphate-buffered saline (PBS) (Life Technologies, Inc.) via the cannula until the perfusate was free of blood. Then, the tissues were fixed by perfusion with 10% buffered formalin, pH 7.2. They were excised, fixed, in the same fixative for 2 days at 4 °C, and embedded in paraffin.

Kidney and liver sections (4 μm thick) were placed on glass slides and immunohistochemically stained using the avidin-biotin-peroxidase complex method (11). The slides were deparaffinized and soaked in 0.3% hydrogen peroxide in absolute methanol at 50 min at room temperature. After hydration and rinsing in PBS (10 ml phosphate buffer (pH 7.2) containing 0.85% NaCl), the sections were treated with a 0.1% detergent for 10 min at room temperature to reduce nonspecific staining and then incubated at 4 °C overnight with 3 μg/ml of polyclonal antibodies against PSP in PBS containing 0.1% bovine serum albumin in a moist chamber. The sections were rinsed in PBS and incubated for 50 min at room temperature in a 1:200 dilution of biotinylated goat anti-rabbit IgG (Vectastain ABC kit, Vector Laboratories, Inc.). Following a rinse in PBS, the sections were incubated for 60 min at room temperature in the avidin-biotin complex, rinsed in PBS, stained for 5 min with 50 mM Tris-HCl (pH 7.6), containing 0.1% 3,3’-diaminobenzidine tetrahydrochloride, 0.02% hydrogen peroxide, and 0.65 mg/ml of sodium azide. After washing with PBS, the section was counter-stained for 10 min with 1% methylgreen, dehydrated, and mounted.

Controls were prepared in the above-mentioned manner, except that nonimmunized rabbit IgG was used instead of the first antibody.

**Screening of a λgt11 Library—**A rat liver cDNA library was purchased from Clontech Laboratories Inc. The screening of the λgt11 library was carried out as described previously (4). The library was propagated in Escherichia coli Y1090, and plagues were transferred to nitrocellulose filters by standard procedures (12). Polyclonal antisera raised against the purified PSP were preadsorbed with extracts from E. coli BNN97 (pico Blue Immunodetection kit, Stratagene) as described by the manufacturer. The preadsorbed serum was used in a 1:500 dilution to screen the cDNA library using duplicate filters blotted from each plate. After an overnight incubation with the anti-PSP antiserum at room temperature, the duplicates filters were washed with TBST (20 mM Tris-HCl, pH 7.5, 0.15 mM NaCl, and 0.05% Tween 20). The filters were incubated with goat anti-rabbit IgG antibody-alkaline phosphatase conjugate (pico Blue Immunodetection kit, Stratagene), and peroxidase reactions were performed as indicated by the supplier. Screening of 1 × 106 plaque-forming units led to the isolation of one positive clone PSP1. The recombinant clone PSP1 had its insert excised and subcloned into the pUC18 vector (Takara, Japan), generating clone pPSP1. The nucleotide sequence of the insert was determined by the dideoxy chain termination procedure of Sanger (13) employing BcaBEST DNA sequencing kit (Takara, Japan).

**Southern Blotting of Rat Genomic DNA—**Twenty μg of rat liver DNA was digested with HindIII, PstI, EcoRI, and BamHI and analyzed by electrophoresis on 1.0% agarose gels. Following transfer to a nitrocellulose filter, hybridization to a [32P]dCTP-labeled pPSP1 was performed under high stringency (washing with 0.1 × SSC, 0.1% SDS, 65 °C for 1 h).

**Northern Blotting of Rat Tissue RNA—**Total RNA was prepared from fresh rat tissues by the method of Chomezynski and Sacchi (14) using Trizol reagent (Life Technologies, Inc.). For each tissue, 20 μg of total RNA was run on a 1% agarose gel and transferred onto a nitrocellulose membrane. The filter was probed using the same pPSP1, and the washing was done in the same manner as described for the Southern blot analysis.

**Homology Search—**Proteins homologous with the PSP have been searched in protein data base, NBRF, using a Genetyx Macintosh software. The alignment of homologous proteins was carried out by the software.

**RESULTS**

Purification and Characterization of the PSP—In order to examine the subcellular localization of the PSP, the crude nuclear fraction, the mitochondria-lysosome fraction, and the postmitochondria supernatant fraction were prepared from the homogenates of rat liver. Proteins including the PSP were extracted from each fraction by 5% PCA, followed by precipitation with 25% trichloroacetic acid. The proteins extracted were electrophoresed on 15% SDS-polyacrylamide gels. One intense band was observed in addition to those corresponding
Isolation and Characterization of a PCA-soluble Protein

