Identification of a Binding Motif for Ankyrin on the α-Subunit of Na⁺,K⁺-ATPase*

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Cytoskeleton membrane associations are important for a variety of cellular functions. The anion exchanger of erythrocytes (AE1) and Na⁺,K⁺-ATPase of polarized epithelial cells provide well studied examples of how integral membrane proteins are anchored via the linker molecule ankyrin to the spectrin-based membrane cytoskeleton. In the present study we have generated several recombinant fragments of the large (third) cytoplasmic domain (CD3) of Na⁺,K⁺-ATPase to define binding sites of ankyrin on CD3 at a molecular level. We provide evidence that a cluster of four amino acids, ALLK, is essential for binding of ankyrin to both recombinant CD3 and to native Na⁺,K⁺-ATPase. Once bound, conformational changes might uncover further binding sites for ankyrin on Na⁺,K⁺-ATPase. A motif related to the ALLK cluster is also present in the cytoplasmic domain of AE1 where this sequence (ALLK) turned out to be also important for ankyrin binding. These motifs are highly conserved during evolution of both Na⁺,K⁺-ATPase and AE1, further underlining their potential role in cytoskeleton to membrane linkage.

Cellular differentiation and several cellular functions depend to a large degree on the compartmentalization of particular membrane proteins such as receptors, adhesion molecules or ion translocating proteins to specialized domains of the cell surface (for review, see Refs. 1–3). One mechanism of how certain membrane proteins are placed at specialized sites of the plasma membrane could be by linkage of their cytoplasmic domains to the cytoskeleton. The HCO₃⁻, Cl⁻-exchanger of erythrocytes (anion exchanger 1, AE1)¹ and the sodium pump (Na⁺,K⁺-ATPase) of transporting epithelia provide two rather well studied examples of how ion-translocating integral membrane proteins are tethered via specific linker molecules to the fibrous scaffold of spectrin and actin that extends underneath the plasma membrane of virtually all cell types of the body (for review, see Refs. 1–3). Ankyrin is the main linker molecule that connects the cytoplasmic domains of both AE1 and Na⁺,K⁺-ATPase to β-spectrin of the membrane scaffold (4–7).

In transporting epithelia interaction of Na⁺,K⁺-ATPase and AE1 with the spectrin-based membrane scaffold is considered important for the polarized restriction of these transporters to either the apical or the basolateral cell surface (5, 8–12). Polarity of the Na⁺,K⁺-ATPase has profound implications for the direction of the transport of sodium and several other ions and molecules across the epithelial layer. In various transporting epithelia (such as kidney tubules, parotid gland, retinal pigment epithelium, choroid plexus, Mardin Darby canine kidney (MDCK) cell line) Na⁺,K⁺-ATPase is codistributed with ankyrin and can be copurified (communoprecipitated) with ankyrin and spectrin (5, 9–13). Binding of erythrocyte ankyrin to kidney Na⁺,K⁺-ATPase in vitro (4, 5, 14) and competitive inhibition of this interaction by addition of the cytoplasmic domain of AE1 (5, 13) suggests that there might be a common binding site on ankyrin involved in binding to both AE1 and Na⁺,K⁺-ATPase. Binding of ankyrin fragments to the α-subunit of Na⁺,K⁺-ATPase indicated the involvement of the AE1-binding domain of ankyrin and, in addition, a further domain of ankyrin not involved in AE1 binding (6). These observations are compatible with a recent report describing binding of erythrocyte and MDCK ankyrin to two of the five putative cytoplasmic domains of Na⁺,K⁺-ATPase, i.e. to cytoplasmic domains 2 and 3 (14).

The present study was performed to obtain more detailed information about the ankyrin binding sites on Na⁺,K⁺-ATPase, if possible at the amino acid level. We confined this study to the large cytoplasmic domain (CD3) because we found in a screening approach that a recombinant protein containing a portion of CD3 blocked binding of erythrocyte ankyrin to native kidney Na⁺,K⁺-ATPase. The most striking outcome of this study is that a motif of four amino acid residues (ALLK) appears to be essential for ankyrin binding. A similar motif (ALLKK) occurs in the sequence of the cytoplasmic domain of AE1 where it appears to participate also in ankyrin binding.

