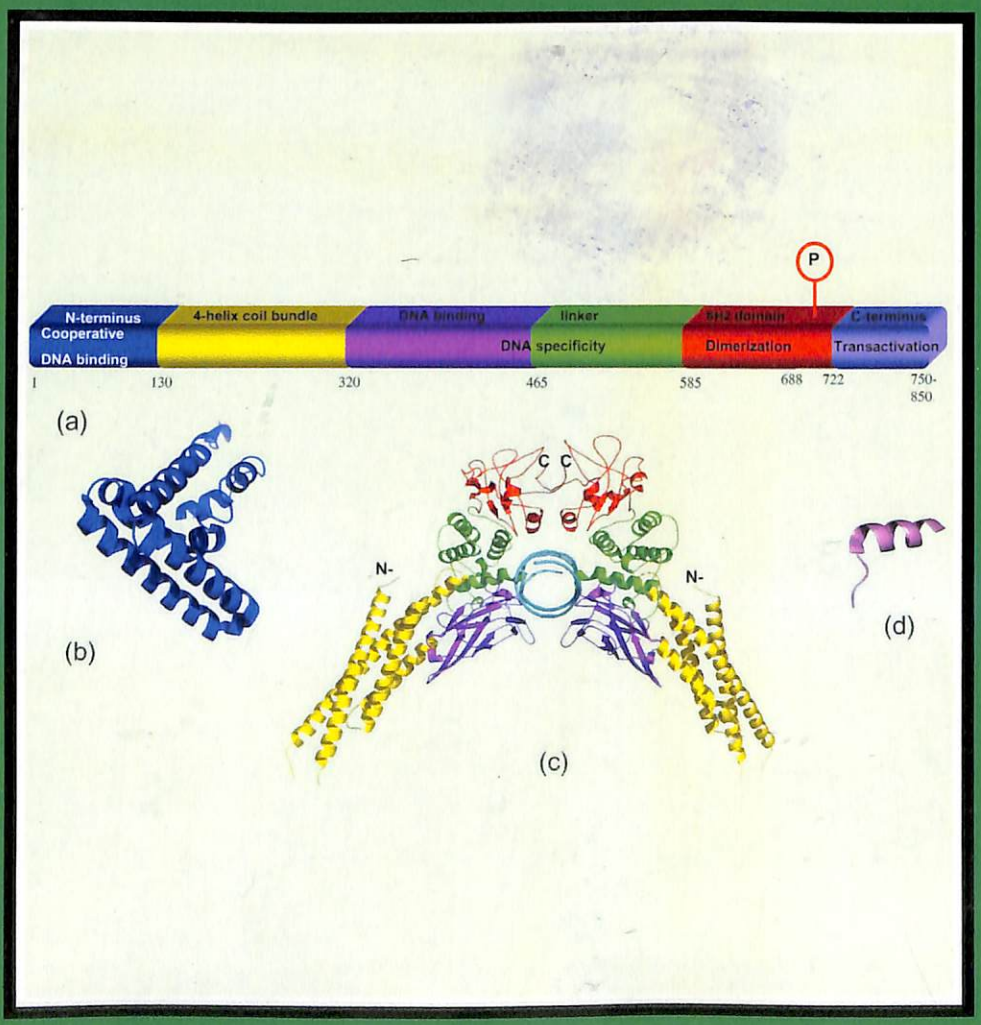
Volume 12 Number 1 January 2012

Current Cancer Drug Targets



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BENTHAM SCIENCE

Current Cancer Drug Targets

Volume 12, Number 1, January 2012

Contents

The cover image is taken from the article by Neamati *et al. Current Cancer Drug Targets* **2007**, *7*(1), 91-107. It illustrates the structural organization of Stat proteins: (a) domain formation, (b) 8-helical N-terminus (aa1-aa123) as resolved in Stat1 (PDB 1YVL), important for cooperative DNA binding, (c) Stat3 structure (PDB 1BG1) covering residues aa136-aa716, the elongated 4-helix coil domain (aa136-aa319) is shown in yellow, the DNA binding domain (aa320-aa465) in magenta, the linker domain (aa466-aa584) in green, the SH2 domain (aa585-aa716) in red, and the bound DNA is in cyan, and (d) five-residue fragment of the C-terminal transactivation domain. The structure of the motif is taken from a complex with PAS-B domain of the nuclear receptor coactivator 1 (PDB 1OJ5).

