Cloning and Complete Primary Structure of the Mouse Laminin α3 Chain

DISTINCT EXPRESSION PATTERN OF THE LAMININ α3 A AND α3B CHAIN ISOFORMS*

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We have isolated and characterized overlapping cDNA clones encoding the α3A and α3B chains of mouse laminin 5. Sequence analysis of the cDNA for the α3B predicts a polypeptide of 2541 amino acids (279,510 Da) comprising a truncated short arm and a carboxyl-terminal long arm common to the laminin α chains identified thus far. The short arm of the α3B chain harbors two alternating epidermal growth factor-like domains and two globular domains. The amino-terminal globular domain, thought to mediate interactions with molecules of the extracellular matrix, shows no significant homology to any globular domain at the tips of the known laminin isoforms. The α3A cDNA predicts a polypeptide of 1711 amino acids (186,230 Da) that substitutes a short sequence of 43 amino acids for the short arm seen in the α3B isoform and displays 77% conservative homology to the α3E β chains of the adhesion ligand epiligrin. Northern and Western blot analyses of skin and lung epithelial cells demonstrated the tissue-specific expression of the laminin α3A and α3B isoforms, and in situ hybridization on mouse embryos revealed a focal localization of α3B in areas of the central nervous system.

Laminins are noncollagenous components of basement membranes that mediate cell adhesion, growth, migration, and differentiation. These cross-shaped molecules constitute a family of proteins consisting of three individual polypeptide chains joined together in a long arm as coiled-coil amphipathic α-helices linked by interchain disulfide bonds. The amino-terminal domain of each of the three chains forms a distinct short arm (reviewed by Tryggvason, 1993).

Laminin chain variants with specific patterns of temporal and spatial expression have been identified in different species. All these isoforms are highly homologous, in that their short arms are comprised of globular domains and characteristic epidermal growth factor-like domains, and their long arms consist of sequences of heptad repeats (Timpl et al., 1979). On the basis of their primary structure deduced from sequence data and homology to the polypeptides that compose laminin 1, the laminin chains characterized thus far have been classified as α, β, or γ chains (Burgeson et al., 1994). Only chains belonging to a different class combine into a trimeric molecule presenting with a large globular domain G contributed by the carboxyl terminus of the α chain.

Epithelial cells express specific laminin isoforms. Laminin 5 was initially identified by a monoclonal antibody that stains subsets of basement membranes (Verrando et al., 1987). The protein is associated with the anchoring filaments, thread-like structures connecting the hemidesmosomes to the lamina densa of the dermal-epidermal junction (Verrando et al., 1987; Roussele et al., 1991). Laminin 5 is composed of three distinct chains of 165 kDa (α3), 140 kDa (β3), and 105 kDa (γ2). This mature species derives from a cell-associated molecule as a result of two extracellular processing events that generate the α3 and the γ2 chains from distinct 200- and 155-kDa precursor polypeptides, respectively (Marinkovich et al., 1992a). The laminin α3 chain is immunologically related to a distinct laminin 190-kDa α chain synthesized by keratinocytes that interacts with a β1 and a γ1 chain to form laminin 6. Laminin 6 and laminin 5 appear to form a complex that functions as a cell adhesion ligand for integrins α6β4 and α3β1 (Carter et al., 1990; Marinkovich et al., 1992b). In amniotic and fetal skin, the 190-kDa laminin α chain associates also with a β2 and a γ1 chain to form laminin 7 (Wewer et al., 1994).

Mutations in the genes encoding laminin 5, including its α3 chain, have been shown to underlie the junctional forms of epidermolysis bullosa, a recessive inherited skin disorder characterized by dysadhesion of the epidermis from dermis (Kivirikko et al., 1995) and references therein; Vidal et al. (1995)).

Screening for cDNA clones from a human keratinocyte expression cDNA library using a polyclonal antibody against the α3 chain of laminin 5 identified two species of mRNA transcripts*.

