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# Identification of the Interaction between Rat Translationally Controlled Tumor Protein/IgE-dependent Histamine Releasing Factor and Myosin Light Chain

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**The translationally controlled tumor protein (TCTP), also known as the IgE-dependent histamine releasing factor (HRF), was used in the yeast two-hybrid system to screen the interacting molecules. We obtained the N-terminus truncated rat fast myosin alkai light chain from the rat skeletal muscle cDNA library in the screening. Since either TCTP/HRF or the myosin light chain is known to be associated with histamine secretion from RBL-2H3 cells, we investigated the possible interaction between rat TCTP/HRF and nonmuscle myosin light chain in these cells. We used affinity chromatography and coimmunoprecipitation. Our data suggests that HRF and the myosin light chain interact, which may play an important role in histamine release in RBL-2H3 cells.**

**Keywords:** Affinity chromatography, Coimmunoprecipitation, IgE-dependent histamine-releasing factor (HRF), Myosin light chain, Yeast two-hybrid

## Introduction

The translationally controlled tumor protein (TCTP)/IgE-dependent histamine-releasing factor (HRF) was initially described as a growth-related protein in mouse ascites and erythroleukemic cells (Yenofsky *et al.*, 1983). The mouse protein was also designated as P21 (Chitpatima *et al.*, 1988) and the human homologue as P23 (Gross *et al.*, 1989). HRF was found in a number of normal cell types, and was not restricted to tumor cells (Sanchez *et al.*, 1997). Recent compilations of the HRF sequence revealed a high degree of conservation among all eukaryotic phyla. This suggests that HRF may play a crucial role in cell functions. Although the exact cellular function of HRF is not fully understood, several of its features have been reported. These include calcium binding (Haghighat *et al.*, 1992; Sanchez *et al.*, 1997; Kim *et al.*, 2000), metal homeostasis (Sturzenbaum *et al.*, 1998), tubulin binding (Gachet *et al.*, 1999), self-interaction (Yoon *et al.*, 2000), B cell growth factor (Kang *et al.*, 2001), and decreased brain HRF in neurodegenerative/dementing disorders (Kim *et al.*, 2001). HRF also induces the secretion of histamine (Macdonald *et al.*, 1995) and interleukin (IL)-4 (Schroeder *et al.*, 1996; Kim, 1997) from human basophils in the presence of immunoglobulin E (IgE). In the present study, a yeast two-hybrid system was used to identify the proteins that interact with HRF in order to understand its physiological roles. In yeast two-hybrid screens with full-length rat HRF as bait, a N-terminus truncated rat fast myosin alkali light chain cDNA was obtained from the rat skeletal muscle cDNA library.

Myosin plays important roles in the contraction of skeletal, cardiac, and smooth muscle. It is also important in a variety of cellular functions, such as cell mobility, cell shape change, and cytokinesis in nonmuscle cells (Seller *et al.*, 1988; Kamm *et al.*, 1989). Sarcomeric myosins and vertebrate nonmuscle myosins are all hexamers that are composed of dimers of two heavy chains (200 kd $M_r$) and two pairs of light chains (20 and 16 kd $M_r$). However, they do differ in the mechanism that mediates their contractile activity. Nonmuscle myosin light chain has been known to be phosphorylated by myosin light chain kinase (MLCK) as well as protein kinase C (PKC). The phosphorylation of the myosin light chain is also associated with secretion from rat basophilic leukemia cells (RBL-2H3) (Ludowyke *et al.*, 1989, 1996; Choi *et al.*, 1991, 1994; Kitani *et al.*, 1992; Peleg *et al.*, 1992). Although the sarcomeric myosin light chain was obtained as a molecule that interacts with HRF in yeast two-hybrid screening, it is likely that the interaction between the nonmuscle myosin light chain and HRF may occur in RBL-2H3 cells. This is because either HRF or the myosin light chain is involved in the histamine secretion from RBL-2H3 cells. RBL-2H3, a cultured analog

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of rat mucosal mast cells, has been a suitable model for the study of IgE-mediated degranulation (Barsumian *et al.*, 1981; Metzger *et al.*, 1986).