EXPERIMENTAL PROCEDURES

Generation of Various cDNAs for Fusion Protein Expression—Various portions of the nucleotide sequence encoding the large cytoplasmic domain (domain 3, CD3) of the α-subunit of rat Na⁺,K⁺-ATPase (15) were generated by polymerase chain reaction using full-length α-subunit cDNA kindly provided by R. Leveson (Yale University, New Haven, CT). Primer 1 (nucleotides 1417–1436) and primer 2 (nucleotides 2315–2332) generated a 915-nucleotide cDNA corresponding to amino acids 387–691 that cover ~90% of the sequence of the large cytoplasmic domain (CD3-1). Combination of primer 2 with either primer 3 (nucleotides 1600–1616) or primer 4 (nucleotides 1657–1673) resulted in 5’-truncated cDNAs corresponding to amino acid residues 447–691 (CD3-4) and 466–691 (CD3-5), respectively. Two additional cDNAs were obtained by exonuclease III truncation of the 5’ end of CD3-1 following the protocol of Henikoff (16). The resulting cDNAs corresponded to amino acid residues 410–691 (CD3-2) and 421–691 (CD3-3), respectively. Two further cDNAs corresponding to amino acid residues 514–691 (CD3-6) and 387–514 (CD3-7) were obtained by RI cleavage of CD3-1 cDNA cloned in vector pRSET A/B/C (Invitrogen, San Diego, CA). A clone covering a 1149-nucleotide fragment of human brain ankyrin (clone i307, nucleotides 5099–6247 (17) was kindly provided by Dr. V. Bennett and Dr. E. Otto.

¹ The abbreviations used are: AE1, anion exchanger 1; CD3, third cytoplasmic domain of Na⁺,K⁺-ATPase α-subunit; MDCK, Mardin Darby canine kidney; PAGE, polyacrylamide gel electrophoresis; RT, room temperature PBS, phosphate-buffered saline; HRP, horseradish peroxidase; BSA, bovine serum albumin; CD-AE1, cytoplasmic domain of AE1.

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Ankyrin Binding Motif on Na\(^+\),K\(^-\)-ATPase \(\alpha\)-Subunit

(Polymerase chain reaction amplification (20 cycles) was conducted under the following conditions: 0.9 pmol plasmid DNA, 0.4 pmol of each primer, 0.4 M of each dNTP, 1 unit of Taq polymerase (Boehringer Mannheim), 50 mM KCl, 1 mM MgCl\(_2\), adjusted to pH 8 (at room temperature, RT) by 10 mM Tris/HC1. Denaturation was performed at 94°C (1 min), followed by 30 cycles of 94°C (1 min), 30°C (1 min), 72°C (2 min). Site-directed Mutagenesis—Point mutation of CD3-7 at position 1632 (A → G) was performed to change the triplet AAG (that codes for lysine) to the triplet GAG that codes for glutamic acid (CD3-7*). Mutagenesis was carried out as described in detail elsewhere (19). For generation of the bacterially expressed urea-containing cDNA used by this method, Escherichia coli strain CJ236 deficient in UTnPase and ura3-N-glycosylase was transformed with CD3-7 DNA cloned in PRSET-A plasmids (20). Single-stranded plasmid DNA purified from this E. coli strain infected with bacteriophage M13KO7 (21) was used for site-directed mutagenesis.

