# sentences with keywords

1. These observations offered a functionally testable model describing how did BRWD1 bind to DNA first and then recruit cohesin via SMC1 /SMC3 and /or form a dynamic active cohesin complex via NIPBL that was absolutely important for loop extrusion and long range genomic interactions (Supplementary Fig

2. For example, vav, a 95 kDa CH-containing GDP/GTP exchange proto-oncogene product expressed exclusively in hematopoietic cells was found to bind to Heterogeneous Nuclear Ribonucleoprotein (hnRNP) C through a region in the C-terminal domain of the protein

3. J Biol Chem 274:32904–32908ArticlePubMedCAS Google ScholarMcMahon LW, Sangerman J, Goodman SR, Kumaresan K, Lambert MW (2001) Human alpha spectrin II and the FANCA, FANCC, and FANCG proteins bind to DNA containing psoralen interstrand cross-links

4. It has been reported that Blt1 can directly bind to membrane lipids (Guzman-Vendrell et al

5. Tightly Bound Divalent CationSeveral molecules of mono- and divalent cations bind to actin at low affinity sites (Kd = 0

6. High affinity Ca2+ and Mg2+ bind to ATP-actin with low nanomolar and low micromolar affinities respectively,17 and the divalent cation dissociation defines an overall rate of nucleotide release from actin

7. Becausecross-linking proteins must bind to two filaments to form a link, wehypothesized that a high avidity might be dominating the behavior suchthat the multiple binding sites prevented complete dissociation

8. WH2 domains are shortdomains (<50 amino acids) that bind to monomeric actin and have arange of attributed roles including actin filament nucleation (130, 266)

9. Full-length Fim1, FimA12, FimA2, and FimEFA1 all bind to and sediment with F-actin, although with different affinities

10. Interestingly, heat shock protein HSP90 can bind to N-WASP and bundle branched filaments but does notinhibit polymerization by Arp2/3 complex and N-WASP (72)

11. It has a sequence that appears to bind to domain 3, similar to theD-loop in actin

12. ACs bind to actinADP monomer at physiological ionic strength with Kds = 25–200 nM (Carlier et al 1997),

13. The bundling behavior of AlfA in vitro suggests that new filaments may bind to an AlfB/parN complex and grow along an existing AlfA bundle until they reach the poles (15, 136)

14. ACs bind to actinADP monomer at physiological ionic strength with Kds = 25–200 nM (Carlier et al 1997),

15. The bundling behavior of AlfA in vitro suggests that new filaments may bind to an AlfB/parN complex and grow along an existing AlfA bundle until they reach the poles (15, 136)

16. Arps have the same fold as actin, includingall of the atoms required to bind to ATP (Robinson et al

17. The drawing does not include tropomyosin and myosin motors,which bind to the sides of filaments

18. However, high concentrations of cofilin sever transiently as the firstfew cofilins bind to a bare filament or if other proteins compete withcofilin for binding

19. A simple explanation is that the complex of the FH2 domainand the end of the filament has two conformations: Actin monomers canbind to the open state but not the closed state (Vavylonis et al

20. As discussed above, members of the gelsolin family are calcium-regulated proteins that not onlysever filaments but also bind to barbed ends (Nag et al

21. Nodes bind to and dissociate from the plasma membrane

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23. 9 μMRng2CHD) plots were measured in the presence of Rng2CHD concentrationsthat were expected to bind to actin protomers at binding ratios whichcaused a 50%, 82%, and 96% reduction in actomyosin movement speed onmuscle HMM, respectively, in in vitro motility assays

24. We speculated that, in order for the detectable fluorescent clusters toform, HMM needs to bind to actin filaments repetitively and transiently, and the dwell time of each binding event must be long enough to allowvisualization

25. 5 μM ATP) were introduced into the flowchamber to loosely bind to the positively charged lipid layer