Partial sequence analysis predicts two polypeptides identical to the α3Epa and α3EpB chain isoforms of the adhesive ligand epiligrin (Ryan et al., 1994), an anchoring filament component shown to mediate basal cell adhesion by interacting with integrin α3β1 in focal adhesion and with integrin α6β4 in hemidesmosome adhesion structures (Carter et al., 1991). The cDNA encoding the α chain of epiligrin predicts two distinct polypeptides with identical COOH-terminal domains, homologous to domain I+II and domain G of laminin α3 chains, and totally divergent amino-terminal domains. The isoform α3Epa substitutes a short arm, thus far uncharacterized, for the truncated amino-terminal domain of the α3EpB counterpart (Ryan et al., 1994).
Cloning and Differential Tissue Expression of Laminin α3 Chains

Identification of cDNA Clones Encoding Mouse Laminin α3 Chain—Screening of a mouse lung cDNA library with a radioactive cDNA coding for the α3 chain of human laminin 5 identified cDNA clones corresponding to the full-length mouse laminin α3 chain.

In this study, we report the complete cDNA sequences of the α3 chains of murine laminin 5, which demonstrate that the laminin α3 chain harbors a short arm with unique structural features. We also provide evidence that the laminin α3B and α3A isoforms display distinct expression patterns.

MATERIALS AND METHODS

Isolation and Analysis of cDNAs—A mouse lung cDNA library (Zap, Stratagene) was screened with a randomly primed 32P-labeled (prime-it, Stratagene) cDNA done MN97 that encodes a region of the I-I domain of the mouse laminin-α3 chain (Aberdam et al., 1994). Twelve positive phage clones were excised into pBluescript SK− (Stratagene). The largest cDNA, M100 (2.2 kb), which corresponded to a 5'-end sequence of cDNA M91, (9.1 kb), and M22 (3.3 kb), were completely sequenced on both strands using a Sequenase kit (Pharmacia Biotech Inc.). To obtain additional clones, the cDNA library was re-screened at low stringency using the human cDNA NA12, which codes for the 3'-end of the human laminin-α3 chain, (Vidal et al., 1995) as a radioactive probe. A positive clone M22B (2.5 kb) containing the 3'-end of the complete nucleotide sequence of the cDNA was thus identified.

cDNA clones representing extensions of cDNA M22 were isolated by PCR amplification of the mouse cDNA expression library. The primers used for 5'-extension were as follows: 5'-CATGAGCAACACCTCTTCA-3' (left) and 5'-CCAGGAGCACACTTGTC-3' (right), which correspond to a 5'-extension sequence of cDNA M90, respectively. The primers used for 3'-extension were as follows: 5'-CATAGAGCAACACCTCTTCA-3' (left) and 5'-TAGGCTGCGCTTTCAAGTA-3' (right), which correspond to a 3'-end sequence of cDNA M22 and to a 5'-end sequence of cDNA M26b, respectively.

After purification and subcloning of the amplification products into PCRTM-II vector (TA cloning kit, InVitrogen), clones M22 (960 nucleotides) and M0.2 (254 nucleotides) were completely sequenced. Clones representing the 5'-end of the complete nucleotide sequence of the laminin-α3A and α3B cDNAs were obtained by 5'-rapid amplification of cDNA ends (5'-RACEkit; Life Technologies, Inc.).

In Situ Hybridization Analysis—Sense and antisense probes specific to laminin transcripts α3A and α3B were obtained by labeling cDNAs PR6H, M22, and M26b with digoxigenin-uridine triphosphate (Boehringer Mannheim, France). In situ hybridization on mouse fetal tissues was performed using a method devised to detect mRNA transcripts in neural tissue (Schaefer-Wiemers and Gerfin-Moser, 1993).

RESULTS

Identification of cDNA Clones Encoding Mouse Laminin α3 Chain—Screening of a mouse lung cDNA library with a radioactive cDNA coding for the α3 chain of human laminin 5 identified cDNA clones corresponding to the full-length mouse laminin α3 chain.
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Sequence analysis revealed that these cDNA clones represent two distinct transcripts, designated as α3A and α3B, homologous to the epiligrin α3Pα and α3Pβ chain isoforms, respectively (Ryan et al., 1994). The schematic structure of overlapping clones is depicted in Fig. 1A.

