Identification of a Binding Sequence for the 14-3-3 Protein within the Cytoplasmic Domain of the Adhesion Receptor, Platelet Glycoprotein Ibα*

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(Received for publication, November 2, 1995)

The 14-3-3 protein (14-3-3ζ) regulates protein kinases and interacts with several signaling molecules. We reported previously that a platelet adhesion receptor, glycoprotein (GP) Ib-IX, was associated with a 29-kDa protein with partial sequences identical to 14-3-3ζ. In this study, the interaction between GPIb-IX and recombinant 14-3-3ζ is reconstituted. Further, we show that the 14-3-3ζ binding site in GPIb is within a 15 residue sequence at the C terminus of GPIbα, as indicated by antibody inhibition and direct binding of 14-3-3ζ to synthetic 14-3-3ζ peptides. The 14-3-3ζ binds to recombinant wild type GPIb-IX but not to the GPIbα mutants lacking C-terminal 5 or more residues, suggesting that the C-terminal 5 residues of GPIbα are critical. Similarity between the GPIbα C-terminal sequence and the serine-rich regions of Raf and Bcr kinases suggests a possible serine-rich recognition motif for the 14-3-3 protein.

The platelet membrane glycoprotein Ib (GPIb)–glycoprotein IX (GPIX) complex (GPIb-IX) plays an important role in the initial platelet adhesion to injured vascular wall under high shear flow conditions such as in arteries and capillaries (1, 2). By binding to the subendothelium-bound von Willebrand factor, GPIb-IX not only mediates the physical adherence of platelets to the site of vascular injury but also initiates an activation signal that is transduced across the membrane resulting in a series of biochemical events. GPIb-induced intracellular biochemical changes include synthesis of thromboxane A2, hydrolysis of phosphoinositide, activation of protein kinase C and tyrosine kinases, elevation of cytoplasmic calcium level, cytoskeleton reorganization, and exposure of ligand-binding function of other adhesion receptors such as the integrin αIIbβ3 (3–8). In addition, GPIbα binds thrombin and is involved in the signaling process of thrombin-induced platelet activation (9–11). The mechanism of signal transduction via GPIb-IX has been unclear. In search for a possible intermediate between GPIb-IX and the intracellular signaling pathways, we have recently found that GPIb-IX is associated with a 29-kDa intracellular protein that is identical in partial amino acid sequence to the 14-3-3 protein (14-3-3ζ) (12).

The 14-3-3 proteins are a family of highly conserved eukaryotic proteins, which are distributed in a variety of cells (13, 14). Several functions have been attributed to the 14-3-3 proteins. At the cellular level, the 14-3-3 proteins have been implicated in the regulation of cell cycle and stimulation of exocytosis (15, 16). At the molecular level, the reported functions of 14-3-3 proteins include the activation of Pseudomonas aeruginosa exoenzyme S (14), phospholipase A2 activity (17), and regulation of protein kinase C and the tyrosine and tryptophan hydroxylases (18–22). The 14-3-3 proteins, including the 14-3-3ζ protein, bind several tyrosine kinases, suggesting a possible serine-rich recognition sequence and a short segment from each of the serine-rich 14-3-3 protein binding regions of Raf and Bcr kinases, suggesting a possible serine-rich recognition motif.

EXPERIMENTAL PROCEDURES

Cloning and Expression of the Recombinant 14-3-3 Protein—A cDNA fragment encoding the 14-3-3ζ protein was amplified by polymerase chain reaction from a cDNA library isolated from a human erythroleukemia cell line (kindly provided by Dr. Jerry Ware and Dr. Z. M. Ruggeri, The Scripps Research Institute, La Jolla, CA). The primers used for the amplification were as follows: TGTAGAATCTTCCACCATGGATAAAAATGAGCTGGTTC and ATGGTCTAGAAATGGTCTACTGGTTA. The cDNA fragments were first cloned into the pBluescript vector (Promega, Madison, WI) and sequenced. The pBluescript plasmid carrying the cDNA fragment was digested with EcoRI and XbaI, and subcloned into the pmal C2 vector (New England Biolabs, Beverly, MA) digested with the same enzymes. The construct was named pmal1433, and its insert matched the published sequence of 14-3-3ζ (17). The pmal1433 encodes a fusion protein with the N-terminal region corresponding to the Escherichia coli maltose-binding protein (MBP) and the C-terminal region corresponding to the entire 14-3-3ζ. Expression of the fusion protein in E. coli (DH5α) cells was performed as described previously (32). The expressed fusion protein has an molecular mass of...
cells were detached from flasks by incubating with 3.5 mM EDTA, 0.01 w/v washes three times with CGS buffer (0.12 M sodium chloride, 0.0129 M tri-sodium citrate, and 0.03 M glucose, pH 6.5) (38). Washed platelets were separated from whole blood by centrifugation and then resuspended in Hepes buffer (137 mM NaCl, 2.7 mM KCl, 1 mM MgCl₂, 5.6 mM glucose, 3.3 mM Na₂HPO₄, 3.8 mM Heps, pH 7.35) and immunoblotting.