In this study, we have demonstrated that HRF and the myosin light chain interact in RBL-2H3 cells by affinity chromatography and co-immunoprecipitation. This finding suggests that the interaction may have an important role in histamine release from RBL-2H3 cells.

## Materials and Methods

**Yeast two-hybrid screening** Full-length cDNA of rat TCTP/HRF was inserted at the EcoRI-XhoI site of the LexA-fusion vector pEG202 to generate pEG202-TCTP/HRF. The yeast strain EGY48/pSH18-34/pEG202-TCTP/HRF was transformed with the rat skeletal muscle cDNA library that was fused into the pJG4-5 library vector on the glucose $Ura^-His^-Trp^-$ plate. The transformants were recovered from the glucose $Ura^-His^-Trp^-Leu^-$ and galactose $Ura^-His^-Trp^-Leu^-$ plates. They were also cultured to the stationary phase in glucose $Ura^-His^-Trp^-$ and galactose $Ura^-His^-Trp^-$ liquid media. They were then analyzed by β-galactosidase assay as previously described (Himmelfarb *et al.*, 1990). The cell growth was monitored by absorbance at the wavelength of 600 nm (Cho *et al.*, 2000a). Potential positive clones that activate the reporter genes were analyzed by restriction mapping and DNA sequencing (Lee *et al.*, 1999).

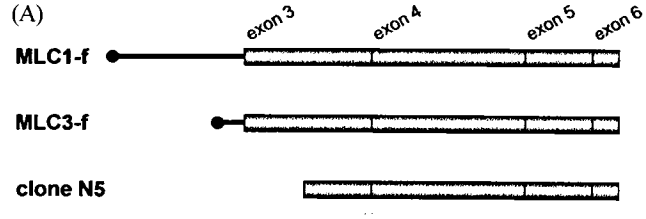
**Affinity chromatography** The *E. coli* strain *BL21(DE3)pLysS* that was transformed with pRSET (6-histidine tagged *E. coli* expression vector/Invitrogen, San Diego, USA)/HRF was grown until $A_{600}$ was 0.6-1.0. It was then induced with 0.4 mM isopropylthio-β-D-galactoside (IPTG). Cell extracts from the induced *E. coli* cells were loaded onto the $Ni^{2+}$- charged column. The column was washed with a binding buffer (40 mM imidazole, 4 M NaCl, 160 mM Tris-HCl, pH 7.9). RBL-2H3 mammalian cell extracts were then prepared by homogenization in a hypotonic lysis buffer (20 mM Hepes, pH 7.4, 2 mM $MgCl_2$, 0.5 mM PMSF, 5 μg/mL aprotinin, 1 μg/mL leupeptin, 1 mM benzamidin), loaded onto the column, and washed with the binding and wash buffers (480 mM imidazole, 4 M NaCl, 160 mM Tris-HCl, pH 7.9). In the control experiment, the loading of *E. coli* cell extracts onto the $Ni^{++}$-charged column was omitted. The other procedures were accomplished in the same manner as described previously. The proteins that bound to the column after the wash were subsequently eluted with an elute buffer (4 M imidazole, 2 M NaCl, 80 mM Tris-HCl, pH 7.9), dialyzed, and determined by the following scheme, described previously (Yoo *et al.*, 1999). They were subjected to 15% SDS-PAGE and immunoblotted with an anti-myosin light chain monoclonal antibody (Sigma, St. Louis, USA). The signal was detected with ECL (Amersham, Buckinghamshire, UK) by LAS-1000 (Fujifilm, Tokyo, Japan).

*BL21(DE3)* that was transformed with the other construct of HRF that was fused to pET-32a(+) (6-histidine tagged *E. coli* expression vector/Invitrogen, San Diego, USA) was also grown and induced with 1 mM IPTG. Furthermore, cell extracts were used in affinity chromatography as described previously.

