Supporting Online Material for

A Shared Docking Motif in TRF1 and TRF2 Used for Differential Recruitment of Telomeric Proteins

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This PDF file includes:

- Materials and Methods
- SOM Text
- Figs. S1 to S14
- Table S1
- References
Supporting Online Material

Materials and Methods

Protein expression and purification

Human TRF1\textsubscript{TRFH} (residues 65-267) and TRF2\textsubscript{TRFH} (residues 42-245) and four TRFH-binding peptides (TIN2\textsubscript{256-276}, Apollo\textsubscript{496-532}, PinX1\textsubscript{286-304} and Nbs1\textsubscript{419-449}) were expressed in E. coli BL21(DE3) using a modified pET28b vector with a SUMO protein fused at the N-terminus after the 6XHis tag. After induction for 16 hours with 0.1 mM IPTG at 25°C, the cells were harvested by centrifugation and the pellets were resuspended in lysis buffer (50 mM Tris-HCl pH 8.0, 50 mM NaH\textsubscript{2}PO\textsubscript{4}, 400 mM NaCl, 3 mM imidazole, 10% glycerol, 1 mM PMSF, 0.1 mg/ml lysozyme, 2 mM 2-mercaptoethanol, and home-made protease inhibitor cocktail). The cells were then lysed by sonication and the cell debris was removed by ultracentrifugation. The supernatant was mixed with Ni-NTA agarose beads (Qiagen) and rocked for 6 hours at 4°C before elution with 250 mM imidazole. Then Ulp1 protease was added to remove the His-SUMO tag. After Ulp1 digestion, the TRFH domains and the peptides were further purified by gel-filtration chromatography on Hiload Superdex75 column (GE Healthcare) equilibrated with buffer A (25 mM Tris-HCl pH 8.0, 150 mM NaCl and 5 mM DTT) and buffer B (100 mM ammonium bicarbonate), respectively. The purified TRFH domains were concentrated to 25 mg/ml and stored at -80°C. The purified peptides were concentrated by Speed Vac system and then lyophilized. The lyophilization products were then resuspended in water at a concentration of 50 mg/ml and stored at -80°C.
Limited protease (trypsin) cleavage of the TRF$_1^{TRFH}$-TIN$_2^{TBM}$ complex

TRF$_1^{TRFH}$ and TIN$_2^{TBM}$ were mixed at 1:1 molar ratio and the mixture was incubated with 0.2% w/w trypsin (Roche) at room temperature in 25 mM Tris-HCl pH 8.0, 150 mM NaCl, and 2 mM DTT. At various time points, 8 µl aliquots of the reaction were withdrawn, diluted with 12 µl of water and 5 µl of SDS loading dye, and run on 15% SDS–PAGE visualized with Coomassie brilliant blue stain.

MALDI mass spectrometry of the limited protease (trypsin) cleavage products

For MALDI mass spectrometric analysis, the preformed TRF$_1^{TRFH}$-TIN$_2^{TBM}$ complex was incubated with 0.2% w/w trypsin in 25 mM Tris-HCl pH 8.0, 150 mM NaCl, and 2 mM DTT at room temperature. Aliquots were withdrawn as described above for SDS–PAGE analysis. At the 30 min time point, 2 ml of the reaction mixture was co-crystallized with 2 ml sinapinic acid matrix. The samples were analyzed by MALDI-TOF-MS in linear mode. The major product by MALDI had an MH(+1) of 3,340.8 Da. Examination of the map of predicted trypsin sites revealed that this fragment corresponds to one predicted fragment: TIN$_2^{248-276}$ [MH(+1) 3,339.7 Da].

Crystallization, data collection and structure determination of the TRFH-peptide complexes