26. Septins themselves can bind to membranes andself-assemble into filamentous scaffolds

27. motors, which bind to actin cytoskeletalfilaments and use chem

28. elements synergistically bind to RhoA andphospholipids to anchor anillin at the cleavage furrow

29. How formins simply bind to filamentbarbed ends in rapid equil

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33. We believe they may be useful for interpreting mutations leading to constitutive catalytic activity in cancer as well as for templates for modeling substrate and inhibitor binding for molecules which bind to the active state

34. These results suggest that binding sites for Myo3 on the ring are available after the completed assembly of the ring, and diffused Myo3 molecules directly bind to the sites rather than migrating towards the ring through ring-associated actin tracks (Arai and Mabuchi, 2002; Huang et al

35. However, it is unclear if they can simultaneously bind to the membrane in a way that allows dimerization or oligomerization of Mid1, and if one domain plays a dominant role

36. Purified Mid1 PH have been shown to bind to negatively charged phosphatidylinositol (PI) lipids, including PIP2 that is present in higher concentration towards the medial region of fission yeast [26]

37. Given the geometry of the dimer in the crystal structure, it is difficult to imagine how the PH domains would bind to the membrane cooperatively with each other or the L3 loops, leading to the hypothesis that the Mid1 dimer membrane binding mode only involves the C2 domain/L3 loops [9]

38. Mid1 L3 loop and PH domain can independently bind to the membraneWe began by examining if the candidate membrane binding domains can bind a lipid bilayer with a composition similar to that of fission yeast in isolation

39. Download figureOpen in new tabFigure 2:L3 loop & PH domain can independently bind to the membrane

40. Mid1 molecules bind to the plasma membrane as oligomers to form a platform for other proteins to bind (Celton-Morizur et al

41. The interactions between modules I and II and their downstream proteinsThe two modules for node assembly can cooperate with each other for successful assembly of the contractile ring in at least four ways: (1) Cdc4 and Rng2 provide a positive feedback for Mid1 recruitment, and Mid1 in turn is essential for the assembly of both modules; (2) both modules are involved in recruiting Cdc12 to nodes; (3) actin filaments nucleated by Cdc12 can recruit and/or bind to Rng2 (Takaine et al

42. Because Cdc15 can bind to the plasma membrane (Takeda et al

43. Actin crosslinkers bind to the sides of actin filamentsto aid in the formation of filament bundles

44. The CC domain has been demonstrated to bind to Ezrin, acytoskeleton-plasma membrane anchor, and the GRD domain transientlyprotects the Cdc42 small GTPase in its activated state by reducing GTPhydrolysis

45. Intriguingly, both fly and human Septins decorated actin filament bundles and rings without forming full rings themselves in vitro72, suggesting that Septins are not acting as a template for actinfilaments to form a ring, but are instead bending actin filaments asthey bind to the side of the filaments

46. Surprisingly, recent studies showed that bipolar Myosin II can also bind to a single actin filament75,98,99

47. Their conserved N-terminal motor domains bind to actin and theirclass-specific C-terminal domains regulate the selection of and bindingto cargos or other cell components94,100–105

48. In the presence of Ca2+, the EF-hands of plastin share a substantial similarity with CaM andbind to a switch helix (homologous to canonical CaM-binding motifs),which is located in the linker segment connecting the regulatory andcore domains (CBM in Figure 3, [171,173])

49. Several lines of evidence suggest that Blt1may directly bind to lipids: (i) nonsaturable binding of Blt1 to thecortex upon overexpression; (ii) in vitro binding of Blt1-GFP but not truncated blt1Δ5-GFP to lipids in cell extracts; (iii) the presence of C-terminal basic-rich motifs with the potential to mediate the electrostatic interaction with acidic phospholipids, such as phosphatidylinositol 4,5-biphosphate[PI(4,5)P2], enriched at the plasma membrane

50. All of the amino terminal CH domains so far analysed have the intrinsic ability to bind to actin albeit with lower affinity than the complete ABD ([17, 18]; S