Nucleotide and Amino Acid Sequences of Laminin Isoform α3B—The complete amino acid sequence of the cDNA for the mouse laminin α3B chain is shown in Fig. 2A. The cDNA sequence contains an open reading frame (ORF) of 7704 nucleotides flanked by 5 nucleotides of 5′-untranslated sequence and 368 nucleotides of 3′-untranslated sequence. The 3′-non-coding sequence contains a polyadenylation signal (AAUAAA) located 16 nucleotides upstream of the poly(A) tail. The ORF encodes a protein of 2586 amino acids. The first in-frame ATG is in favorable context for initiation of translation (Kozak, 1991) and precedes a stretch of 26 hydrophobic amino acids typical of a signal peptide. According to the rule of Von Heijne (1986), a cleavage site was predicted following Gln-27. After cleavage of the signal peptide, the protein consists of 2541 residues with a predicted molecular mass of 279,510 Da. The mature protein has 11 putative N-linked glycosylation sites (Asn-X-Ser/Thr), and the molecular mass of the glycosylated peptide is estimated to exceed 300,000 Da.

Domain Structure of Laminin α3B Chain—The laminin α3B chain is comprised of a short arm of 1056 amino acids, a long arm comprising a rod-like region of 589 residues and a carboxy-terminal globular domain G of 920 residues (Fig. 2A). Recently, Ryan et al. (1994) reported the partial cDNA sequence for the α3Pβ chain of epiligrin, which resulted identical to the sequence of the α3B chain of human laminin 5 (Vidal et al., 1994). Since the cDNA sequence for the amino-terminal region of human laminin α3B chain is not available, we compared the domain structure of the short arms of mouse laminin chains α3B and α3A. Alignment with the amino acid sequence of mouse laminin α1 chain reveals that the polypeptide chain α3B harbors a truncated amino-terminal end missing the most amino-terminal domains V and VI. Therefore, the short arm of the chain comprises the cysteine-rich EGF-like domains IIIa (residues 889-1057) and IIb (residues 498-699), which are predicted to have rigid rod-like structures, and domains IVa (residues 700-888) and IVB (residues 27-497), which are predicted to form globular structures (Fig. 1B).

No significant homology is found between regions of the large amino-terminal domain IV of the α3B chain and sequences in the amino-terminal domains of the laminin isoforms characterized thus far. In particular, the conserved sequences WWQS and Y(Y/F)YX6(G/R)G, located in the amino-terminal domain VI of most of the laminin chains (Sasaki et al., 1988; Hunter et al.), are not conserved in α3B.

Double horizontal traits between the compared sequences underlined amino acid identities and single horizontal traits indicate conservative substitutions. The arrow below the putative signal peptide cleavage site. Cysteine residues are boxed, and the potential N-linked glycosylation sites (NX(S/T)) are indicated by full circles. Amino acid sequences with a putative biological interest are underlined. Asterisk delimits the carboxy-terminal sequences common to laminin α3A and α3B chains. Domains are boxed and labeled on the right. The sequence of the mouse α3B chain is available from EMBL under accession number X84014. B, nucleotide sequence of the cDNA encoding the amino-terminal domain specific to mouse laminin α3A chain (upper line) and deduced amino acid sequence (middle line). The amino acid sequence of the human epiligrin α3α chain is also reported (lower line) (Ryan et al., 1994). Conserved amino acid residues are indicated by a horizontal line and differing residues by the appropriate one-letter code. Missing or mismatching amino acid residues are depicted by hatched squares. The putative cleavage site of the peptide signal is indicated by a triangle. The mouse α3A sequence is available from EMBL under accession number X84013.
al., 1989; Beck et al., 1990; Kusche-Gullberg et al., 1992; Gerecke et al., 1994; Vuolteeho et al., 1994; Wewer et al., 1994), are not found. Domain IV of laminin α3B chain has no significant homology with domain IV of laminin α1 and α2 chains. However, it displays 28% homology (42.6% if conservative changes are included) with domain IV′ (residues 872–1374) of Drosophila laminin α chain (Kusche-Gullberg et al., 1992). On the contrary, the globular domain IVa of the mouse laminin α chain (residues 700–888) displays 19.8% homology to domain IVa of mouse laminin α1 (residues 1143–1344) and is 12 amino acids shorter. Moreover, the EGF-like domains IIIb of laminin α3B chain shows 47.2% homology with its counterpart in laminin α1 chain and is 249 amino acids shorter (Table I). The best alignment is obtained with a sequence overlapping EGFs 7–11 of domain IIIb of laminin α1 chain (between positions 981 and 1142) (Sasaki et al., 1988). Domains IIIa of laminin chains α3B and α1 are 39.5% homologous. In the α3 chain, domain IIIa retains the four EGFs that constitute domain IIIa of the α1 chain (Sasaki et al., 1988). However, EGF 4 comprises only 6 cysteine residues, which are found in conserved positions (Sasaki et al., 1988). The size of the different domains of the short arm of mouse laminin α3B chain and their sequence homology with the corresponding domains of mouse and Drosophila α chains are summarized in Table I.