The TRFH domains of TRF1 and TRF2 and their binding peptides were mixed at a molar ratio of 1:1.5 and the mixtures were used for crystallization. Crystals were grown by hanging-drop-vapor-diffusion at 16°C. The precipitant/well solutions are solution A for TRF$_1^{TRFH}$-TIN$_2^{TBM}$, solution B for TRF$_2^{TRFH}$-TIN$_2^{TBM}$, and solution C for TRF$_2^{TRFH}$-
Apollo\textsubscript{TBM} (solution A: 100 mM Tris-HCl pH 8.6, 2.5 M sodium chloride, 350 mM magnesium chloride and 5 mM DTT; solution B: 50 mM Hepes PH 7.0, 14% PEG 550 MME, 5 mM magnesium chloride and 5 mM DTT; solution C: 50 mM MES pH 5.6, 2.6 M ammonium sulfate, 10 mM magnesium acetate and 5 mM DTT). All crystals were gradually transferred into harvesting solutions (100 mM Tris-HCl pH 8.6, 0.5 M sodium chloride, 150 mM magnesium chloride and 5M sodium formate for TRF1\textsubscript{TRFH-TIN2\textsubscript{TBM}}; 50 mM Hepes pH 7.0, 17.5% PEG 550 MME, 5 mM magnesium chloride and 25% glycerol for TRF2\textsubscript{TRFH-TIN2\textsubscript{TBM}}; and 50 mM MES pH 5.6, 500 mM ammonium sulfate, 10 mM magnesium acetate and 5 M sodium formate for TRF2\textsubscript{TRFH-Apollo\textsubscript{TBM}}). The crystals were then flash-frozen in liquid nitrogen for storage and data collection under cryogenic conditions (100K). Data were collected at the Advanced Photon Source beamlines 21ID-B and D and 23ID-B and D, and processed using HKL2000 (S1). The TRF1\textsubscript{TRFH-TIN2\textsubscript{TBM}}, the TRF2\textsubscript{TRFH-TIN2\textsubscript{TBM}}, and the TRF2\textsubscript{TRFH-Apollo\textsubscript{TBM}} structures were solved by molecular replacement in CNS (S2) by using the previously published unliganded TRF1\textsubscript{TRFH} (PDB: 1h6o) and TRF2\textsubscript{TRFH} (PDB: 1h6p) structures as search models (S3). Structure refinement was done in CNS and model building in O (S4).

**Isothermal Titration Calorimetry**

The equilibrium dissociation constants of the wild-type and mutant TRFH-peptide interactions were determined by using a VP-ITC calorimeter (MicroCal). The enthalpies of binding between the TRFH domains (15-40 µM) and the TRFH-binding peptides (TIN2\textsubscript{TBM}, Apollo\textsubscript{TBM}, PinX1\textsubscript{TBM} and Nbs1\textsubscript{TBM}) (150-500 µM) were measured at 20°C in 20 mM sodium phosphate (pH 7.0) and 100 mM NaCl. Two independent experiments
were performed for every interaction described here. ITC data were subsequently analyzed and fit using Origin 7 software (OriginLab) with blank injections of peptides into buffer subtracted from the experimental titrations prior to data analysis.

**Coimmunoprecipitation**

We plated human 293T cells (4-5 X10^6) and transfected them 20–24 h later by the calcium-phosphate coprecipitation method using 10 µg of total plasmid DNA per 10-cm dish. We changed the medium after 12 h and collected cells 24–30 h after transfection. For immunoprecipitations, we dislodged 293T cells from the dish by flushing with cold phosphate-buffered saline (PBS), collected them by centrifugation and lysed them in ice-cold buffer (50 mM Tris-HCl (pH 7.4), 1 mM EDTA, 400 mM NaCl, 1% Triton X-100, 0.1% SDS, 1 mM DTT, 1 mM phenylmethylsulfonyl fluoride, 1 g/ml of aprotinin, 10 g/ml of pepstatin and 1 g/ml of leupeptin). After 10 min on ice, we added an equal volume of ice-cold water and thoroughly mixed. After centrifugation in a microcentrifuge (14,000 r.p.m. for 10 min) we collected the supernatants and used them for immunoprecipitation. We prepared lysates from one 10-cm dish and mixed them with 2µg of the mouse monoclonal antibody 9E10 for each immunoprecipitation (at 4 °C for 5–6 h, nutating). During the final hour, we added 30 µl (settled volume) of protein G–Sepharose beads (preblocked overnight with 10% bovine serum albumin in PBS) to each tube. We washed the beads four times with lysis buffer, eluted proteins with Laemmli loading buffer and analyzed them by SDS-PAGE.

