Lhx3, a member of the LIM homeodomain family of transcription factors, is required for development of the pituitary and is implicated in the transcription of pituitary-specific hormone genes. In this report we describe a novel gene product, SLB, that selectively interacts with Lhx3 and the closely related LIM factor, Lhx4. The SLB cDNA encodes a 1749-residue protein that contains seven WD40 repeats near the amino terminus and a putative nuclear localization signal and does not contain other recognizable motifs. SLB is expressed in a tissue-specific manner with the highest concentrations of SLB mRNA in the testis and pituitary cells. We demonstrate that SLB specifically binds to Lhx3 and Lhx4 with high affinity both in vitro and in vivo. SLB has much lower affinity or no detectable affinity for other LIM domains. An expression vector for a fragment of SLB containing the LIM-interaction domain was shown to reduce expression of Lhx3-responsive reporter genes. The ability of the LIM-interacting domain of SLB to alter reporter gene activity as well as the tissue-specific expression and the specificity of SLB binding to LIM factors suggest a possible role in modulating the transcriptional activity of specific LIM factors.

LIM homeodomain proteins comprise a family of transcription factors that are important regulators of development (1–3). Lhx transcription factors contain a homedomain DNA-binding motif and two LIM domains each consisting of two cysteine/histidine zinc fingers. It has recently become clear that specific Lhx proteins are important regulators of pituitary development and gene expression (4–9). Disruption of the lhx3 gene in mice and Drosophila melanogaster has demonstrated a role for Lhx3 in specification of motor neuron subtype identity and pathway selection (8, 9). Studies of mutant mice with disruptions of the lhx3 and lhx4 genes have further demonstrated the role of these LIM factors in organogenesis of the pituitary gland and in differentiation and proliferation of pituitary cell lineages (6, 7).

In addition to their roles in pituitary development, Lhx2 and Lhx3 also play a role in stimulating the expression of some pituitary-specific genes (5, 10, 11). For example, basal transcription and hormonally regulated expression of the glycoprotein hormone α subunit gene involves a binding site for Lhx2 and/or Lhx3 (5, 10, 12). The homeodomain of Lhx3 can also bind to DNA elements within the thyroid-stimulating hormone β subunit gene and the prolactin gene (5). Lhx3 can also act synergistically with Pit-1 to activate reporter genes containing promoter sequences from these same genes (5, 13). Transfection of an expression vector for Lhx3 into the AtT20 pituitary cell line can induce expression of the silent, endogenous prolactin gene in the absence of Pit-1 expression (11).

The LIM domain likely functions as a modular protein-protein interaction surface (1–3). For LIM homeodomain factors, the LIM domain may modulate the DNA binding affinity of the homeodomain (1–3). The LIM domain has also been shown to bind to a widely expressed nuclear adapter protein designated NLI, LBD, or CLIM (14–17). Genetic experiments have provided evidence that CHIP, the Drosophila homolog of NLI, functionally cooperates with LIM factors to modulate transcription (18, 19).

We have cloned a novel LIM-interacting protein that contains a WD40 repeat. The remainder of the protein other than the WD40 domain has no substantial similarity to other known proteins. This 190-kDa protein is expressed in a tissue-specific manner with the highest expression in testis and pituitary. Unlike NLI, which binds to all nuclear LIM domain factors, this protein binds selectively to Lhx3 and Lhx4.

MATERIALS AND METHODS

Cell Culture, DNA Constructs, and Transfections—GH3 cells were maintained in DMEM supplemented with 15% equine serum and 2.5% fetal bovine serum. Rat-1 cells were maintained in DMEM containing 10% calf serum. All other cells were maintained in DMEM supplemented with 10% fetal bovine serum. Reporter genes containing 0.6 kilobase pairs of 5′-flanking region of the rat prolactin gene fused to the firefly luciferase coding sequence (20) and 5 copies of a GAL4-binding site upstream of the E1b TATA box linked to luciferase (21) have been described previously. Mammalian expression vectors for GAL4 and VP16 fusions have been described previously (22). The coding sequences for various LIM domains and NLI were amplified by the polymerase chain reaction using standard protocols. The products were all confirmed by automated DNA sequencing. Cells were transfected with a total of 1 μg of DNA and 5 μl of LipofectAMINE (Life Technologies, Inc.) in 35-mm well plates using a protocol provided by the supplier.