Affinity Chromatography Using 14-3-3 Protein-conjugated Sepharose Column—Purified 14-3-3 protein or MBP were conjugated onto CNBr-activated Sepharose 4B (Pharmacia Biotech Inc.) (5 mg of protein/ml of Sepharose), respectively, according to manufacturer’s recommendation. Platelets were separated from whole blood by centrifugation and then washed three times with CSG buffer (0.12 M sodium chloride, 0.0129 M trisodium citrate, and 0.03 M glucose, pH 6.5) (38). Washed platelets were resuspended in Hepes buffer (137 mM NaCl, 2.7 mM KCl, 1 mM MgCl₂, 5.6 mM glucose, 3.3 mM Na₂HPO₄, 3.8 mM Heps, pH 7.35) and solubilized by adding an equal volume of the solubilization buffer (2% Triton X-100, 0.1 M Tris, 0.01 M EGTA, and 0.15 M NaCl, 1 mM dithiothreitol, pH 7.4) containing 0.2 M E64 (calpain inhibitor, Boehringer Mannheim) and 1 mM phenylmethylsulfonyl fluoride (12). The solubilized platelets were centrifuged at 100,000 × g for 30 min, and supernatants were loaded onto the 14-3-3 protein or control MBP column. After washing with 1% Triton X-100, 0.01 M Tris, 1 M EGTA, 0.15 M NaCl, and 1 M dithiothreitol, the bound proteins were eluted with a NaCl gradient from 0.15 M to 1 M. In some experiments, the bound proteins were eluted with 1 M NaCl in column buffer. The eluates were analyzed by SDS-PAGE, followed by silver staining and immunoblotting.

Blotting and Immunoprecipitations—Fresh human platelets (10⁷/ml) were solubilized as described above. CHO cells or melanoma cells were detached from flasks by incubating with 3.5 mM EDTA, 0.01 M Na₂HPO₄, 0.15 M NaCl, pH 7.4, washed twice and also solubilized in the solubilization buffer as described above. After centrifuging at 10,000 × g for 10 min, the lysates (200 μl) were incubated with 25 μl (50% v/v) of MBP-conjugated control beads or the 14-3-3 protein-conjugated MBP-Sepharose 4B beads at 4°C for 2 h. The beads were then washed three times in solubilization buffer by centrifugation. Bound proteins were extracted by adding SDS-PAGE sample buffer (0.125 M Tris, pH 6.8, 20% (v/v) glycerol, 0.004% (w/v) bromphenol blue, 4% (w/v) SDS), and analyzed by SDS-PAGE followed by Western blot with antibodies.

Binding of the 125I-labeled 14-3-3ζ to Synthetic Peptides—The recombinant 14-3-3ζ protein was 125I-labeled using IODOBEADS (Pierce). The synthetic peptides were solubilized in distilled water and then diluted to 50 μg/ml in 0.1 M NaHCO₃, pH 9.2. The peptides were coated onto the microtiter wells (Immunon II Removawells, Dynatech Laboratories, Chantilly, VA) by incubation at 22°C for 6 h and then 4°C overnight. The peptide coated microtiter wells were incubated at 22°C

2 J. Cunningham, personal communication.

![Figure 1](image-url)
through the 14-3-3\textsubscript{C}-conjugated column or the control column. Bound proteins were eluted with 1 M NaCl and analyzed by SDS-PAGE, followed by immunoblotting with an antibody against the C-terminal region of GPIb\textsubscript{x} cytoplasmic domain (anti-Ib\textsubscript{C}).

B. Washed platelets were solubilized by treatment with 1 M NaCl and analyzed by SDS-PAGE, followed by immunoblotting with an antibody against the C-terminal region of GPIb\textsubscript{x} cytoplasmic domain (anti-Ib\textsubscript{C}).