The α3B chain presents 77% homology to the available sequences of the human α3B chain (Fig. 2A). 42 cysteine residues detected in the human α3B chain are conserved between the two species; two extra cysteines (positions 1441 and 1585) are found in the human sequence (Ryan et al., 1994). Domain I+I of the mouse laminin α3B chain (residues 1057–1647) matches at 77% domain I+I of the human counterpart. In the mouse, one residue (Lys-618) of the amino acid sequence is missing. A protein adhesion Arg-Gly-Asp (RGD) sequence is found at position 1512 and matches the RGD sequence of the human laminin α3A counterpart (Fig. 2B). The Gin residue at position 44 (Gln-901 in laminin α3B chain) is the first amino acid common to both α3A and α3B isoforms (Fig. 2A). The full-length α3A cDNA (5563 nucleotides) comprises a 5′-untranslated region of 62 nucleotides and an ORF (5133 nucleotides), beginning with a Met codon surrounded by sequences fitting the eukaryotic translation start sites (Kozak, 1991). The initiation methionine precedes an appropriate signal sequence of 17 amino acids with consensus cleavage site following residue Glu-18 (Von Heijne, 1986) (Fig. 2B, arrow). The ORF encodes a protein of 1711 amino acids with nine consensus sites for N-linked glycosylation. The mature peptide has a predicted mass of 186230 Da. The mass of the glycosylated peptide is estimated at 214000 Da.

Differential Expression of Laminin Variants α3A and α3B—Since previous studies on the tissue distribution of the murine laminin α3 chain were performed using probes specific to the peptide COOH-terminal domains common to both α3A and α3B variants (Aberdam et al., 1994), we investigated whether cDNA probes for the distinct amino-terminal domains of the α3 chain isoforms detected expression patterns specific to each polypeptide. We first assessed the expression rate of the α3A and α3B transcripts by Northern blot analysis of mRNA purified from mouse lung and skin epithelial cells. Hybridization performed with the radioactive CDNA M100, which encodes for the rod-like domain common to both α3A and α3B chains, identified faint bands corresponding to transcripts with a size ranging between 5.5 kb in skin cells and 8.0 kb detected only in the lung (Fig. 3A). Using cDNA M22 as a radioactive probe specific for the α3B chain, a unique 8.0-kb band was detected in lung extracts (Fig. 3B), whereas using cDNA PR6, which encodes the
amino-terminal domain of α3A, only the 5.5-kb band was specifically observed in epidermal cells (Fig. 3C). These results therefore indicated that some epithelia may express only one of the laminin α3 chain isoforms.

To verify this possibility, we further investigated the expression of laminin α3A and α3B isoforms at protein level. Western analysis was realized on total extracts prepared from mouse skin and lung using the polyclonal antibody SE152 specific to domain I-II of the two mouse α3 chain isoforms (Aberdam et al., 1994). In skin extracts, the antibody detected a band with an apparent mass of 200 kDa and a 150–165-kDa band doublet (Fig. 4), which is the electrophoretic migration pattern characteristic of the precursor and mature forms of laminin α3A chain (Marinkovich et al., 1992a; Aberdam et al., 1994). In skin extracts, the antibody detected a band with an apparent mass of 200 kDa and a 150–165-kDa band doublet (Fig. 4), which is the electrophoretic migration pattern characteristic of the precursor and mature forms of laminin α3A chain (Marinkovich et al., 1992a; Aberdam et al., 1994). In lung extracts, antibody SE152 reacted with a single band with an apparent mass of 280–300 kDa (Fig. 4), which is a value concordant with the estimated molecular weight of the polypeptide encoded by the full-length laminin α3B chain cDNA. It was thus clearly demonstrated that immunoreactivity to the polyclonal antibody SE152 in mouse skin and lung correlated with the presence in these tissues of mRNA for the laminin α3A and α3B chains, respectively. These results are therefore consistent with a cell type-specific expression of laminin α3A and α3B chain isoforms.