**Far Western Analysis**
The far western was carried out as previously described (S5). GST-fusion proteins were prepared from induced bacterial cultures, subjected to SDS-PAGE, and then blotted onto nitrocellulose. The blots were incubated in blocking buffer (10 mM HEPES pH 7.5, 50 mM NaCl, 10 mM MgCl$_2$, 0.1 mM EDTA, 1 mM dithiothreitol, 10% glycerol, and 5% milk) for 3 h at 4 °C. Following the blocking step, the blots were probed overnight at 4 °C with $^{35}$S-labeled in vitro translated (IVT) protein prepared using the TNT T7 Coupled Reticulocyte Lysate System (Promega) (a 50-µl reaction mixture in 5 ml of blocking buffer). The next morning, the blots were washed five times every 30 min in wash buffer (10 mM HEPES pH 7.5, 50 mM NaCl, 10 mM MgCl$_2$, 0.1 mM EDTA, 1 mM dithiothreitol, 10% glycerol, and 0.25% milk) and then incubated with Amplify (Amersham Biosciences) for 10 min. The blots were exposed on a PhosphorImager screen overnight.

**Immunofluorescence and fluorescent in situ hybridization (IF-FISH)**

Cells grown on coverslips were pre-extracted for 90 sec in 0.5% Triton-X 100 and fixed for 10 min in 2% paraformaldehyde at room temperature. Cells were blocked for 1 h in PBG (0.2% [w/v] cold water fish gelatin [Sigma G-17765]; 0.5% BSA in PBS) and incubated for 2 h at room temperature with HA.11 (Covance). The coverslips were then incubated for 1 h with secondary antibodies raised against mouse and labeled with Rhodamine Red-X (RRX, Jackson). IF-FISH was performed as described (Herbig et al. 2004) using a FITC-OO-(AATCCC)$_3$ PNA telomere probe (Applied Biosystems). DNA was counterstained with 4,6-diamidino-2-phenylindole (DAPI) and slides were mounted in 90% glycerol/10% PBS containing 1 µg/ml p-phenylene diamine (Sigma). Digital
images were captured with a Zeiss Axioplan II microscope with a Hamamatsu C4742-95 camera using Improvision OpenLab software.

Supporting Text

Identification of the TRF1-binding peptide of TIN2.

The TRF1-TIN2 interaction was previously mapped to TRF1_TRFH and the C-terminus of TIN2 (S6). In order to study this interaction, we reconstituted the complex of TRF1_TRFH and the C-terminal fragment of TIN2 (TIN2_{181-354}) (Fig. S1A). However, the purified complex showed a large apparent molecular weight (>200 kDa) on gel filtration chromatography. Given that TRF1_TRFH adopts a compact conformation (S3), the abnormal chromatographic behavior of the TRF1_TRFH-TIN2_{181-354} complex suggests that a significant portion of TIN2_{181-354} is very likely to be unstructured. Limited proteolysis and mass spectrometry identified a protease-resistant peptide of TIN2 (residues 248-276) in the presence of TRF1_TRFH (Fig. S1). Further mapping revealed that a short peptide of TIN2, TIN2_{256-276}, retains the TRF1_TRFH binding activity.

Discussion

Attempt to search for structural homologous proteins of the TRFH domains using DALI (S7) failed to identify common topology (S3). However, a comparison of the TRF1_TRFH-TIN2_TBM and the TRF2_TRFH-Apollo_TBM complexes with a TPR (tetratrico-peptide repeat) domain-peptide complex (HopTPR2A-Hsp90) (S8) reveals an intriguing similarity. Upon maximizing the fit of the bound peptides, helices α1, α2 and α3 of the TRFH peptide-binding interface align well with three peptide-binding helices in TPR (Figs. S14A and
S14B). Notably, similar to the central leucine residues in TBMs, the side chain of V853 in Hsp90 also inserts into a hydrophobic pocket formed by HopTPR2A (Fig. S14C). This suggests that although the TRFH and TPR domains have very different overall structures, they use similar architectural principles for peptide binding. However, because the topological relationships of the peptide-binding helices are completely different in TRFH and TPR (Fig. S14D), it would be impossible to detect the hidden similarity between the two domains without the 3D structural information.

A distinct feature of the TRFH mediated interactions is that each TRFH monomer binds to one TBM peptide and the two peptide-docking sites do not interfere with each other (Figs. 1B and 4B). This is likely to have important consequences for function. A TRFH dimer may, like PCNA in DNA replication and repair (S9), recruit two different shelterin associated factors into close vicinity at telomeres and orchestrate complicated mechanisms to perform in an ordered pathway.