C. The 20-kDa C-terminal fragment of GPIb\textsubscript{x} was detected in the eluates from the 14-3-3\textsubscript{C}-conjugated column or the control column. This indicates that the C-terminal globular domain of GPIb-IX binds to 14-3-3\textsubscript{C}. Since the C-terminal domain of GPIb-IX does not bind to ABP in lysates in which calpain is active (40), this result also indicates that 14-3-3\textsubscript{C} interaction with GPIb is not mediated by ABP. This conclusion was further supported in the experiment in which recombinant GPIb-IX expressed in an ABP-deficient cell line (34) was solubilized and incubated with the 14-3-3\textsubscript{C}-conjugated Sepharose beads or control MBP-conjugated beads. GPIb-IX bound to the 14-3-3\textsubscript{C} beads even though ABP was absent (data not shown).

GPIb\textsubscript{x} Cytoplasmic Domain Contains the 14-3-3\textsubscript{C} Binding Site—To further characterize the binding site for 14-3-3\textsubscript{C} on GPIb-IX, the platelet lysates were preincubated with either a control rabbit serum, an anti-peptide antibody directed against the C-terminal 15 residues of GPIb\textsubscript{x} (anti-Ib\textsubscript{C}), or an anti-peptide antibody against the cytoplasmic domain of GPIb\textsubscript{b} (anti-Ib\textsubscript{C}). The platelet lysates were then incubated with Sepharose beads conjugated with 14-3-3\textsubscript{C}. As shown in Fig. 3, GPIb-IX binds to the 14-3-3\textsubscript{C} but not MBP-conjugated beads. Pretreatment with control serum had no effect on the binding of GPIb-IX.

With anti-Ib\textsubscript{C}, the binding of GPIb-IX to 14-3-3\textsubscript{C}-conjugated beads was dramatically inhibited. In contrast, preincubation with anti-GPIb\textsubscript{b} antibody had no inhibitory effect. The different effects between the anti-GPIb\textsubscript{b} and anti-GPIb\textsubscript{c} cytoplasmic domain antibodies in inhibiting GPIb-IX binding to 14-3-3\textsubscript{C} is not due to a difference in the antibody binding to GPIb-IX, because equivalent amounts of GPIb-IX were immunoprecipitated by these two antibodies (Fig. 3B). These data suggest that the C-terminal 15 residues of GPIb\textsubscript{x} is critical for the 14-3-3\textsubscript{C} binding.

The C-terminal 15 Residues of GPIb\textsubscript{x} Contains the 14-3-3\textsubscript{C} Binding Site—To verify whether the C-terminal 15 residues of GPIb\textsubscript{x} recognized by the inhibitory antibody contains the 14-3-3\textsubscript{C} binding site, C\textsubscript{125I}-labeled 14-3-3\textsubscript{C} was incubated in microtiter wells coated with the synthetic peptides corresponding to the C-terminal 15 residues of GPIb\textsubscript{x} (Ib\textsubscript{C}) or C-terminal 15 residues of GPIb\textsubscript{b} (Ib\textsubscript{C}). The 14-3-3\textsubscript{C} bound to the Ib\textsubscript{C} peptide in a saturable manner, with an estimated K\textsubscript{d} of 850 nM (Fig. 4). In contrast, there was no specific binding to the Ib\textsubscript{C} peptide (Fig. 4) or a control peptide corresponding to C-terminal 15 residues of the integrin \( \beta_1 \) subunit (not shown). To exclude the possibility of nonspecific binding, a truncated form of the 14-3-3\textsubscript{C} (1433T3, truncated at residue 36) fused with E. coli MBP was shown not to bind to the Ib\textsubscript{C}-coated wells (Fig. 4). In addition, binding of the 14-3-3\textsubscript{C} to Ib\textsubscript{C} peptide was inhibitable by the unlabeled 14-3-3 protein and by the anti-Ib\textsubscript{C} antibody (data not shown). Thus, the C-terminal 15-residue sequence of GPIb\textsubscript{x} (Asp\textsubscript{596}–Leu\textsubscript{610}) contains the 14-3-3\textsubscript{C} binding site.