Focal Distribution of Laminin α3A and α3B Chain Isoforms—We then determined the tissue distribution of laminin α3A and α3B chains by in situ hybridization on mouse tissues using RNA probes specific for each isoform. According to our previous results (Aberdam et al., 1994), transcripts for both laminin α3A and α3B chains were detected in the basal membrane of the upper alimentary tract and urinary and nasal epithelia. The α3A chain appeared prominently expressed in the skin and, specifically, in hair follicles (Fig. 5A) and developing neurons of the trigeminal ganglion (13.5 days postcoitum) (Table II). Strong expression of the α3B chain was detected in the salivary glands and teeth, where the presence of α3A transcripts was also noticed. Conversely, α3B transcripts were exclusively found in the bronchi and alveoli, in the stomach and intestinal crypts, in the whisker pads (Fig. 5D), and in the central nervous system (Table II). In the brain, strong hybridization signals were seen in the telencephalic neuroectoderm (Fig. 5F) and a transient (13.5 days postcoitum), and focalized expression was also observed in the thalamus, the Rathke’s pouch, and the periventricular subependymal germinal layer (Fig. 5F).
TABLE II

| Tissue type               | cDNA probes |
|---------------------------|-------------|
|                           | PR6H | M22 | MZ6b |
| Skin                      | +    | +   | +    |
| Epidermis                 | +    | +   | +    |
| Nasal mucosa              | +    | +   | +    |
| Hair follicle             | +    | +   | +    |
| Whisker pads              | -    | +   | +    |
| Teeth                     | ±    | ±   | +    |
| Ameloblasts               | ±    | ±   | +    |
| Respiratory tract         | ±    | ±   | +    |
| Trachea                   | -    | +   | +    |
| Bronchi                   | -    | +   | +    |
| Alimentary tract          | ±    | ±   | +    |
| Salivary gland            | ±    | ±   | +    |
| Esophagus                 | +    | +   | +    |
| Stomach                   | -    | -   | -    |
| Intestine                 | -    | -   | +    |
| Kidney                    | ±    | ±   | +    |
| Collecting tubules        | -    | -   | +    |
| Central nervous system    | ±    | +   | +    |
| Trigeminal ganglion       | +    | +   | +    |
| Thalamus                  | -    | +   | +    |
| Subependymal germinal layer | -  | +   | +    |
| Telencephalic neuroectoderm | -  | +   | +    |
| Rathke’s pouch            | -    | +   | +    |

DISCUSSION

In the present study, we relate the cloning of cDNAs coding for the full-length α3A and α3B isoforms of mouse laminin α3 chain, and we demonstrate the tissue-specific distribution of these laminin variants.

Sequence data reveal that the α3B chain isoform (300 kDa) substitutes the amino-terminal short arm with two alternating cysteine-rich domains and two globular domains for the short amino-terminal peptide found in the α3A counterpart (200 kDa). These observations are concordant with previous results reporting that human laminin α3A chain harbors a short arm, consisting of a reduced thread-like structure comprised of four EGF-like repeats, and a long arm, identical to the COOH-terminal regions of the α3B chain isoform (Ryan et al., 1994). Apart from a restrained region matching 29.7% of the amino acid sequence of domains III and IV of laminin α1 chain, the short arm of mouse laminin α3B chain presents no homology with laminin α1 and α2 chains. The most amino-terminal domain of the polypeptide displays a weak sequence similarity with domain IV of Drosophila laminin α1 chain, which has been suggested to arise from the fusion of a duplicated domain IV of laminin β1 chain (Kuschke-Gullberg et al., 1992). However, in this, the two laminin α chains differ because no homology is found between the α3B chain and laminin β isoforms.