Yeast Two-hybrid Screen for Lhx3-interacting Factors—The two-hybrid screen described by Hollenberg et al. (23) was used to identify cDNAs for factors that can interact with Lhx3. Briefly, the polymerase chain reaction was used to prepare an Lhx3 cDNA fragment coding for amino acids 25–136 which was subcloned into the vector pBTM116. The ade2 gene was also subcloned into pBTM116 to allow the host strain L40 to be cured of the bait vector. A library of GH3 cDNA fused to the VP16 transcriptional activation domain was constructed using the pVP16 vector as described (23). Yeast transformations, curing, and mating to the strain AM170 were carried out as described (23) with the exception of the inclusion of 3-amino-1,2,4-triazole to increase stringency of selection.

Isolation of an SLB cDNA Containing an Extended Open Reading Frame—The SLB cDNA fragment isolated from the two-hybrid screen was used to screen a λ Zap II rat testis cDNA library (Stratagene) using
standard protocols. The library contains cDNA prepared from the testis of 6-week-old Sprague-Dawley rats. A polymerase chain reaction approach was used to isolate cDNAs representing the 5’ and 3’ termini of SLB using commercial reagents and protocols provided by the supplier (Marathon cDNA Amplification Kit, CLONTECH).

Hybridization Analysis of SLB mRNA—Poly(A)-containing cellular RNA was isolated by solubilizing GH3 or HeLa cells in guanidine HCl and sedimentation through cesium chloride as described (24) followed by chromatography of oligo(dT)-cellulose (25). The poly(A)-containing RNA (2 μg) was electrophoresed through an agarose gel containing formaldehyde (26) and transferred by blotting to a nylon filter. A membrane containing size-fractionated poly(A) RNA from several rat tissues was purchased from CLONTECH. The blots were hybridized with a 32P-labeled SLB cDNA fragment of about 1000 base pairs using hybridization buffers purchased from CLONTECH and following hybridization and wash conditions provided by the supplier.

Preparation of Nuclear and Whole Cell Extracts—Cell monolayers were washed once with ice-cold 0.15 M NaCl, 10 mM Hepes, pH 7.4, and then scraped from the dish in 5 ml of 10 mM Hepes, pH 7.4, 1 mM EDTA, 5 mM dithiothreitol, 1 mM benzamidine, and 0.2 mM phenylmethylsulfonyl fluoride (homogenization buffer). Cells were homogenized with 10 strokes of the tight pestle of a Dounce homogenizer, and the homogenate was centrifuged through a cushion of 0.5 M sucrose in homogenization buffer at 1200 × g for 10 min at 4 °C. Nuclear pellets were resuspended in homogenization buffer containing 0.4 M NaCl. After 10–20 min on ice the mixture was centrifuged at 10,000 × g for 10 min at 4 °C, and the supernatant was saved as the nuclear extract. The cell pellets were collected by centrifugation. The protein A or anti-FLAG-agarose (Eastman Kodak Co.) was then resuspended in homogenization buffer containing equal amounts of total protein were combined with 15 μl of a 50% slurry of protein A-agarose (Santa Cruz Biotechnology) or, in some cases, anti-FLAG agarose (Eastman Kodak Co.). The immunoprecipitation mixtures were rotated for 2 h at 4 °C, and the protein A-agarose was collected by centrifugation. The protein A or anti-FLAG-agarose was then washed 3 times with 1 ml each of 10 mM Tris, pH 7.4, 150 mM NaCl) with 0.1% Tween 20 in a final volume containing size-fractionated poly(A) RNA from several rat tissues was purchased from CLONTECH. The blots were hybridized with a 32P-labeled SLB cDNA fragment of about 1000 base pairs using hybridization buffers purchased from CLONTECH and following hybridization and wash conditions provided by the supplier.
library made from GH3 cDNA fused to the VP16 coding sequence. About 100 colonies survived and were tested for transactivation of the β-galactosidase gene under control of a LexA operator. False positives were identified by curing the yeast of the LexA/Lhx3 bait and mating to a yeast strain carrying a LexA/Lamin bait. Clones that interacted with lamin were excluded. A sampling of the remaining clones were then tested in a mammalian two-hybrid assay (22) by subcloning the VP16/cDNA fusions from the yeast library plasmid into a eucaryotic expression vector. These were transfected into GH3 cells with an expression vector coding for a GAL4 DNA binding domain fusion to the LIM domains of Lhx3 and a GAL4-responsive luciferase reporter. Only one clone demonstrated a strong interaction (data not shown). This clone was subsequently shown to bind to a limited number of LIM homeodomain transcription factors (see below). Therefore we designated this clone as SLB for selective LIM domain-binding protein.