Expression and Purification of Bacterial Fusion Proteins—All amplified cDNAs were subcloned in frame in a pRSET-A or pRSET-C vector (Invitrogen) and transformed in E. coli XL1 blue using the CaCl\(_2\) procedure (20). After checking all pRSET constructs by restriction mapping and DNA sequencing, expression and purification of fusion proteins was performed according to the manufacturer's manual (Invitrogen). Briefly, transformed bacteria were grown up to an OD of 0.3 at 37°C. Then isopropyl-\(\beta\)-D-thiogalactoside was added to a final concentration of 1 mM, and 1 h later the transcription and translation of the recombinant cDNAs was initiated by the addition of the bacteriophage T7/M13 at a multiplicity of infection of 5–10 plaque-forming units/cell. After further growth of 4–5 h, the bacteria of a 100-ml suspension were harvested by centrifugation at 4°C. The bacteria were then washed with PBS, pH 7.4, with 0.5 ml/min) equilibrated with urea buffer (8 M urea, 0.5 M NaCl, 20 mM sodium phosphate, pH 8). Contaminating bacterial proteins were removed by dialysis against solid polyethylene glycol 20,000 (Roth, Karlsruhe, FRG). A concentrated bacterial lysate was then applied to a 1.5-ml Eppendorf tube and centrifuged at RT for 8,000 g. The supernatant was loaded onto a Ni\(^{2+}\)-preloaded chelating Sepharose (Pharmacia, Uppsala, Sweden) column (12 × 25 cm; flow rate, 1.5 ml/min) and equilibrated with urea buffer (8 M urea, 0.5 M NaCl, 20 mM sodium phosphate, pH 8). Bacterial proteins were removed by washing the column with urea buffer at pH 8 and 6. Fusion proteins were eluted with urea buffer at pH 4, dialyzed at 4°C against 0.1 M EDTA, 10 mM Tris/HCl (pH 8), and concentrated by dialysis against solid polyethylene glycol 20,000 (Roth, Karlsruhe, FRG). A typical yield was 50–90 μg of fusion protein per ml of bacterial suspension.

Purity of the preparation was assayed by SDS-PAGE (10%). Removal of fusion peptides was performed by cleavage with enterokinase (23) that requires 10 mM CaCl\(_2\) for full activity. After dialysis of fusion proteins (concentration of up to 120 μg/ml) with 10 mM CaCl\(_2\) in 20 mM sodium phosphate, pH 8, enterokinase (Boehringer Mannheim) was added, and cleavage was allowed to proceed for 12–20 h at 37°C.

Purification of Ankyrin, Cytoplasmic Domain of AE1, and Na\(^+\),K\(^-\)-ATPase Vesicles—The cytoplasmic domain (CD3) of Na\(^+\),K\(^-\)-ATPase, its mutated counterpart (CD3-*), and the cytoplasmic domain of AE1 were purified from one unit of concentrated human erythrocyte AE1 (30) revealed a short common motif (DASESALLKCIEVCCG) with the sequences of rat, mouse, and human erythrocyte AE1 (30). The ankyrin binding motif on Na\(^+\),K\(^-\)-ATPase vesicles was performed to change the triplet AAG (that codes for lysine) to the triplet GAG that codes for glutamic acid (CD3-7*).

Localization and Electrophoretic Characterization of Recombinant Fragments of Na\(^+\),K\(^-\)-ATPase and AE1. To test the possibility that the Na\(^+\),K\(^-\)-ATPase is essential for ankyrin binding, Na\(^+\),K\(^-\)-ATPase vesicles were isolated by the rat brain synaptosomes (28).

RESULTS

Expression and Purification of Bacterial Fusion Proteins

They have been fused to the N-terminus of the E. coli codon. After expressing in M13KO7 at a multiplicity of infection of 5–10 plaque-forming units/colony, a 100-ml suspension was used for binding studies with Na\(^+\),K\(^-\)-ATPase vesicles as described above. Controls included all steps in the absence of Na\(^+\),K\(^-\)-ATPase vesicles.
show that the N-terminal third of cytoplasmic domain 3 of Na\textsuperscript{+},K\textsuperscript{+}-ATPase (\(\alpha\)-subunit) used for binding studies. Numbering of amino acid residues follows Herrera et al. (15). CD3-7\(^*\) carries a point mutation in which lysine 458 was exchanged against glutamic acid. SDS-PAGE of purified pig kidney Na\textsuperscript{+},K\textsuperscript{+}-ATPase and human erythrocyte ankyrin is also shown.