**Co-immunoprecipitations** RBL-2H3 cells were obtained from the American Type Culture Collection. They were cultured in DMEM (high glucose, with L-glutamine) that was supplemented with 10% fetal bovine serum and penicillin/streptomycin. When the RBL-2H3 cells were 80-90% confluent, they were lysed with an ice-cold lysis buffer that consisted of 50 mM Tris-HCl, pH 7.5, 150 mM NaCl, 1% Nonidet P40, 0.25% sodium deoxycholate, Complete™ protein inhibitor cocktail (Boeringer, Mannheim , Germany), and 1 μM pepstatin. They were then centrifuged at 12,000 × g for 10 min at 4°C, and an anti-HRF polyclonal antibody was added to the supernatant. Following an overnight incubation at 4°C on a rocking platform, protein A agarose was added and the suspensions were incubated for 4 hrs at 4°C. The immune complexes were washed 3 times with an ice-cold phosphate-buffered saline and centrifuged. The pellets were resuspended in a Laemmli sample loading buffer, subjected to 15% SDS-PAGE, and immunoblotted with an anti-myosin light chain monoclonal antibody. RBL-2H3 cell extracts were also immunoprecipitated with an anti-myosin light chain antibody, then immunoblotted with an anti-HRF antibody on 15% SDS-PAGE.

## Results and Discussion

**Yeast two-hybrid screening for rat TCTP/HRF binding proteins** We screened a rat skeletal muscle cDNA library using a yeast two-hybrid system in order to identify the proteins that may interact with rat HRF. The characterization of the bait protein prior to the library screening demonstrated




(B)

EFKEAFLLFDRTGECKITLSQVGDVLRALGTNPTNAEVKKVLGNPSN
exon 3
EEMNAKKIEFEQFLPMMQAISNNKDQGGYEDFVEGLRVFDKEGNGTV
exon 4
MGAELRHVLATLGEKMKEEEVEALLAGQEDSNGCINYEAFVHIMSV
exon 5
exon 6

**Fig. 1.** Schematic representation of N-terminus truncated rat fast myosin alkali light chain (clone N5) demonstrated to interact with rat HRF in yeast two-hybrid screening. (A) MLC1-f and MLC3-f have a complete sequence homology for the first 141 amino acids in their C-termini, from exons 3 to 6 (cylinder). Clone N5, part of the homologous sequence region of MLC1 and MLC3, interacts with rat HRF. (B) The homologous amino acid sequence region of MLC1-f and MLC3-f. Clone N5 corresponded to the amino acid sequence from the 23rd amino acid of exons 3 to 6. This region is underlined. The first amino acid of each exon is indicated in bold.

**Table 1.** β-Galactosidase assay: Quantitative assay of β-galactosidase activity from the LacZ reporter gene.

| Constructs | Selective media | |
|---|---|---|
| | Glucose $Ura^-His^-Trp^-$ | Galactose $Ura^-His^-Trp^-$ |
| Positive control | 20 | 3665 |
| Negative control | 15 | 19 |
| pEG202HRF/clone N5 | 36 | 386 |

β-Gal unit = $O.D_{420} \times 1000/t$ (min) × vol. of extract (ml) × protein (μg/ml)