Initial analysis of total RNA from rat tissues suggested that the highest levels of SLB transcripts were found in the testis. A near full-length cDNA was obtained by screening a rat testis cDNA library with the DNA probe isolated from the two-hybrid screen. The 5' and 3' ends of the SLB coding sequence were obtained by polymerase chain reaction amplification of cDNA termini from rat testis cDNA. The rat SLB cDNA encodes a 1749-amino acid protein. Comparison of the predicted protein sequence of rat SLB to the GenBankTM database reveals that the Caenorhabditis elegans genome contains a similar open reading frame which predicts a 1758-amino gene product. The predicted C. elegans protein is 37% identical and 57% similar to the rat protein over the entire length. The coding sequences are co-linear over the entire sequence with only a few gaps. The first 250 amino acids of SLB contain a seven WD40 repeats similar to those found in a number of proteins including G protein β subunits (30). A putative bipartite nuclear localization signal is located near the amino terminus in the first WD40 repeat. (31). This putative bipartite nuclear localization signal consists of the sequence RRRKSTDTPADMKYGRK (where boldface indicates consensus residues) that fits the consensus proposed by Robbins et al. (31) of two basic residues, a spacer of 10 amino acids and a second basic cluster with 3 out of 5 amino acids being basic. The remaining 1500 amino acids of SLB have no significant similarity to any known gene product in the current GenBankTM database.

Hybridization analysis of poly(A)-containing RNA (Fig. 2) demonstrates the greatest expression of SLB transcripts in testis with significant expression also detectable in pituitary and the GH3 pituitary, lactotroph, cell line. Although SLB mRNA is expressed at lower levels in the pituitary and GH3 cells than in testis (less testis RNA was loaded for the right panel of Fig. 2), the significant expression of SLB in these pituitary cells is consistent with a possible function in this tissue. The apparent size difference between testis and pituitary SLB transcripts was not observed in other experiments.

A GST fusion to the SLB LIM-interaction domain was used to immunize rabbits for production of antiserum. The specificity of the resulting antiserum was tested by transfecting COS-7 cells with expression vectors for carboxyl-terminal fragments of SLB, either SLB-(1213–1540) or SLB-(1213–1749). Cell extracts from the transfected cells were then resolved by denaturing gel electrophoresis, and the SLB antiserum was used to detect immunologically related proteins (Fig. 3A). The antiserum strongly recognized bands of the appropriate size in cell extracts expressing the fragments of SLB. The antiserum was then used for immunoblot analysis of nuclear extracts from GH3 pituitary cells and from Rat-1 fibroblasts (Fig. 3B). A band of approximately 190 kDa was observed only in GH3 nuclear extract.

An additional two-hybrid screen of the VP16/GH3 cDNA library was performed with the SLB-(1213–1265) LIM-interaction fragment as bait. Approximately 1 million yeast transformants were screened, and six colonies survived. Five of the colonies contained identical cDNA fragments coding for the second LIM domain of Lhx3. We were unable to isolate the pVP16 plasmid from the sixth colony. This confirms the original yeast two-hybrid interaction and demonstrates that the second LIM domain of Lhx3 is sufficient to bind SLB.

