Calendar

If you know of any meetings other than those listed below, or of changes to this information, please let *Chemistry & Biology* know by fax (44 (0)171 580 8428) or e-mail (chembiol@current-biology.com).

Chemistry & Biology September 1998, 5:R238–R239

ISSN 1074-5521

1–30 September, 1998
ECSOC-2: 2nd international electronic conference on synthetic organic chemistry, World Wide Web.
Contact: Esteban Pombo-Villar, ECSOC-2 Chairman, Preclinical Research, Novartis Pharma AG, 4002 Basel, Switzerland.
Tel: +41 61 324 9865
Fax: +41 61 324 9794
e-mail: esteban.pombo@pharma.novartis.com
http://www.mdpi.org/ecsoc-2.htm

15–20 September, 1998
The first international conference on the inhibitors of protein kinases, Warsaw, Poland.
Contact: Barbara Kleyny, Interdisciplinary centre for mathematical and computational modelling (ICM), University of Warsaw, Pawinskiego 5a, 02-106, Warsaw, Poland.
Tel: + 48 22 874 9115
Fax: + 44 22 874 9200
e-mail: ipk98@icm.edu.pl
http://www.icm.edu.pl/ipk98

21–23 September, 1998
Biochemical society meeting, Leicester, UK.
Contact: Katie Steptoe, The Meetings Office — Biochemical Society, 59 Portland Place, London W1N 3AJ, UK.
Tel: + 44 171 637 7626
e-mail: meetings@biochemsoc.org.uk

21–24 September, 1998
4th Annual conference and exhibition of the society for biomolecular screening, Baltimore, MD, USA.
Contact: Christine Giordano, Society for Biomolecular Screening, 36 Tamarack Avenue, Suite 348, Danbury, CT 06811, USA.
Tel: +1 203 743-1336
Fax: +1 203 748-7557
e-mail: c_giordano@prodigy.com
http://sbsonline.org

24–28 September, 1998
AACR Special conference in cancer research: cellular targets of viral carcinogenesis, Dana Point, CA, USA.
Contact: California American Association for Cancer Research, Public Ledger Building, Suite 826, 150 South Independence Mall West, Philadelphia, PA 19106-3483, USA.
Tel: +1 215 440 9300
Fax: +1 215 440 9313
e-mail: aacr@aacr.org
http://www.aacr.org

26 September – 1 October, 1998
European research conferences – membrane dynamics in exocytosis: molecular mechanisms, Giens, France.
Contact: J Hendovic, European Science Foundation, 1 quai Lezay-Marnésia, 67080 Strasbourg Cedex, France.
Fax: + 33 388 3669 87
e-mail: euresco@esf.org
http://www.esf.org/euresco

4–7 October, 1998
Structure-based functional genomics, Avalon, NJ, USA.
Contact: Becky Watson, Center for Advanced Biotechnology & Medicine, Rutgers University, 679 Hoes Lane, Piscataway, NJ 08854-5638, USA.
Tel: +1 732 235 5321
Fax: +1 732 235 5321
e-mail: watson@cabm.rutgers.edu,
http://www.cabm.rutgers.edu/bioinformatics_meeting/

4–9 October, 1998
3rd Australian peptide conference: from discovery to therapeutics, Queensland, Australia.
Contact: Dr AI Smith, Baker Medical Research Institute, PO Box 348, Prahran, Victoria 3181, Australia.
Tel: +61 3 9522 4333
Fax: +61 3 9521 1362
e-mail: Ian.Smith@Baker.edu.au
http://www.hfi.unimelb.edu.au/peptideoz/

5–7 October, 1998
G Protein-coupled receptors IV, Orlando, FL, USA.
Contact: BC USA Conferences, Inc., 225 Turnpike Road, Southborough, MA 01772-1749, USA.
Tel: +1 508 481 6400
Fax: +1 508 481 7911
e-mail: inq@ibcusa.com
http://www.ibcusa.com/conf/gprotein/index.html

6–7 October, 1998
IBC's 6th Annual conference on transcription regulation of clinically relevant genes, La Jolla, CA, USA.
Contact: IBC USA Conferences, Inc., 225 Turnpike Road, Southborough, MA 01772-1749, USA.
Tel: +1 508 481 6400
Fax: +1 508 481 7911
e-mail: inq@ibcusa.com

7–9 October, 1998
Strategies and techniques for identification of novel bioactive compounds, Zurich, Switzerland.
Contact: Cambridge Healthtech Institute, 1037 Chestnut Street, Newton Upper Falls, MA 02464, USA.
Tel: +1 617 630 1300
Fax: +1 617 630 1325

8–9 October, 1998
Nuclear hormone receptors: transcriptional mechanisms and novel targets, Philadelphia, USA.
Contact: NMHCC, Inc., PO Box 102713, Atlanta, GA 30368-2713, USA.
Tel: +1 941 365 4471
Fax: +1 941 365 0157

Elsewhere in biology

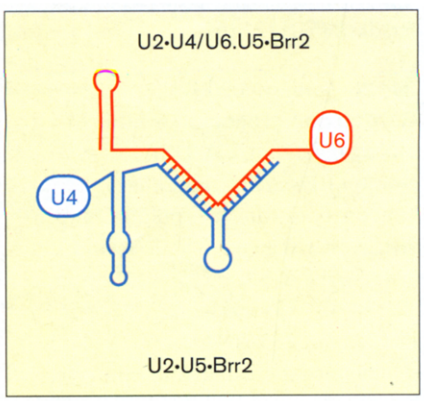
A selection of interesting papers published last month in *Chemistry & Biology*'s sister journals, *Current Biology*, *Folding & Design* and *Structure*, chosen and summarized by the staff of *Chemistry & Biology*.