51. Hence, the general assumption that single CH domain-containing proteins bind to actin via their CH domain and that a single CH domain is sufficient for F-actin binding, is inadequate

52. As expected,EF1-4 was not able to bind to the ABD–SR1–NEECK construct, supportingthe specificity of the EF3-4–neck hydrophobic interaction (SI Appendix, Fig

53. Therefore, Rng2 might bephosphorylated from metaphase by CDK Cdc2 and becomes active inassembling the CR, whereas its actin-binding activity is weakened andthen dephosphorylated after anaphase to bind to and stabilize CR F-actin

54. Many PH domains bind to lipids directly

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56. The different proteins that bind to actin filaments influence how quickly actin filaments are assembled and organized intonetworks

57. The experiments show thatsome actin binding proteins, like tropomyosin, cooperate to bind toactin

58. In addition, the poor ability of individual Cdc8 molecules to bind toan actin filament makes it unlikely to be able to displace other ABPsonce they’re bound to an actin filament, resulting in Cdc8's completeexclusion from certain F-actin networks, such as actin patches

59. In vitro high-speedsedimentation assays and preliminary TIRFM assays at high Cdc8concentrations determined that the I76C and D142C Cdc8 mutants behavedclosest to wild-type Cdc8 in ability to bind to F-actin (Figure 1—figure supplement 1B–D)

60. As two distinct Cdc8 cables can bind to an actin filament (oneon the surface of each groove of the helical actin filament), werepresented the actin filament as a lattice with two rows representingthe two actin surfaces potentially bound by Cdc8

61. An ideal experiment would enhance the ability of Cdc8 to bind to F-actin while inhibiting its end-to-endbinding

62. 007 Less dynamic α-actinin Ain1 associates with actin patchesUltrastructural and mutational studies offimbrin/plastin and α-actinin from several organisms demonstrate thatthey bind to a similar site on F-actin (Galkin et al

63. Although fimbrin/plastin and α-actinin isoforms bind to a similar siteon F-actin, Cdc8 and α-actinin Ain1 both associate with the contractilering

64. Although these proteins all bind to the side of actin filaments, theyare differentially utilized for specific cellular processes

65. In addition to Fim1p, Cdc8p, and Adf1p, fission yeast containsmany other proteins that bind to the side of actin filaments andlocalize to specific cellular structures, including Arp2/3 complex,myosin, α-actinin, transgelin Stg1p, IQGAP Rng2p, coronin, and Aip1

66. Here, we demonstrate using high-speed cosedimentation assays thatCLIP-170 can bind to filamentous actin (F-actin) directly

67. Furthermore, local actinfilament deformation enhances the assembly of myosin-II molecules intobipolar filaments able to bind to multiple actin filaments, therebyeffectively increasing the myosin-II duty ratio (29,30)

68. Recent studies have shown, for example, that crosslinking proteins such as fascin, which bind to polar, parallel F-actin, do not supportcontraction to the same extent as other crosslinking proteins, such ascortexillin and fimbrin, that bind to filaments in apolarity-independent fashion72

69. The Structure and Assembly of Myo2 and Myp2Myo2 and Myp2 share the same basic domains as myosin-IIs in other organisms: a head domain harboring ATPase activity and anactin-binding site, two IQ motifs that bind to ELC and RLC, and a taildomain that is made of CCs, with a NHR in the middle of Myp2 tail (Figure 1; Bezanilla et al

70. The type 1 CH domain (CH1) has the intrinsic ability to bind toF-actin

71. By constructing calponin without C-terminal tandemrepeats, the resulting protein with the CH domain failed to bind toactin (Gimona and Mital, 1998), suggesting that a single CH domain is neither sufficient nor necessary for the binding of F-actin (Gimona and Mital, 1998)

72. Binding With Signaling ProteinsBesides its ability to bind to actin and tubulin, the CH domain can participate in signal transduction by binding to differentprotein partners such as extracellular regulated kinase (ERK) andcalmodulin (Figure 1A)