SLB Binding to Lhx3—To determine if SLB can directly interact with Lhx3, the GST-SLB-(1213–1749) was used for binding studies. SLB-(1213–1749) contains the region that is sufficient for interacting with Lhx3 in the yeast two-hybrid assay plus additional carboxyl-terminal residues. Radiolabeled mouse Lhx2 and Lhx3 were incubated with immobilized GST or GST-SLB fusion proteins, and the bound proteins were analyzed by denaturing gel electrophoresis (Fig. 4). Neither Lhx2 nor Lhx3 bound to GST, and only Lhx3 bound to the GST-SLB fusion protein. It appears that Lhx3 has a rather high affinity for this fragment of SLB as more than 50% of the input Lhx3 was bound to SLB as determined by PhosphorImager analysis. In control experiments, neither Pit-1 nor the estrogen receptor bound to the GST-SLB fusion protein (data not shown). The failure of SLB to interact with Lhx2 suggests that SLB has considerable selectivity for interacting with specific LIM homeodomain factors. This is particularly interesting as the LIM domain of Lhx2 is 47% identical to the LIM domain Lhx3, and both factors are expressed in the pituitary. This finding contrasts with the ability of the putative LIM coactivator, NLI, to bind to a wide variety of LIM factors (14–16). Thus the in vitro binding data confirm the two-hybrid data and demonstrate a direct and selective interaction of SLB with Lhx3.

We used the SLB antiserum to examine the interaction of Lhx3 and SLB in vitro. To date, we have not been able to detect the interaction of endogenous SLB and Lhx3 by co-immunoprecipitation assays. In part, this is probably due to relatively low
expression of SLB in GH3 cells. It has also been somewhat difficult to test the interaction of SLB and Lhx3 in intact cells by forced expression in heterologous cells. Experiments using expression vectors for SLB tagged with various reporters have suggested that overexpression of SLB appears to be toxic to most cells. Fortunately, 293 cells appear to be somewhat resistant to the toxic effects of SLB expression. Also, the carboxyl-terminal fragment of SLB which contains the LIM-interacting domain (SLB-(1213–1749)) is not toxic in 293 or other cells. By using 293 cells it has been possible to demonstrate co-immunoprecipitation of FLAG-tagged Lhx3 with either full-length SLB or SLB-(1213–1749) (Fig. 5). No SLB was co-immunoprecipitated in cell extracts expressing SLB alone or with untagged Lhx3. These co-immunoprecipitation experiments provide evidence that SLB can bind to Lhx3 in vivo.

To examine further in vivo interaction of SLB with Lhx3, the subcellular localization of these proteins was examined after transfecting COS-7 cells (Fig. 6). COS-7 cells do not contain detectable SLB mRNA, and no immunoreactive SLB was detected in untransfected cells. To assist in identifying the nuclear compartment, DNA was visualized with Hoechst stain (Fig. 6, right panels). Transfection of an expression vector for SLB-(1213–1749) resulted in the distribution of SLB immunoreactivity throughout the cell including both the cytoplasmic and nuclear compartments. A putative nuclear localization signal that is located near the amino terminus is deleted from SLB-(1213–1749). Thus the approximately 60-kDa SLB-(1213–1749) fragment probably passively distributes throughout the cytosol and the nucleus. When SLB-(1213–1749) was co-transfected with an Lhx3 expression vector, immunoreactivity was located predominantly in the nucleus, consistent with an interaction between the two proteins in vivo. An expression vector for Lhx2, which does not bind SLB, was used as a control. Lhx2 did not result in nuclear concentration of SLB-(1213–1749). These findings offer further evidence that SLB can interact with Lhx3 in cells. Indeed, the finding that expression of Lhx3 results in localization of the majority of SLB to the nucleus is consistent with a rather high affinity interaction.
Selective LIM-binding Nuclear Factor