The C-terminal 5 Residues of GPIb\textsubscript{x} Cytoplasmic Domain Are Critical for the Binding of 14-3-3\textsubscript{C}—To further examine the region of GPIb\textsubscript{x} cytoplasmic domain involved in the binding of 14-3-3\textsubscript{C}, recombinant wild type and mutant GPIb-IX expressed in the CHO cells were used. The mutants incorporated stop codons at the C-terminal side of the residues 599 (\( \Delta 559 \)), 591 (\( \Delta 591 \)), and 605 (\( \Delta 605 \)) of GPIb\textsubscript{x} (Fig. 5C). GPIb-IX from \( \Delta 559 \) cell line does not bind ABP, while GPIb\textsubscript{x}\( \Delta 605 \) only lacks 5 residues from the C terminus of the wild type GPIb\textsubscript{x} and is still able to bind ABP. The CHO cells expressing recombinant wild type GPIb-IX or GPIb-IX mutants were solubilized and then incubated with the 14-3-3\textsubscript{C}-conjugated Sepharose beads or the
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**Discussion**

In this study, we have reconstituted the binding of the 14-3-3 protein to an important platelet adhesion receptor, GPIb-IX, and identified the 14-3-3 binding site at the C terminus of GPIbα cytoplasmic domain. The identification of a short peptide sequence that binds the 14-3-3 protein provides insight into the structural basis required for the 14-3-3 protein recognition. The similarity between the 14-3-3 protein binding sequence in GPIbα and the segments from the 14-3-3 protein binding region of the Bcr and Raf kinases suggests a possible serine-rich recognition motif in the ligands of the 14-3-3 protein. In addition, characterization of the interaction of the 14-3-3 protein with the membrane receptor may help to understand the roles of the 14-3-3 protein in the receptor-mediated signaling pathways.

Reconstitution of the binding between the recombinant 14-3-3 and GPIb-IX (Fig. 1) confirmed the identity of the previously reported GPIb-IX-associated 29-kDa protein (12) as 14-3-3. In platelets, 14-3-3 is first purified and cloned as a platelet intracellular phospholipase A2 (17, 43), although this function of 14-3-3 was recently disputed by other groups (44). The 14-3-3 protein is relatively abundant in platelets and has been shown to be both associated with the plasma membrane and present in the cytosol (43). Purified 14-3-3, however, does not bind to phospholipid vesicles (45). Thus, association with the cytoplasmic domain of GPIb-IX may account for at least part of the membrane-associated fraction of this protein.

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**Fig. 4.** Binding of 125I-labeled 14-3-3 to synthetic peptides. Microtiter wells were coated with synthetic peptides DLLSTYSIRYSGHSL corresponding to C-terminal 15 residues of GPIbα (IbαC) or TDPLVAERAGTDES corresponding to C-terminal 14 residues of GPIbα (IbαCl) (26, 27). Various concentrations of 125I-labeled recombinant 14-3-3 protein or a truncated 14-3-3-MBP fusion protein (1433T3, as negative control) were added to the microtiter wells and incubated at 22°C for 2 h. Bound proteins were estimated by γ-counting. Data shown are the mean value of triplicate samples ± standard deviation. Closed circles, 14-3-3 binding to IbαC peptide; open circles, 14-3-3 binding to IbαCl peptide; open square, 1433T3 binding to IbαC peptide.

**Fig. 5.** Binding of 14-3-3 to the recombinant wild type and mutant GPIb-IX. A, wild type GPIb-IX and GPIb-IX mutants ∆559 expressed in CHO cells were solubilized and 250 μl of lysates were incubated in the absence or in the presence of 50 μl rabbit anti-GPIb-IX monoclonal antibody, WM23. Cell lysates (Lysate) expressing wild type GPIb-IX, ∆559, or CHO cells were also directly analyzed by SDS-PAGE and Western blotting with WM23. B, the cell lysates (Lysate) expressing wild type GPIb-IX, or ∆591 and ∆605 GPIb-IX mutants were solubilized and incubated with control beads (MBP) or the 14-3-3-conjugated beads (14-3-3) at 4°C for 1 h. Bound proteins were solubilized in SDS-PAGE sample buffer and analyzed by SDS-PAGE, followed by Western blot with an anti-GPIb monoclonal antibody, WM23. Cell lysates (Lysate) expressing wild type GPIb-IX, or ∆559, or CHO cells were also directly analyzed by SDS-PAGE and Western blotting with WM23 to visualize the quantity of GPIb-IX in each of these cell lines. C, a schematic of the cytoplasmic domain of GPIbα indicating locations of the C-terminal ends of the truncated GPIbα mutants and the 14-3-3 binding site.
Thus, if this sequence were to form an
serine every 3–4 residues (Fig. 6). Indeed, from the recently resolved structure
of the 14-3-3 protein, a ligand that fits into the ligand binding
groove of the 14-3-3 protein was predicted to be an amphipathic
helix (46, 47). Furthermore, the serine-rich regions in Bcr and Raf kinases appear to be critical for the 14-3-3 protein binding, and serine residues from a 30-residue segment from each of the serine-rich regions of Bcr and Raf kinases are well aligned with serine residues from the 14-3-3 binding sequence of GPIbα (Fig. 6). This suggests that clusters of serine residues in a helical structure may be a common recognition motif important for the binding of the 14-3-3 protein.