The electron microscopy images of laminin 5 purified from keratinocytes depict the molecule as a rod-like entity missing the short arms characteristic of classical laminins or the globular structures of the laminin α3B chain short arm. It has thus been suggested that the α3B transcript corresponds to the α chain polypeptide of laminin 6 (K-laminin) (Ryan et al., 1994). However, lines of evidence suggest that the α chains of these two laminins are distinct isoforms. First, the α chain of laminin 6 is a truncated polypeptide lacking the amino-terminal short arm (Marinkovich et al., 1992b). Second, the deduced molecular mass (300 kDa) of polypeptide α3B is inconsistent with the estimated mass of the α chain of laminin 6 (190 kDa) (Marinkovich et al., 1992b). No evidence for processing of the α chain of laminin 6 that could account for this discrepancy has thus far been provided. Third, synthesis of laminin 6 in H-J EB patients is not affected by mutations in the LAMA3 gene, resulting in hampered expression of α3A and α3B transcripts (Vidal et al., 1995). Therefore, detection of the 300-kDa polypeptide corresponding to the α3B transcript in lung epithelia, where the laminin α3A chain is not detected, supports the assumption of the existence, and the coexistence in some epithelia, of laminin 5 isoforms harboring distinct α3 chains.

Thus far, information on laminin 5 has been gathered from studies performed on the protein secreted by epidermal keratinocytes. In the lamina lucida of the dermal-epidermal junction, laminin 5 immunolocalizes with the anchoring filaments of hemidesmosomes (Rousselle et al., 1991; Verrando et al., 1993) and codistributes with integrin α6β4, the transmembrane receptor associated with hemidesmosomes (Sonnenberg et al., 1991). The major importance of laminin 5 for the formation of the hemidesmosomal adhesion complex and for the cohesion of the dermal-epidermal junction is deduced from the observation that in H-J EB an impaired expression of the protein correlates with abnormality in hemidesmosome structures and extensive skin blistering (Verrando et al., 1991). Herlitz J EB is also characterized by disadhesion of gastrointestinal and lung epithelia in which hemidesmosomal complexes have not been described (Jones et al., 1994). Therefore, the possibility exists that, in gut and basal membranes, laminin 5 incorporates the α3B chain to associate with morphological structures distinct from hemidesmosomes via the globular domains of the short arm of the α3B chain. This would be consistent with the detection of laminin α3B chain in brain tissues where laminin 5 may assume roles other than epithelial adhesion.

The intense expression of the α3B transcript in neuroectoderm and cerebellum confirms previous studies on expression of laminin 5 during organogenesis, suggesting a role in brain and nerve development (Aberdam et al., 1994). Laminin α1 chain harbors the peptide SIKVAV that mediates cell attachment, migration, and neurite outgrowth (Tashiro et al., 1989). Since in the mouse laminin α3 chain the peptide is not conserved, the sequence divergence within this area may reflect a functional difference in laminin α3 chain. The murine laminin α3A chain, however, is focally expressed in the developing trigeminal nerve, which strengthens the idea that this polypeptide may play a role in the migration and polarization of motor neurons. The laminin β2 chain, which directs the growing axons of intraspinal commissural neurons via the motor neuron-selective adhesive site (LRE), is expressed in the central nervous system (Sanes et al., 1990; Aberdam et al., 1994). Interestingly, the human and murine α3 chains contain LRE sites, which suggests that this isomamin may be physiologically active in the development of areas of the central nervous system. However, the possible function of laminin 5, or that of laminin isoforms comprising α3 chains, in the development of the central nervous system deserves further investigations and accurate clinical evaluation of J EB patients with mutations in the LAMA3 gene.

From our results, it seems likely that structural variants of the α3 chain may contribute to regulate diverse functions of laminin 5. Cloning of the cDNAs for the murine laminin α3 chains sets the stage for experiments on gene disruption in mice embryonic stem cells for the analysis of the specific role of the laminin α3A and α3B chain isoforms.

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Cloning and Differential Tissue Expression of Laminin α3 Chains

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