Our results provide a general framework for the identification and understanding of other TRFH mediated interactions within the shelterin complex. Based on the three TRFH domain-binding proteins characterized here, it is now possible to quickly establish the involvement of the TRFH docking site in the binding of a variety of proteins to shelterin. The prediction is that such interactions should be abolished by mutating F142 in TRF1 or F120 in TRF2, which are essential for stabilizing the refolded conformation of loop L_{34} induced by ligand binding (Figs. 1C, S4, S10C and S10E). Furthermore, given that TRF1 and TRF2 function as the protein docking sites for shelterin, it is anticipated that more TRFH-TBM interactions will be discovered in the future. As more TBM sequences become available, it should be possible to derive the accurate consensus
sequences for the TRFH domains of TRF1 and TRF2, which in turn can help identify novel shelterin associated proteins that may have escaped being implicated in telomere biology based on other approaches.
Supporting Figures

Fig. S1. Identification of the TRF1-binding peptide of TIN2. (A) SDS-PAGE time course of limited protease (trypsin) cleavage of the TRF1<sub>TRFH</sub>-TIN2<sub>181-354</sub> complex. Lanes in minutes of time of the reaction are labeled. (B) MALDI-TOF mass spectrum of trypsin digestion products.
**Fig. S2.** Organization of the TRF1 and TIN2 polypeptide chains. In TRF1, the N-terminal acidic region is colored in red, the C-terminal Myb/SANT domain in slate, and the TRFH domain in green. In TIN2, the N-terminal domain (TIN2_{1-220}) is in light blue, and the central TBM region in yellow. The shaded area between TRF1 and TIN2 indicates that the TRF1-TIN2 interaction is mediated by TRF1_{TRFH} and TIN2_{TBM}. Residue numbers at the various subdivisions are indicated.

**Fig. S3.** Electron density map of TIN2_{TBM}. The Sigma-A weighted 2F\textsubscript{o}-F\textsubscript{c} map that shows residues 257-268 are ordered. The refined model of the TIN2\textsubscript{TBM} peptide is superimposed on the map. Contours are draw at the 1.0 \(\sigma\) level. The peptide is shown as a stick representation with carbon colored in yellow, nitrogen blue, and oxygen red. TRF1\textsubscript{TRFH} is shown in ribbon model and colored in green.
**Fig. S4. Stereo image of the TRF1\textsubscript{TRFH}-TIN2\textsubscript{TBM} interaction.** TIN2\textsubscript{TBM} and the TIN2\textsubscript{TBM} interacting residues of TRF1\textsubscript{TRFH} are presented as stick models and colored in yellow and cyan, respectively. TRF1\textsubscript{TRFH}-TIN2\textsubscript{TBM} intermolecular hydrogen bonding and ion pairing interactions are shown as dashed magenta lines.

**Fig. S5. Organization of the TRF2 and TIN2 polypeptide chains.** In TRF2, the N-terminal basic region is colored in blue, the C-terminal Myb/SANT domain in slate, the TRFH domain in cyan, and TRF2\textsubscript{352-366} in pink. The domains of TIN2 are colored as in Fig. S2.
Fig. S6. Structure based sequence alignment of the TRFH domains of TRF1 (green) and TRF2 (cyan). Magenta and orange blocks denote the residues that make hydrophobic and hydrophilic interactions with the TIN2_{TBM} (for TRF1 and TRF2) and the Apollo_{TBM} (for TRF2 only) peptides, respectively. Red boxes denote E146 and E192 of TRF1 and their equivalent residues in TRF2 (A124 and K173). E146 and E192 of TRF1 make four electrostatic interactions with R266 and R267 of TIN2.