Fig. 1. Position and electrophoretic (SDS-PAGE) characterization of recombinant fragments of CD3 of rat Na\textsuperscript{+},K\textsuperscript{+}-ATPase (\(\alpha\)-subunit) used for binding studies. Numbering of amino acid residues follows Herrera et al. (15). CD3-7\(^*\) carries a point mutation in which lysine 458 was exchanged against glutamic acid. SDS-PAGE of purified pig kidney Na\textsuperscript{+},K\textsuperscript{+}-ATPase and human erythrocyte ankyrin is also shown.

Fig. 2. Dot blot assay demonstrating specific binding of biotinylated ankyrin (0.5 \(\mu\)g/ml) to 10 \(\mu\)g of immobilized CD3-1. (1) No significant binding of ankyrin is detectable to the following control proteins/peptides (10 \(\mu\)g per dot); (2) BSA; (3) recombinant fragment of human brain ankyrin; (4) soluble bacterial proteins; and (5) fusion peptides.

Fig. 3. Binding of biotinylated ankyrin to various recombinant fragments of CD3 (compare with Fig. 1). No binding occurs to CD3-4, CD3-5, and CD3-6. However, after removal of the fusion peptide by enterokinase cleavage (\(\Delta\)CD3-4, \(\Delta\)CD3-5) binding is seen to \(\Delta\)CD3-4 but not to \(\Delta\)CD3-5, indicating that the binding site for ankyrin on CD3 is located between amino acid residues 447 and 465.

Fig. 4. Dot blot assay to test binding of biotinylated ankyrin to mutated CD3-7 (CD3-7\(^*\)). In CD3-7\(^*\) lysine 458 was replaced by glutamic acid. Ankyrin binds to CD3-7, but not to CD3-7\(^*\), suggesting that lysine 458 is essential for binding.

Fig. 5. Cosedimentation of biotinylated ankyrin (1 \(\mu\)g/ml) with pig kidney Na\textsuperscript{+},K\textsuperscript{+}-ATPase vesicles (40 \(\mu\)g/ml) in the absence or presence of 18 \(\mu\)g/ml CD3-7 and CD3-7\(^*\), respectively. Pellets were dotted onto nitrocellulose filters and stained for biotinylated ankyrin. Binding (cosedimentation) of ankyrin to Na\textsuperscript{+},K\textsuperscript{+}-ATPase vesicles is inhibited by CD3-7, but not by the point mutated CD3-7\(^*\) variant, in which lysine 458 was exchanged against glutamic acid.

Ankyrin Binding Motif on Na\textsuperscript{+},K\textsuperscript{+}-ATPase \(\alpha\)-Subunit

Na\textsuperscript{+},K\textsuperscript{+}-ATPase. To determine whether the ALLLK cluster in the cytoplasmic domain of the erythrocyte AE1 is also essential for ankyrin binding, we extended binding studies to the purified cytoplasmic domain of AE1 (CD-AE1). Since the ALLLK peptide was too hydrophobic (hardly soluble), we used the extended AE1 sequence LRALLLKSH. Both peptides (ALLK and LRALLLKSH) inhibited binding of ankyrin to immobilized CD-AE1 and also to CD3-7 of Na\textsuperscript{+},K\textsuperscript{+}-ATPase. Nonsense and control peptides did not interfere with binding of ankyrin to either the CD-AE1 or CD3-7. This indicates that the ALL(L)K clusters on both Na\textsuperscript{+},K\textsuperscript{+}-ATPase and AE1 participate directly in ankyrin binding.
DISCUSSION

Interactions of integral membrane proteins with components of the cytoskeleton are probably important for diverse cellular functions, as for example for the assembly of specific plasma-membrane adhesion domains and for the generation or maintenance of cellular polarity (1–3, 31). The erythrocyte AE1 and kidney Na⁺,K⁺-ATPase provide well studied examples of such cytoskeleton-membrane associations. Both integral membrane proteins are linked via ankyrin to the spectrin-based membrane cytoskeleton. The main ankyrin binding site on AE1 appears to be located in the midportion of the cytoplasmic domain (7, 32). In Na⁺,K⁺-ATPase, cytoplasmic loops 2 and 3 (CD2, CD3) have been implicated in ankyrin binding (14).