*Editor's Choice

Current Cancer Drug Targets

Volume 12, Number 2, February 2012

Contents

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*Editor's Choice

Current Cancer Drug Targets

Volume 12, Number 3, March 2012

Contents

Hot Topic

Target Therapy in Brain Tumours and Metastases

Guest Editors: M. Caraglia and R. Addeo

The cover image is taken from the article by Caraglia *et al. Current Cancer Drug Targets* **2012**, *12*(3), 186-196. It illustrates that several molecular characteristic of the blood-brain-barrier can be used in order to allow the penetration of nano-encapsulated anti-cancer agents: gaps induced by metastasis-mediated disruption, metabolic carrier mediated-transportation, receptor mediated-endo-cytosis or trans-cytosis and fusion peptides mediated-penetration.

Current Cancer Drug Targets

Volume 12, Number 4, May 2012

Contents

Hot Topic

Molecularly Targeted Treatments for Colorectal Cancer: Advances and Limitations

Guest Editor: Silvio Parodi

Contd.....

*Editor's Choice

General Articles

The cover image is taken from the article by Neamati *et al. Current Cancer Drug Targets* **2007**, 7(1), 91-107. It illustrates the structural organization of Stat proteins: (a) domain formation, (b) 8-helical N-terminus (aa1-aa123) as resolved in Stat1 (PDB 1YVL), important for cooperative DNA binding, (c) Stat3 structure (PDB 1BG1) covering residues aa136-aa716, the elongated 4-helix coil domain (aa136-aa319) is shown in yellow, the DNA binding domain (aa320-aa465) in magenta, the linker domain (aa466-aa584) in green, the SH2 domain (aa585-aa716) in red, and the bound DNA is in cyan, and (d) five-residue fragment of the C-terminal transactivation domain. The structure of the motif is taken from a complex with PAS-B domain of the nuclear receptor coactivator 1 (PDB 1OJ5).

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*Editor's Choice

Current Cancer Drug Targets

Volume 12, Number 5, June 2012

Contents

Hot Topic

Novel and Emerging Drugs for Leukemias

Guest Editor: Tadeusz Robak

General Articles

Contd.....

*Editor's Choice

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Current Cancer Drug Targets

Volume 12, Number 6, July 2012

Contents

Contd.....

*Editor's Choice

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Current Cancer Drug Targets

Volume 12, Number 7, September 2012

Contents

Hot Topic

New Approaches in the Treatment of Multiple Myeloma: From Target-based Agents to the New Era of microRNAs (Dedicated to the Memory of Prof. Salvatore Venuta)

Guest Editors: P. Tassone and P. Tagliaferri

Contd....

Editor's Choices

General Articles

The cover image is taken from the article by Tagliaferri *et al Current Cancer Drug Targets* 2012, 12 (7). The biogenesis and the action of miRNAs are described: miRNAs are expressed as a pri-miRNA long transcript which is then cleaved by DROSHA to a pre-miRNA hairpin that traslocates in the cytoplasm following exportin 5 binding. Pre-miR DICER digestion produces miR/miR* duplex. The RISC complex incorporates one strand of the duplex, the mature miRNA, driving it to the 3'UTR of mRNA target and inducing translational repression (partial homology) or mRNA deadenylation (perfect homology). Alternatively, mature miRNAs may bind the open reading frame or the 5'UTR of target genes inducing transcription activation or may act as decoy for translation repressor ribonucleoproteins.

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*Editor's Choices

Current Cancer Drug Targets

Volume 12, Number 8, October 2012

Contents

The cover image is taken from the article by Tang *et al.* Current Cancer Drug Targets 2012, 12(8), 1032-1042. It illustrates that LRRC4 inhibits glioma cell growth and invasion by miR-185-mediated targeting of CDC42 or RhoA. (A) and (B) LRRC4 upregulates miR-185 expression by ISH and qRT-PCR. (C) and (D) LRRC4 inhibits the expression of CDC42, RhoA, VEGFA and Vimentin, increases GFAP by WB and IHC. (E) and (F) Knockdown of miR-185 or LRRC4 reverses the expression of CDC42, RhoA, VEGFA, Vimentin, and GFAP in U251/L cells. (G) and (H) Knockdown of miR-185 reverses cells proliferation and invasion in U251/L cells.

*Editor's Choices

Current Cancer Drug Targets

Volume 12, Number 9, November 2012

Contents

Hot Topic

Recent Advances in the Prevention and Therapy of Hepatocellular Carcinoma

Guest Editor: Anupam Bishayee

Contd....

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