Fig. S7. A short motif of TRF2 (TRF2_{350-366}) binds the N-terminal domain of TIN2. (A) Overlay of the gel filtration chromatography profiles (Superdex 200) of the purified Sumo-TRF2_{350-366}, TIN2_{1-220} and the Sumo-TRF2_{350-366}-TIN2_{1-220} complex indicates that Sumo-TRF2_{350-366} and TIN2_{1-220} forms a stable complex. Elution positions of 158, 44 and 17 kDa proteins markers are indicated. (B) SDS-PAGE gels of Sumo-TRF2_{350-366}, TIN2_{1-220} and the Sumo-TRF2_{350-366}-TIN2_{1-220} complex fractions corresponding to the gel filtration profiles in panel A.
Fig. S8. The TRF2<sub>TRFH</sub>-TIN2<sub>TBM</sub> interaction. (A) In vitro ITC measurement of the TRF2<sub>TRFH</sub>-TIN2<sub>TBM</sub> interaction. (B) The structure of the TRF2<sub>TRFH</sub>-TIN2<sub>TBM</sub> complex. TRF2<sub>TRFH</sub> and TIN2<sub>TBM</sub> are colored in cyan and yellow in one complex and blue and orange in the other. (C) Stereo image of the TRF2<sub>TRFH</sub>-TIN2<sub>TBM</sub> interface. TRF2<sub>TRFH</sub> is shown in a cyan ribbon model. The TIN2<sub>TBM</sub> peptide and the TIN2<sub>TBM</sub> interacting residues of TRF2<sub>TRFH</sub> are presented as stick models and colored in yellow and green, respectively. TRF2<sub>TRFH</sub>-TIN2<sub>TBM</sub> intermolecular hydrogen bonding and ion pairing interactions are shown as dashed magenta lines. (D) Schematic depiction of the TRF2<sub>TRFH</sub>-TIN2<sub>TBM</sub> interaction. The main-chain atoms of TIN2<sub>TBM</sub> are shown as small circles (carbon in yellow, C<sub>α</sub> in orange, oxygen in red and nitrogen in blue). Residues of TRF1<sub>TRFH</sub> are shown as oval (side-chain interaction) and square (main-chain interaction) boxes and shaded cyan. Hydrophilic and hydrophobic interactions are shown as straight magenta and curved red lines, respectively. The paleyellow arrows denote the intermolecular β-sheet.
Chen et al.

Fig. S9. Comparison of the TIN2R266 binding sites in TRF1_{TRFH} and TRF2_{TRFH}. (A) and (B) The TIN2R266-binding sites in TRF1_{TRFH} (panel A) and TRF2_{TRFH} (panel B). The surface representations of TRF1_{TRFH} and TRF2_{TRFH} are colored in green and cyan, respectively. *E*146 and *E*192 of TRF1_{TRFH} are highlighted in red, whereas their structurally equivalent residues in TRF2_{TRFH} are *A*124 (orange) and *K*173 (blue). The electrostatic interactions in the TRF1_{TRFH}-TIN2_{TBM} complex are shown as dashed magenta lines. (C) *In vitro* ITC binding data of the swapping mutants of TRF1_{TRFH} and TRF2_{TRFH}. TRF1*E*146 is less important than *E*192 for the TRF1_{TRFH}-TIN2_{TBM} binding specificity.