The PSP was shown to be insensitive to automated Edman degradation, indicating that peptide I is the N-terminal peptide. Therefore peptide I was treated with the acyl amino acid-releasing enzyme, and the deblocked peptide was sequenced by automated amino acid sequencer. The other eight peptides were directly sequenced. The amino acid sequences determined of the nine peptides were summarized in Table I. The amino acid sequence of the protein. Screening of the cDNA library with the anti-PSP antiserum led to the isolation of one positive cDNA clone, pPSP-1, which was shown, by restriction enzyme analysis, to contain an insert of 0.9 kb in size (data not shown). The complete sequence of cDNA pPSP-1 presented an open reading frame of 411 base pairs encoding a 136-amino acid protein. The deduced amino acid sequence was fully covered with the amino acid sequences of the above nine peptides as shown in Table I. Finally the PSP was established to be a 136-amino acid protein with a molecular mass of 14,149 Da. Poly(A) signal was located at nucleotide 852. The homology search in the NBRF data base revealed that the sequence of the PSP was completely matched with the sequence of the 10-kDa protein reported by Levy-Favier et al. (6), except for 37 N-terminal amino acid residues and the C-terminal amino acid residue (Fig. 4). Also the search found that the PSP showed 70% sequence similarity to ORF 1 from Azotobacter vinelandii as shown in Fig. 5. The ORF 1 was found as a hypothetical protein located upstream from the vnfA gene, the product of which appears to be required for the synthesis of nitrogenase-2 in A. vinelandii, but the function of ORF 1 is unknown (15).

The pPSP-1 insert, encoding the PSP, was 32P-labeled by random-primed synthesis and used as a probe in a Southern
blot of rat liver DNA digested with different restriction enzymes. The Southern blot analysis demonstrated a single hybridization band for each restriction digest (Fig. 6). These results suggest that in the rat genome there is one copy of the PSP gene.

Tissue Distribution of the PSP—The PSP in the homogenate samples obtained from various tissues was examined by immunoblotting with the antisera raised against PSP in New Zealand and White rabbits. The PSP was found to be only in liver and kidney (Fig. 7). On the other hand, the Northern blot analysis of mRNA purified from various tissues using the PSP-specific probe showed that the size of the mRNA for the PSP was 0.9 kb, and its mRNA was detected only in liver and kidney. (Fig. 8).

Rat liver (Fig. 9A) and kidney (Fig. 9B), which were stained with the antisera, were observed by a light microscope. In the liver, positive reaction products were observed in both the nuclei and cytoplasm of most hepatocyte cells. Some of these cells along the interlobular connective tissue were more intensely stained than cells in other sites. The interlobular connective tissue, arteries, veins and bile ducts were less stained. In the kidney, immunopositive deposits were seen in both the nuclei and cytoplasm in some epithelial cells of the upper renal tubules of the cortex. The glomeruli were not immunostained. No reaction products were observed in any control sections prepared using nonimmunized rabbit IgG as the first antibody (data not shown).

Effect of the PSP on Cell-free Protein Synthesis—The effect of the PSP on cell-free protein synthesis was examined by measuring the inhibition of de novo protein synthesis in a cell-free rabbit reticulocyte lysate (Fig. 10). The PSP exhibited a dose-dependent inhibition of protein synthesis and 50% inhibition (IC50) at a concentration of 8 nM, indicating that the PSP inhibits in vitro protein synthesis though IC50 of the PSP that is 20-fold higher than that of RNase A (IC50; 0.4 nM). For the first several minutes, the protein synthesis proceeded in the presence of the PSP at the same rate as that the absence of the PSP, but the PSP showed the strong inhibition of the protein synthesis with a prolonged incubation (Fig. 11). The inhibition was stimulated as the concentration of the PSP increased. For the initial period, the protein synthesis was not inhibited even at relatively high concentrations of the PSP. The inhibition of protein synthesis in the reticulocyte lysate by the PSP was shown to be accompanied with the disaggregation of polyribosomes in the lysate (Fig. 12). In order to eliminate the possibility that the polyribosome disaggregation was due to RNase activity in the PSP fractions, we investigated a trace of RNase activity in the PSP preparation obtained (Fig. 13). RNase A clearly showed RNase activity at a concentration of 1 ng, but the PSP did not show any RNase activity at a concentration up to 1 μg in the activity staining on the polyacrylamide gel (Fig.

**Fig. 2.** ESI mass spectra of the N-terminal peptide of the PSP (A) and its deblocked peptide (B). The N-terminal peptide of the PSP was isolated as described in the text. The deblocked peptide was obtained from the N-terminal peptide by digestion with the acyl amino acid-releasing enzyme.
When the RNase activity was measured using yeast tRNA as a substrate, RNase A showed activity at 1 nM of protein, but the PSP did not show any activity even at 1 mM of the protein (Fig. 13B). On the other hand, the inhibitory activity of the cell-free protein synthesis by the PSP was lost by heat treatment and precipitation with antisera against PSP (data not shown).

**DISCUSSION**

We have purified to homogeneity a novel protein that was co-extracted with H1 and HMG proteins by perchloric acid. By polyacrylamide gel electrophoresis, its molecular mass was found to be 30 kDa under the nonreducing condition and 14 kDa under the reducing condition. This native protein thus consists of two identical subunits with a molecular mass of 14 kDa.