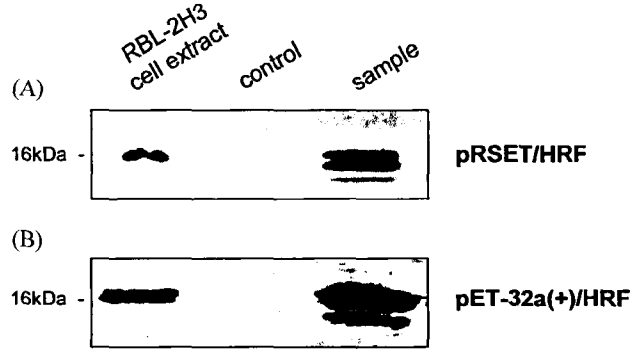



**Fig. 2.** Characterization of interaction between HRF and myosin light chain in RBL-2H3 cells through affinity chromatography. The recombinant His-tagged HRF that fused to the *E. coli* expression vector was shown to interact with the myosin light chain in RBL-2H3. Two different constructs of HRF-pRSET/HRF (A) and pET-32a(+)/HRF (B) were used in the assay. *Left lane*, RBL-2H3 cell extracts were immunoblotted with an anti-myosin light chain Ab (RBL-2H3 cell extract). *Middle lane*, the His-tagged HRF protein was not loaded onto the column, although the RBL-2H3 cell extracts were loaded and immunoblotted with an anti-myosin light chain Ab (control). *Right lane*, HRF-binding proteins from RBL-2H3 cells (eluted from the column where His-tagged HRF was bound and RBL-2H3 cell extracts were loaded) were immunoblotted with an anti-myosin light chain Ab (sample).

that it was correctly expressed as a 48 kDa $M_r$ protein, as determined by immunoblotting with a LexA polyclonal antibody (data not shown). Several positive clones were analyzed by DNA sequencing. One (clone N5) of these clones corresponded to the gene for the rat fast myosin alkali light chain, according to a computer database search using BLAST. The rat fast myosin light chain (MLC) that was obtained from the screening coincided with part of the homologous sequence region of MLC1-f and MLC3-f, the first 119 amino acids in their C-termini (Fig. 1). MLC1-f and MLC3-f were produced from a single gene by a combined process of differential RNA transcription and splicing. They have a complete sequence homology for the first 141 amino acids in their C-termini, from exons 3 to 6, although they differ in length and amino acid sequence at their N-termini (Periasamy *et al.*, 1984).

We introduced the clone N5 into the yeast cells that harbor reporter genes. It grew on the galactose $Ura^-His^-Trp^-Leu^-$ plate, but not on the glucose $Ura^-His^-Trp^-Leu^-$ plate (data not shown). We also measured the activity of the LacZ-reporter gene in the cells that were grown in the glucose $Ura^-His^-Trp^-$ or galactose $Ura^-His^-Trp^-$ media by β-galactosidase assay and clone N5. The N-terminus truncated rat fast myosin alkali light chain was demonstrated to interact with rat HRF (Table 1). In the β-galactosidase assay, pEG202α2CD3 ($3^{rd}$ domain of $Na^+$, $K^+$-ATPase α2 isotype)/pJG4-5cofilin was used as a positive control (Lee *et al.*, 2001), and pEG202/pGJ4-5 as a negative control.

**Characterization of the interaction between TCTP/HRF and myosin light chain in RBL-2H3 cells** We obtained the N-terminus truncated sarcomeric myosin light chain as a molecule that interacts with HRF in the yeast two-hybrid screening. We also studied the interaction between HRF and nonmuscle myosin light chain in RBL-2H3 cells, because either HRF or the myosin light chain is known to be associated with histamine secretion from RBL-2H3 cells.

We first investigated the interaction between HRF and the myosin light chain in RBL-2H3 cells *in vitro* through affinity chromatography. As a result, the recombinant His-tagged HRF was demonstrated to interact with the myosin light chain from RBL-2H3 cells (Fig. 2). RBL-2H3 cell extracts were loaded onto the column where his-tagged pRSET/HRF was bound. Subsequently, the HRF-binding proteins were eluted and immunoblotted with an anti-MLC antibody. The myosin light chain from the RBL-2H3 cell extract was detected as a 16 kDa $M_r$ protein, whereas no protein was detected in the control (Fig. 2A). The same results were obtained with the His-tagged pET-32a(+)/HRF fusion protein (Fig. 2B).

To demonstrate that HRF interacts with the myosin light chain *in vivo*, we immunoprecipitated the extracts from the RBL-2H3 cells with an anti-HRF antibody and analyzed the immune complex by immunoblotting with an anti-MLC antibody. As shown in Fig. 3A, the endogenous HRF was co-immunoprecipitated with the endogenous 20 kDa $M_r$ and 16 kDa $M_r$ myosin light chain in RBL-2H3 cells. We also immunoprecipitated the RBL-2H3 cell extracts with an anti-MLC antibody and immunoblotted the immune complex with an anti-HRF antibody (Fig. 3B). The endogenous myosin light chain was co-immunoprecipitated with the endogenous 23 kDa $M_r$ HRF in RBL-2H3 cells.