Chemistry & Biology September 1998, 5:R240–R243

 ISSN 1074-5521

☐ **RNA unwinding in U4/U6 snRNPs requires ATP hydrolysis and the DEIH-box splicing factor Brr2.** Pratima L Raghunathan and Christine Guthrie (1998). *Curr. Biol.* **8**, 847–855.

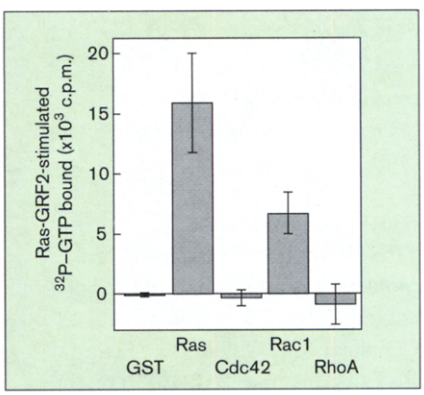
The dynamic rearrangements of RNA structure that occur during pre-mRNA splicing are thought to be mediated by members of the DExD/H-box family of RNA-dependent ATPases. Although



three DExD/H-box splicing factors have recently been shown to unwind synthetic RNA duplexes in purified systems, in no case has the natural biological substrate been identified. A duplex RNA target of particular interest is the extensive base-pairing interaction between U4 and U6 small nuclear RNAs. Because these helices must be disrupted to activate the spliceosome for catalysis, this rearrangement is believed to be tightly regulated *in vivo*. The authors have immunopurified Brr2, a DEIH-box ATPase, in a native complex containing U1, U2, U5 and duplex U4/U6 small nuclear ribonucleoprotein particles (snRNPs). Addition of hydrolyzable ATP to this complex results in the disruption of U4/U6 base-pairing, and the release of free U4 and U6 snRNPs. A mutation in the helicase-like domain of Brr2 (*brr2-1*) prevents these RNA rearrangements. Notably, U4/U6 dissociation and release occur in the absence of exogenously added pre-mRNA. Disruption of U4/U6 base-pairing in native snRNPs requires ATP hydrolysis and Brr2. This is the first assignment of a DExD/H-box splicing factor to a specific biological unwinding event. The unwinding function of Brr2 can be antagonized by the annealing activity of Prp24. The existence of a dynamic cycle, uncoupled from splicing, that interconverts free and base-paired U4/U6 snRNP is proposed.
29 June 1998, Research Paper, *Current Biology*

☐ **The exchange factor Ras–GRF2 activates Ras-dependent and Rac-dependent mitogen-activated protein kinase pathways.** Wing-Tze Fan, C Anne Koch, Carmen L de Hoog, Neil P Fam and Michael F Moran (1998). *Curr. Biol.* **8**, 935–938.

Ras and Rac are membrane-associated GTPases that function as molecular switches activating intracellular mitogen-activated protein kinase (MAPK) cascades and other effector pathways in response to extracellular signals. Activation of Ras and Rac into their GTP-bound conformations is directly controlled by specific guanine-



nucleotide exchange factors (GEFs), which catalyze GDP release. Several Ras-specific GEFs that are related to the budding yeast protein Cdc25p have been described, whereas GEFs for Rac-related GTPases contain a region that is homologous to the oncoprotein Dbl. The Ras–GRF1 and Ras–GRF2 proteins, which couple Ras activation to serpentine receptors and calcium signals, contain both Cdc25 and Dbl homology (DH) regions. Here, the authors demonstrate that Ras–GRF2 is a bifunctional signaling protein that is able to bind and activate Ras and Rac, and thereby coordinate the activation of the extracellular-signal-regulated kinase (ERK) and stress-activated protein kinase (SAPK) pathways.
27 July 1998, Brief Communication, *Current Biology*

☐ **Parallel evolution of CCR5-null phenotypes in humans and in a natural host of simian immunodeficiency viruses.** Emil Palacios, Laura Digilio, Harold M McClure, Zhiwei Chen, Preston A Marx, Mark A Goldsmith and Robert M Grant (1998). *Curr. Biol.* **8**, 943–946.

The C–C chemokine receptor CCR5 in humans and rhesus macaques (Macaca mulatta) serves as the primary coreceptor for cellular entry by macrophage-tropic strains of human immunodeficiency virus type 1 (HIV-1) and all reported strains of simian immunodeficiency virus (SIV). Humans homozygous for a 32 bp deletion allele of CCR5, resulting in a null phenotype, are highly resistant to infection by HIV-1, prompting development of therapies and vaccines targeting CCR5. The authors now report a novel deletion allele of CCR5, with an allele frequency of 0.04, in sooty mangabey monkeys (*Cercocebus torquatus atys*), a natural host of SIV (SIVsmm). The mutant protein was not expressed at the cell surface and accordingly did not function as a viral coreceptor. Primary activated lymphocytes from mangabeys heterozygous for the deletion allele expressed significantly less CCR5 on