73. Does vav bind to f-actin through a CH domain? FEBS Lett

74. Upon phosphorylation by Pom1, which localizes at cell ends, Cdr2 cannot bind to the membrane at these sites, leading to node formation in thecell middle

75. The Ena/VASP family proteins enhance actin nucleation using tandem WH2 (WASP homology 2) motifs that bind to actin [13]

76. Activated NPFs bind to both G-actin and the Arp2/3 complex to initiate the binding of Arp2/3 to the preexisting F-actin and nucleation [60,61]

77. This multidomain protein is able to bind to negatively charged phospholipids (PIP2 and phosphatidylserine) through a basic domain located in theN-terminus of the protein, which mediates its recruitment at the plasmamembrane, and an additional membrane-binding site in the C-terminalregion has been shown to inhibit mDia1 F-actin polymerization activity (Ramalingam et al

78. Moreover, Fim1A1 did not bind toF-actin

79. In fission yeast, ABD2 maybind tightly to F-actin, whereas the cross-linking activity of Fim1 maybe controlled by ABD1: Fim1 may contribute to formation of the 3-D actin cytoskeleton when ABD1 is somehow activated to bind to F-actin

80. Later in the drug development process, medicinal chemistsoptimize ligands on the basis of structure–activity relationships bysynthesizing different ligands that share a core chemical scaffold andare assumed to bind to their target in a similar fashion75

81. Together with the tethering PH domain, three membrane-associatingelements synergistically bind to RhoA and phospholipids to anchoranillin at the cleavage furrow

82. Mid1 doesnot bind to the PC/PE liposome and it shows a modest affinity toward PS(Kd ~1 µM, Figure 3D)

83. Thus, the structureof anillin allows all these elements: the C-terminal tail of RhoA, theL3 loop of the C2 domain and the PH domain, to orient toward andsynergistically bind to the plasma membrane (Figure 7A)

84. ABPsutilize their calponin-homology (CH) domains to bind to F-actin

85. Therefore, we investigated the importance of positivelycharged residues in the L2 region because they bind to F-actin (Figure 6), which was also confirmed in fission yeast by the mutants of Ain1 (Figure 7)

86. Thusutrophin could possibly bind to actin in an extended conformation sothat the sites previously identified as being important for actinbinding may be directly involved in this interaction

87. Both domain constructs exist in solution as compact monomers and bind to actin as 1:1 complexes

88. Thusutrophin could possibly bind to actin in an extended conformation sothat the sites previously identified as being important for actinbinding may be directly involved in this interaction

89. Both domain constructs exist in solution as compact monomers and bind to actin as 1:1 complexes

90. A PH (pleckstrin homology) domain and a region of basic residues allowEct-2 to bind to the plasma membrane, an interaction required toactivate Rho (35)

91. Mechanism of action of these toxins varies with respectto the site where they bind to actin

92. It has been demonstrated that the CaM domain can bind to this linker and its binding is relieved by PIP2binding to the ABD, freeing the CaM domain for interaction with thetitin Z-repeats

93. Ca2+ ions bind to two EF-hands in the RD of the protein, whose locationrelative to the actin-binding core remains unknown

94. Plastinscan bind to F-actin via their ABD1 domain independent of bundling27 and ABD1 alone is sufficient to positively affect Arp2/3-mediated actin dynamics in vitro

95. In the presence of Ca2+, plastins bind to actin via ABD1 only,27 while CH3–CH4 are locked in the inhibited state by RD, which restrictsthe rearrangements required for the CH3 binding to actin (Fig

96. b In the presence of Ca2+, plastins bind to actin via ABD1 only, while CH3–CH4 of ABD2 are lockedin the inhibited state (red dashed) by stapling ABD2 to ABD1 by RD

97. The allostery lies in the fact that, if IQGAP2 binds to the primary binding site,then IQGAP1 cannot bind to the secondary binding site