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<td>TIN2_{TBM}</td>
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Fig. S10. **The TRF2\textsubscript{TRFH}-Apollo\textsubscript{TBM} interaction.** (A) Organization of the TRF2 and Apollo polypeptide chains. The domains of TRF2 are colored as in Fig. S5. In Apollo, the N-terminal mettalo-β-lactamase domain is in green, the β-CASP domain in purple, and the C-terminal TBM in orange. (B) Electron density of Apollo\textsubscript{TBM} in Sigma-A weighted 2F\textsubscript{o}−F\textsubscript{c} map shows residues 498-509 are ordered. The refined model of the Apollo\textsubscript{TBM} peptide is superimposed on the map. Contours are draw at the 1.0 σ level. (C) Stereo image of the TRF2\textsubscript{TRFH}-Apollo\textsubscript{TBM} interaction. The Apollo\textsubscript{TBM} peptide and the Apollo\textsubscript{TBM} interacting residues of TRF2\textsubscript{TRFH} are presented as stick models and colored in yellow and green, respectively. (D) The shape of the hydrophobic pocket of TRF2 (blue mesh) complements the side chain of Apollo L506 well. (E) Schematic depiction of the TRF2\textsubscript{TRFH}-Apollo\textsubscript{TBM} interaction. The color scheme and symbol usage are the same as those in Fig. S8D. The paleyellow cylinder denotes the Apollo helix (G499-Y504).
**Fig. S11. Apollo\textsubscript{TBM} specifically binds to TRF2\textsubscript{TRFH}.** (A) and (B) Surface representations of the TIN2\textsubscript{TBM}F258 binding site of TRF1\textsubscript{TRFH} (panel A) and the Apollo\textsubscript{TBM} helix binding site of TRF2\textsubscript{TRFH} (panel B). (C) and (D) Superposition of the TRF1\textsubscript{TRFH}-TIN2\textsubscript{TBM} and the TRF2\textsubscript{TRFH}-Apollo\textsubscript{TBM} complexes suggests that Apollo\textsubscript{TBM} is specific for TRF2\textsubscript{TRFH}. Upon superposition of the F/Y-x-L-x-P motifs of TIN2\textsubscript{TBM} and Apollo\textsubscript{TBM}, it is obvious that the local conformations of loop L\textsubscript{23} and helices \textalpha{}2 and \textalpha{}3 of TRF1\textsubscript{TRFH} are significantly different from those of TRF2\textsubscript{TRFH} so that the Apollo helix (especially residues L500 and Y504) cannot fit into the peptide binding site of TRF1\textsubscript{TRFH}. 
**Fig. S12. The TRF1<sub>TRFH</sub>-PinX1<sub>TBM</sub> interaction.** (A) Sequence alignment of the TRFH binding peptides of TIN2, PinX1 and Apollo. The black box denotes the F/Y-x-L-x-P motif. The conserved residues are colored in red. (B) ITC measurement of the interactions of TRF1<sub>TRFH</sub> (red) and TRF2<sub>TRFH</sub> (blue) with the PinX1<sub>TBM</sub> peptide. (C) ITC data of binding for mutant peptides derived from PinX1<sub>TBM</sub> and TRF1<sub>TRFH</sub>.
**Fig. S13. Identification of putative TRFH-binding proteins.** (A) List of telomere-associated proteins that contain the F/Y-x-L-x-P motif. Putative TRFH-binding proteins were identified through program Scansite (S10). The F/Y-x-L-x-P motif is highlighted by yellow boxes. (B) Human Nijmegen breakage syndrome protein (Nbs1) is a component of the Mre11-Rad50-Nbs1 (MRN) complex and was found to associate with TRF2 at telomeres during S phase (S11). Our *in vitro* ITC measurement showed that a peptide of Nbs1 (aa 419-449) containing the F/Y-x-L-x-P motif (see panel A) binds to the TRFH domain of TRF2 but not TRF1. Whether the F/Y-x-L-x-P motif of Nbs1 mediates the TRF2-Nbs1 interaction in cells needs further investigation.
Fig. S14. The TRFH and TPR domains use similar architectural principles for peptide binding. For simplicity, only the comparison with the TRF1<sub>TRFH</sub>-TIN2<sub>TBM</sub> complex is shown here and the same result can also be obtained with the TRF2<sub>TRFH</sub>-Apollo<sub>TBM</sub> complex. (A) and (B) Two different views of the superposition of the peptide-binding sites of the TRF1<sub>TRFH</sub>-TIN2<sub>TBM</sub> and the HopTPR2A-Hsp90 complexes. The TIN2 and Hsp90 peptides are colored in yellow and cyan, respectively. Hsp90 and the well-alignment region of the TIN2 peptide are in stick model. The rest of TIN2 is in ribbon model. The side chains of TIN2 L260 and Hsp90 V853 are shown to highlight the local similarity of the conformations of two peptides. The three peptide-binding helices of TRFH and TPR are colored in green (α2, α3 and the C-terminal half of α1 in TRF1) and red (A1, A2 and B1 in HopTPR2A). The rest of TRF1 and HopTPR2A are in gray and salmon, respectively. (C) The shape of the hydrophobic pocket of HopTPR2A (red mesh) complements the side chain of Hsp90 V853. (D) Schematic diagrams of the TRF1<sub>TRFH</sub>-TIN2<sub>TBM</sub> and the HopTPR2A-Hsp90 interactions show the difference in topology between the TRF1 and HopTPR2A peptide-binding helices. TRF1 and HopTPR2A are colored as in panels A and B. Two colored arrows represent the TIN2 (yellow) and Hsp90 (cyan) peptides.
**Supporting Table**

**Table S1.** Data collection, crystallization and refinement statistics of the TRFH-TBM peptide complexes.

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<td>TRFH domain: 34.4, TBM Peptide: 48.1, Water: 46.9</td>
<td>TRFH domain: 51.2, TBM Peptide: 65.9, Water: 62.0</td>
<td>TRFH domain: 49.5, TBM Peptide: 51.1, Water: 49.0</td>
</tr>
<tr>
<td>R.m.s. deviations</td>
<td>Bond lengths (Å): 0.0057, 0.0080, 0.0065</td>
<td>Bond angles (°): 0.95, 1.69, 1.63</td>
<td>Bond lengths (Å): 0.0057, 0.0080, 0.0065</td>
</tr>
</tbody>
</table>
Supporting References