Recently, Levy-Favatier et al. (6) found a 10-kDa perchloric acid-soluble protein in rat liver and cloned a cDNA that consists of an open reading frame of 297 base pairs and encodes a 99-amino acid protein with a molecular mass of 10,355 Da. The first half of the deduced polypeptide sequence presents 27% similarity with a region of the 83-90-kDa hsp, which is highly conserved from Drosophila to human. The hsp has been shown to belong to the class of molecular chaperones that plays a role...
in the folding of proteins (16). On the basis of this fact, they proposed that the perchloric acid-soluble 10-kDa protein could play a role as a molecular chaperone. The nucleotide sequence of the cDNA reported by Levy-Favatier et al. (6) was shown to be fairly consistent with that of the cDNA encoding our protein, although some differences were observed. Eventually, the amino acid sequence of the protein is completely identical with sequence 38–136 of our protein. We carefully examined the nucleotide sequence reported by Levy-Favatier et al. (6) and noticed that if G is inserted at nucleotide at 76, their nucleotide sequence could be fully identical with our nucleotide sequence, indicating that their nucleotide sequence codes for amino acid sequence 1–37 of our protein. We repeatedly carried out the sequencing of our cDNA and confirmed that the present nucleotide sequence of our cDNA is correct (Fig. 4).

![FIG. 6. Southern blot analysis of the PSP gene. Total rat liver DNA (10 μg/lane) was digested with BamHI (lane 1), EcoRI (lane 2), and PstI (lane 3) and processed as described under "Experimental Procedures." The positions of HindIII fragments of λ phage DNA are indicated at the left (23.1, 9.4, 6.5, 4.3, 2.3, 2.0, 0.5 kb in descending order).](image)

![FIG. 7. Immunoblot analysis of the PSP from various rat tissues. Twenty μg of proteins in the homogenates from various tissues were electrophoresed on a 15% SDS-polyacrylamide gel. Immunoblot analysis was carried out as described under "Experimental Procedures."](image)

![FIG. 8. Northern blot analysis of the PSP mRNA from various rat tissues. Total rat liver RNA (20 μg/lane) was loaded on the gel and hybridized to a [32P]dCTP-labeled EcoRI fragment pPSP-1 as described under "Experimental Procedures." The positions of 28 and 18 S are indicated at the left. Also PSP mRNA of 0.9 kb is indicated by the arrowhead.](image)
residue. The C-terminal amino acid residues of our protein was determined to be a Leu residue by amino acid sequencing (Table I). The nucleotide sequence encoding the C-terminal region of our protein was repeatedly sequenced, and the nucleotide 442 was determined to be C (Fig. 4). These findings clearly demonstrate that our protein is identical with that reported by Levy-Favatier et al. (6) and that our protein is a 136-amino-acid protein with a molecular mass of 14,149 Da, the N terminus of which is acetylated.

Interestingly, the PSP showed a high similarity (70%) with a
hypothetical protein ORF1 from A. vinelandii. "Experimental Procedures." Lane 1, RNase A (1 ng of protein); lane 2, PSP (10 ng); lane 3, PSP (1 µg). B, RNase activity was determined as described under "Experimental Procedures." •, RNase A; ■, the PSP.

The PSP was shown to be only abundant in kidney and liver by immunoblotting, Northern blotting, and immunohistochemistry (Figs. 7–9). The protein is especially localized in some epithelial cells of the upper renal tubules in the kidney cortex and in the cells along the interlobular connective tissue of liver (Fig. 9). We have recently purified a PSP-like protein from rat heart. The antisera against the PSP-like protein was shown to cross-react with that from rat muscle, and the PSP-like protein inhibited cell-free protein synthesis at a concentration of 2 nM as the PSP did (data not shown). These findings are the first report on a new inhibition mode of cell-free protein synthesis by the PSP and PSP-like protein and indicate that these proteins may play an important role in the regulation of protein synthesis in rat tissues.

REFERENCES
1. J. ohns, E. W. (1982) in The HMG Chromosomal Proteins (ohns, E. W., ed) pp. 69–87, Academic Press, New York
2. E. H. and G. B. (1989) Exp. Cell Res. 156, 295–310
3. O. T., N. Y., I. M., N. K., K. I., and Matsukura, T. (1982) J. Nutr. 112, 1504–1514
4. O. T., I. M., K. I., M. Y., T. K., and N. Y. (1983) J. Nutr. 113, 20–22
5. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
6. O. T., S. Y., T. Y., and N. Y. (1983) J. Nutr. 113, 20–22
7. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
8. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
9. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
10. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
11. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
12. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
13. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
14. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
15. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
16. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
17. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
18. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
19. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
20. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
21. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
22. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
23. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
24. O. T., S. Y., T. Y., K. I., and N. Y. (1983) J. Nutr. 113, 20–22
Isolation and Characterization of a Novel Perchloric Acid-soluble Protein Inhibiting Cell-free Protein Synthesis
Tatsuzo Oka, Hideaki Tsuji, Chie Noda, Kentaro Sakai, Yeong-man Hong, Isao Suzuki, Saturnino Muñoz and Yasuo Natori

J. Biol. Chem. 1995, 270:30060-30067.
doi: 10.1074/jbc.270.50.30060

Access the most updated version of this article at http://www.jbc.org/content/270/50/30060

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 22 references, 5 of which can be accessed free at http://www.jbc.org/content/270/50/30060.full.html#ref-list-1