In the present study we were able to identify a main binding site for ankyrin on CD3 of Na⁺,K⁺-ATPase. Generation of various CD3 fragments expressed by E. coli narrowed the protein binding site of ankyrin to a stretch of 19 amino acids (VAGDASESALLKCLIEVCCG). This stretch contains the only site of homology between Na⁺,K⁺-ATPase and the cytoplasmic domain of AE1, that consists of the cluster ALLK (Na⁺,K⁺-ATPase) or ALLLK (AE1) (5, 13). Several lines of evidence were provided indicating that the ALLK motif is essential for binding of ankyrin to Na⁺,K⁺-ATPase (Fig. 7): (a) no ankyrin binding was obtained with those CD3-fragments of Na⁺,K⁺-ATPase that lack the ALLK motif; (b) mutation of ALLK to ALLE in the CD3-7 fragment (CD3-7*) abolished binding of ankyrin; (c) the nonmutated CD3-7 fragment inhibited binding (cosementation) of ankyrin to native Na⁺,K⁺-ATPase, whereas the mutated analogue (CD3-7*) did not interfere with ankyrin binding; and (d) both the ALLK peptide of Na⁺,K⁺-ATPase and the ALLLK peptide of AE1 inhibited binding of ankyrin to CD3-7, whereas nonsense peptides (LAKL, WAGHRPTLGP) did not interfere with ankyrin binding.

Further support for the involvement of ALLK in ankyrin binding to Na⁺,K⁺-ATPase came from experiments in which CD3-4 was used for binding studies. In this CD3 portion the ALLK motif is located very close to the N terminus (VAGDASESALLK...). Binding of ankyrin to CD3-4 occurred only when the fusion peptide was removed by enterokinase cleavage (ACD3-4) (Fig. 3), whereas in all other CD3 fragments the fusion portion had no effect on ankyrin binding. The fusion peptide alone did also not interfere with ankyrin binding. The inability of the uncleaved CD3-4 fusion protein to bind ankyrin can be tentatively interpreted by steric hindrance caused by the close proximity between the ALLK motif and the polyhistidine containing fusion portion.

The ALLK/ALLLK motif turned out to be also essential for binding of ankyrin to the purified cytoplasmic domain of AE1 as indicated by inhibition of ankyrin binding to CD-AE1 in the presence of both ALLK and the ALLLK-containing peptide LRALLLKSH.

Taken together, these data indicate that the ALLK/ALLLK clusters of the cytoplasmic domains of Na⁺,K⁺-ATPase and AE1, respectively, are directly involved in ankyrin binding. This notion is further supported by sequence data showing that the ALLK motif of AE1 is an evolutionary highly conserved part in all vertebrates studied so far (fish, chicken, and all mammalian species) (30, 33–35). The same holds true for the ALLK motif of Na⁺,K⁺-ATPase which has been conserved without any change from Drosophila to humans (15, 36–40).

On the basis of these data it appears that the ALLK motif on Na⁺,K⁺-ATPase serves as a primary docking site for ankyrin. Once bound, conformational changes within CD3 might uncover further binding sites located on the second cytoplasmic domain of Na⁺,K⁺-ATPase (14). This model for ankyrin bind-
ing to Na\(^+\), K\(^-\)-ATPase is also compatible with the observation that ankyrin can bind to Na\(^+\), K\(^-\)-ATPase with both the AE1-binding and spectrin-binding fragments (6), and that two different portions on AE1 appear to crosstalk in ankyrin binding (32).

In view of the location of the ALLK motif in close proximity to the ATPase center of Na\(^+\), K\(^-\)-ATPase (41) it is tempting to speculate that such complex interactions between ankyrin and Na\(^+\), K\(^-\)-ATPase might be somehow regulated by the activity of the pump, in that the active pump might fully expose the ALLK motif thereby facilitating its attachment to the membrane cytoskeleton and its exposure at the cell surface. The inactive (dead) pump, on the other hand, might no longer expose the ALLK motif and hence might become prone to cytoskeleton detachment and removal from the cell surface. Such a mechanism might explain the low half-life of apically delivered inactive Na\(^+\), K\(^-\)-ATPase in MDCK cells (42).

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