Fig. 6. Co-expression of Lhx3 results in changes in the subcellular localization of SLB-(1213–1749). COS-7 cells were transfected with expression vectors for a Lhx3, Lhx2, SLB-(1213–1749) (SLB\text{CooH}), or the indicated combination. The amount of expression vector was kept constant by the inclusion of empty expression vector. Cells were then fixed and immunostained with a 1:50 dilution of the SLB antiserum. Hoechst nuclear stain was included in the incubation of Cy3-labeled anti-rabbit secondary antibody. Cells were visualized in both the Hoechst channel (right panel) and the Cy3 channel (left panel).

To explore the specificity of SLB binding to LIM homeodomain proteins a binding assay using immobilized GST-SLB-(1213–1749) was used (Fig. 7). Expression vectors for the LIM-domain GAL4 fusion proteins were transfected into 293 cells. Extracts from these cells were then incubated with agarose-bound GST-SLB-(1213–1749) fusion protein, and the bound proteins were analyzed by denaturing gel electrophoresis. The various GAL4 fusion proteins were visualized by Western blotting with a monoclonal antibody to the GAL4 DNA binding domain. The results demonstrate that substantial amounts of Lhx3 and Lhx4 bound the immobilized SLB fragment. Lhx2 and Lmx-1 bound much less efficiently, and Isl-1 and Lin-11 did not demonstrate binding. The observation of weak binding of Lhx2 to SLB in this experiment appears to contrast to the absence of binding observed in Fig. 4. This is likely due to the presence of substantially greater concentrations of the LIM proteins in extracts from transfected 293 cells as compared with the amount of protein synthesized in the in vitro transcription/translation reactions. The higher concentration of the LIM factors in 293 extracts would facilitate detection of the weaker binding of SLB to Lhx2. These data provide additional evidence that SLB interacts selectively with specific LIM domains.

Transcriptional Effects of Lhx3 and SLB—The finding that SLB can bind to Lhx3 and Lhx4 suggests a possible role in modulating transcription. The ability of the LIM-interacting domain of SLB (residues 1213–1749) to function as a possible dominant negative was tested in transfection experiments. The co-immunoprecipitation and nuclear co-localization experiments provided evidence that SLB-(1213–1749) can associate with Lhx3 in the nucleus. The ability of SLB-(1213–1749) to interfere with Ras-induced activation of a prolactin reporter gene in GH\textsubscript{3} cells was tested (Fig. 8). The SLB-(1213–1749) expression vector was found to partially block Ras-induced prolactin reporter gene activity. Although the effect was somewhat modest, it has been reproducible in several experiments. Importantly, the SLB-(1213–1749) vector did not appreciably alter the ability of a GAL4-Elk1 fusion protein to activate a GAL4-dependent reporter gene in a Ras-responsive manner (Fig. 8B). Thus, the effects of SLB-(1213–1749) are specific to the Lhx3-responsive prolactin promoter, and SLB-(1213–1749) did not inhibit a presumably Lhx3-independent, Ras-responsive transcription unit. We also tested the ability of SLB-(1213–1749) to block the function of Lhx3 (Fig. 9) in a heterologous cell line. As reported previously (13) Pit-1 and Lhx3 strongly synergize to activate the prolactin reporter gene in 293 cells. In this experiment the effects of SLB-(1213–1749) were compared with effects of NLI. Although NLI probably functions as a positive regulator of transcription (16, 19, 32), it has been somewhat difficult to demonstrate positive effects on transcription in transient transfection studies. For instance, it has been reported that NLI disrupts the synergy between the LIM homeodomain protein Lmx1 and the basic helix loop helix protein E47 (17). We found that both SLB-(1213–1749) and NLI sub-
Expression vector for SLB-(1213–1749) and SLB-(1213–1749) (SLBCOOD) as indicated. The cells also received an expression vector for β-galactosidase driven by a cytomegalovirus promoter as an internal standard. The amount of expression vector was kept constant for all transfections by the inclusion of empty expression vector. Values were corrected for β-galactosidase activity and are the average ± S.E. of three independent transfections.