98. Our simulations showed that, as observed experimentally, CKsprimarily assemble in the cytoplasm and, after assembly, they can moveand bind to the membrane

99. The observation that α-actinins and fimbrins bind to the same region of actin (Matsudaira, 1994b) suggests that they may have related but complementary functions

100. Our results demonstrate thatfull-length human IQGAP1 forms dimers that stably bind to actin filament sides and transiently cap barbed ends

101. Full-length IQGAP1 and its N-terminal half tightly bind to actin filament sidesTo define the kinetics of IQGAP1 interactions with actin filaments, wepurified and fluorescently labeled SNAP-tagged full-length IQGAP1(649-SNAP-IQGAP1)

102. However, it did not bind tofilament sides (Supplemental Figure S4A), and it failed to suppressbarbed end growth (Supplemental Figure S4B), suggesting that the SNAPtag may interfere with actin binding

103. Several of these ligands bind to a C-terminal domain in IQGAP1,suggesting that their activities may be coordinated with the transientcapping activity of IQGAP1 to control actin assembly (Figure 6)

104. Affinity of tandem CH1–CH2 domains for F-actin is known to ariseprimarily from the CH1 domain, as CH2 alone cannot bind to actin (Singh et al

105. IQGAPs have been proposed to bind to F-actin via the CH domain and thus connect the actin cytoskeleton to cell-signalling pathways [8], [9]

106. The cytoskeletal proteins that contain CH domains, are thought to bind tothe same site on F-actin

107. In a higher resolution study, the actin-binding region of fimbrin has been observed to bind to the same region [16] inducing a relative movement of actin subdomains

108. It is less clear whether proteins that contain only a single CH domainbind to actin via this domain

109. We have foundthat in contrast to the complete actin-binding region, the isolated sCH2 domain does not bind to F-actin with a significant affinity (Figure 7a)

110. Conversely, a previous study on α-actinin showed that a construct corresponding to the CH1 domain does bind to F-actin [22]

111. Regardingits function, little is known about how the proteins harbouring a single CH domain bind to actin — if they do at all

112. A 538-residue fragment of IQGAP that includes the CH domain is able to bind to F-actin [23]

113. It has been proposed that IQGAP would bind to actin as a dimer [23]

114. Alternatively spliced isoforms of proteins, which bind to intermediatefilaments, are known to exist

115. The actin-binding region of the proteins that bind to intermediatefilaments (dystonin, ACF7 and plectin) is most closely related to thecorresponding region in proteins of the spectrin family

116. Experimental evidence suggests that the CH1 domain has a key role in the interaction with actin filaments, whereas the CH2 domain enhances thebinding affinity but it alone does not bind to F-actin

117. SarasteDoes vav bind to F-actin through a CH domain?FEBS Lett, 374 (1995), pp

118. Mitotic Spindle Positioning (MISP) is an actin bundler that selectively stabilizes the rootlets of epithelial microvilli2022, Cell ReportsCitation Excerpt :Considering the cooperative effects of MISP and fimbrin on rootlet length andstability, it is tempting to speculate that these factors bind todifferent sites on F-actin

119. Tropomyosins bind to the sides of actin filaments by spanning overseveral subunits (up to half a pitch per protein for the longesttropomyosin isoforms) and closely follow the filament strands

120. Pan1 contains a WH2 domain expected to bind actin and a CA (central-acidic) region expected to bind to Arp2/3 complex

121. Profilin–actin complexes bind to multiple polyproline sequences in the Cdc12 formin homology 1 (FH1) domain20,23,26 and transfer rapidly onto the fast growing barbed end of the filament26,28,29, whereas the FH2 domain moves processively on the growing end without dissociating26,28

122. Sla1 has been recently shown to bind to the polyproline motif of WASP protein, Las17, and to prevent its nucleation promotion factor (NPF) activity during the early stages of endocytosis (Feliciano & Di Pietro, 2012)

123. Most actin cross-linkers bind to actin filaments transiently in vitro (Xu et al