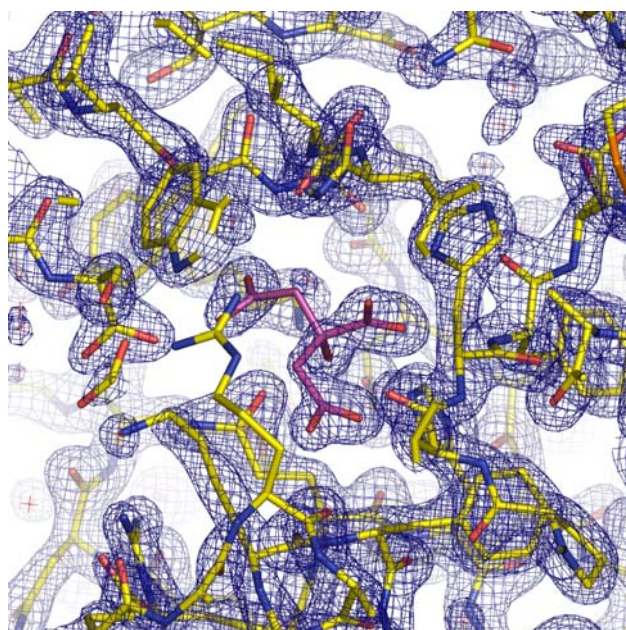
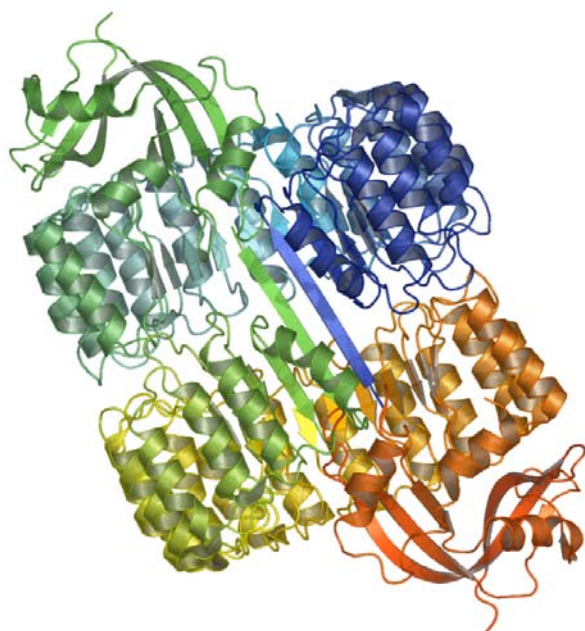




<b>Target ID</b>	GO.78623 and GO.74631	
<b>Source Organism</b>	<i>Homo sapiens</i>	
<b>Target Name</b>	RNase/hRI Complex	
<b>PDB Entry</b>	1Z7X	Deposition: 21-Jun-2005
<b>Function</b>	pancreatic ribonuclease A complexed with inhibitor (EC 3.1.27.5) (FF/Refine: 2Q4G)	
<b>Produced From</b>	<i>E. coli</i> BL21(DE3) pET-22B	
<b>Structure by X-ray</b>	Resolution: 1.95 Å	R-value (R-free): 17.8% (23.6%)
	No. of Residues/ASU: 1175	Complexes/ASU: 2
<b>Data Collected At</b>	Advanced Photon Source SBC 22-ID 12-Mar-2005	
<b>Authors</b>	J.G. McCoy, G.N. Phillips, Jr., E. Bitto, G.E. Wesenberg, C.A. Bingman	



### Structural Features

The x-ray crystal structure of human ribonuclease inhibitor (hRI) in complex with human pancreatic ribonuclease (RNase 1), solved in collaboration with the Ronald Raines lab (also at UW-Madison Biochemistry), gives a molecular picture of how the cell regulates the potentially damaging activity of RNase 1. It is the third structure of hRI in complex with a member of the ribonuclease A (RNase A) superfamily. By comparison to the other three structures, the hRI/RNase 1 complex shows the plasticity and generality of the leucine-rich repeat structure of hRI for accommodating, but still tightly inhibiting different members of the RNase A superfamily. Ribonucleases show promise as possible cancer therapeutics. Ribonucleases can be transformed into cancer therapeutics by blocking the inhibitory interaction of hRI. Creating variants of RNase 1 that evade hRI has been difficult, but this crystal structure may provide the important link to create cancer therapeutics using RNase 1.

*References:* (1) Johnson, R.J., McCoy, J.G., Bingman, C.A., Phillips, G.N., Jr., Raines, R.T. (2007) Inhibition of human pancreatic ribonuclease by the human ribonuclease inhibitor protein. *J Mol Biol* 368(2):434-49.

<b>Percent Identity with Nearest PDB Structure at Time Solved</b>	91% over 127 aa (1E21)
	95% over 460 aa (2BEX)
<b>Pfam Cluster</b>	RNase_A,
<b>Sequence Cluster Size : Structures in PDB</b>	449 and 883

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