Fig. 9. Expression of SLB-(1213–1749) in 293 cells strongly inhibits synergistic activation of a prolactin reporter gene by Pit-1 and Lhx3. Cultured 293 cells were transfected with a reporter gene containing the proximal region and promoter of the rat prolactin gene linked to luciferase and expression vectors for Pit-1, Lhx3, NLI, or Pit-1 and Lhx3. Cultured 293 cells were transfected with a reporter gene containing the proximal region and promoter of the rat prolactin gene linked to luciferase and expression vectors for Pit-1, Lhx3, NLI, or Pit-1 and Lhx3.

Expression Vector

**DISCUSSION**

We have identified SLB as a novel gene product that interacts with the LIM domains of Lhx3 and Lhx4. A partial SLB cDNA was isolated in a two-hybrid screen for factors that interact with the LIM domain of Lhx3. The coding sequence of rat SLB is similar over its entire length to a *C. elegans* open reading frame of unknown function. The conserved similarity from *C. elegans* to mammals suggests the possibility of a conserved function. The only clearly identifiable domain in SLB is the presence of seven WD40 repeats in the first 250 amino acids (30). The WD40 repeating unit is usually about 40 amino acids long and often ends with a tryptophan followed by an aspartate. This motif occurs in a wide variety of eucaryotic proteins but is not indicative of a specific function. It has been noted that most WD40 repeat-domain-containing proteins are involved in some form of regulation and are not enzymes (30).

We have used several different assays to examine the interaction of SLB with LIM factors. SLB binds to Lhx3 and Lhx4 both in vitro and in vivo. Interestingly, SLB selectively interacts with Lhx3 and Lhx4 and either does not bind or binds with much lower affinity to Lhx2, Lmx1, Isll, or Lin11. The LIM domains of Lhx3 are most similar to the LIM domains of Lhx4, being 79% identical. Lhx3 and Lhx4 have overlapping expression patterns and functional roles in the pituitary and specific neuronal populations (6–8, 33). It is possible that selective binding to SLB plays a role in mediating some shared activity of these two LIM factors. The selectivity of SLB binding to specific LIM factors contrasts to the lack of selectivity in binding by the LIM cofactor, NLI (14–16). Importantly, co-immunoprecipitation experiments offer evidence that SLB can interact with Lhx3 in cells. Analysis of subcellular localization provided additional evidence that SLB can associate with Lhx3 in cells. Indeed, the finding that co-transfection of Lhx3 could lead to the redistribution of an SLB fragment to the nuclear compartment suggests that under these conditions the majority of the SLB fragment is associated with Lhx3. Of course, forced expression in transient transfection experiments may not reflect physiological conditions. None the less, several different experimental approaches clearly provide evidence for relatively high affinity, selective interaction of SLB with Lhx3.

Transfection experiments using expression vectors encoding the LIM-interacting domain of SLB have provided evidence that SLB may play a role in modulating the transcriptional activity of Lhx3. In heterologous 293 cells, there is a strong synergism between Lhx3 and the pituitary-specific transcription factor, Pit-1, for activation of the prolactin promoter. Expression of the LIM-interaction domain of SLB is a potent suppressor of synergistic activation by Lhx3 and Pit-1. In view of these transfection studies and the binding data, it seems clear that SLB can interact with Lhx3 in intact cells and affect transcriptional activation. At the present, we have been unable to assess the effects of full-length SLB on transcriptional activity due to toxic effects of overexpressing this factor. It seems likely that approaches other than transient transfections will be required to address this issue. Perhaps the most informative studies of the function of NLI, a structurally different LIM-interacting factor, have involved genetic experiments in *Drosophila* (18, 19, 32). Studies have been initiated to utilize genetic model systems to explore further the functional role of SLB.

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Identification of a Conserved Protein That Interacts with Specific LIM Homeodomain Transcription Factors
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