The RBL-2H3 cell secretion that was stimulated with antigen was accompanied by a marked alteration in cell morphology and redistribution of myosin and actin-filaments (Pfeiffer *et al.*, 1985). The exact role for these morphological changes is unknown. However, it has been hypothesized that actin microfilaments may be involved in the down-regulation of the degranulation response. Secretion by HRF in RBL-2H3 cells also includes extensive changes in cell morphology. HRF is a tubulin binding protein that is temporarily associated with microtubules and the spindle apparatus (Gachet *et al.*, 1999).

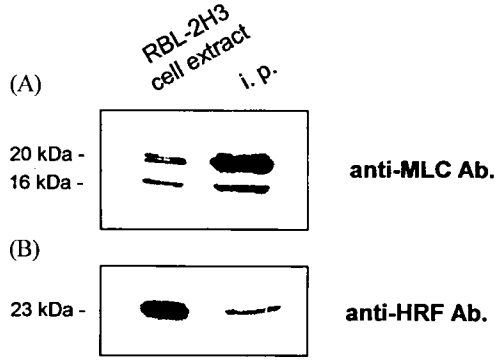



**Fig. 3.** Co-immunoprecipitation of HRF and myosin light chain in RBL-2H3 cells. (A) the RBL-2H3 cell extracts were immunoblotted with an anti-myosin light chain Ab (*left lane*). The extracts were immunoprecipitated with an anti-HRF Ab and then immunoblotted with an anti-myosin light chain Ab on 15% SDS-PAGE (*right lane*). (B) the RBL-2H3 cell extracts were immunoblotted with anti-HRF Ab (*left lane*). The extracts were immunoprecipitated with an anti-myosin light chain Ab and then immunoblotted with HRF Ab on 15% SDS-PAGE (*right lane*).

Therefore, actin microfilaments may be involved in the IgE-dependent histamine release by HRF in RBL-2H3 cells. The interaction between HRF and myosin light chain may be a clue for it. However, we do not know the exact physiological role of the interaction between HRF and the myosin light chain.

Secretion from the RBL-2H3 cells that are stimulated with antigen is associated with the phosphorylation of myosin light chains by myosin light chain kinase (MLCK), as well as by protein kinase C (PKC) (Ludowyke *et al.*, 1989, 1996; Choi *et al.*, 1991, 1994; Kitani *et al.*, 1992; Peleg *et al.*, 1992). The selective suppression of the phosphorylation by MLCK (with KT5926 or ML-7 or wortmanin) or by PKC (with Ro31-7549) inhibited the IgE-mediated histamine release from antigen-stimulated RBL-2H3 cells (Kitani *et al.*, 1992; Choi *et al.*, 1994). According to a computer aided sequence motif search using the PROSITE scan tool, HRF has a serine-98, which can be phosphorylated by PKC. Walsh *et al.* (1995) reported that the TCTP/HRF expression increased after *in vitro* PMA (PKC activator) stimulation in the human monocytoid U937 cell line. Sanchez *et al.* (1997) demonstrated that three isoforms of TCTP/HRF exist. They are likely due to differential post-translational modifications. The three isoforms have a similar molecular weight, although a differing isoelectric point, suggesting the possibility of phosphorylation. Therefore, the interaction between HRF and the myosin light chain can be altered by the phosphorylation by PKC, or dephosphorylation. These alterations can affect histamine release in RBL-2H3 cells.

In this paper, we demonstrated that rat TCTP/HRF interacts with both the muscle and the nonmuscle myosin light chain using a yeast two-hybrid system and RBL-2H3 cells, respectively. This suggests that the interaction is likely to be involved in histamine release in RBL-2H3 cells. The exact function of the interaction requires additional study.

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