The location of the 14-3-3 binding site on GPIbα is likely to occur in intact platelets, and thus may be of relevance to the functions of the cytoplasmic domain of GPIbα. The cytoplasmic domain of GPIbα is known to bind to the cytoskeletal protein ABP (48). ABP, however, is not required for the binding of the 14-3-3 protein, as co-immunoprecipitation of the 14-3-3 with GPIbα was not inhibited by the treatment of cell lysates with DNase I and N-ethylmaleimide, which disrupted the interaction between GPIbα and ABP (12). Furthermore, in the present study, we show that lysis of platelets under conditions in which calpain was active and the ABP-GPIbα interaction disrupted, did not prevent the interaction of the C-terminal domain of GPIbα with 14-3-3 (Fig. 2). Moreover, 14-3-3 bound to GPIbα from an ABP-deficient
cell line (not shown). Conversely, the 14-3-3 protein is not required for the ABP-GPIbα interaction, as the GPIbα mutant that was truncated at residue 605 retained its capacity to interact with ABP, yet lost its 14-3-3 binding capacity. The GPIbα binding site is located in the central region (Thr436, Phe568) of the GPIbα cytoplasmic domain (48), while the C-terminal region contains the 14-3-3 binding site (Fig. 4). Binding of ABP to the cytoplasmic domain of GPIbα links GPIbα to the membrane skeleton framework (40). It is interesting to speculate that the adjacent location of the binding sites of the membrane skeleton protein, ABP, and a kinase regulator, 14-
3-3, within the GPIbα cytoplasmic domain may be important in the shear-dependent signaling transduction through GPIbα. For example, it is possible that mechanical force generated by immobilized von Willebrand factor binding to N-terminal domain of GPIbα under high shear stress may change the conformation of the C terminus of GPIbα by leverage of the membrane skeleton and thus regulate the binding or signaling functions of the 14-3-3.

It has been unclear how the 14-3-3 proteins regulate their target proteins. A recent report suggests that by binding to Raf kinase, the 14-3-3 protein may prevent its inactivation by protein phosphatase (49). It is possible that 14-3-3 protein may prevent the inactivation of Raf by interaction with phosphorylated serine residues in the serine-clustered region. Similarly, it is possible that by binding to the C-terminal serine-rich region of the cytoplasmic domain of GPIbα, the 14-3-3 protein may regulate the function of the GPIbα cytoplasmic domain by preventing serine (or phosphoserine) residues from being modified. Simi-
larly between the GPIbα C-terminal region and a segment from the 14-3-3 protein binding region of both Raf and Bcr also suggests a possibility that GPIbα may compete with kinases for the 14-3-3 protein binding sites in the membrane compartment and thus regulate kinase activity. Alternatively, as the membrane translocation is a mechanism of the Raf and protein kinase C activation, it is also possible that the dimeric 14-3-3 may be involved in the translocation of the protein kinases to the membrane (50). Although phospholipid association of some isoforms of the 14-3-3 protein family has been indicated, 14-
3-3 does not appear to associate with phospholipid vesicles (45). Thus, a possible mechanism is that by binding to the cytoplasmic domain of GPIbα, the 14-3-3 protein may mediate trans-
location of the protein kinases to cytoplasmic face of the membrane and to the cytoplasmic domain of this membrane receptor in order to relay signals. If this were to be the case, there may also be membrane receptors in other cell types that inter-
act with the 14-3-3 proteins.

Acknowledgments—We thank Dr. Mark Ginsberg for valuable discussions and help, Dr. Michael C. Berndt and Dr. Zaverio Ruggeri for providing monoclonal antibodies, Dr. Jerry Ware for providing the cDNA library, and Dr. Janet Cunningham and Dr. Sylvie Meyer for providing GPIbα-transfected cells.

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Xiaoping Du, Joan E. Fox and Susan Pei

J. Biol. Chem. 1996, 271:7362-7367.
doi: 10.1074/jbc.271.13.7362

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