Elsewhere in Biology

Chosen and summarized by the staff of *Chemistry & Biology*

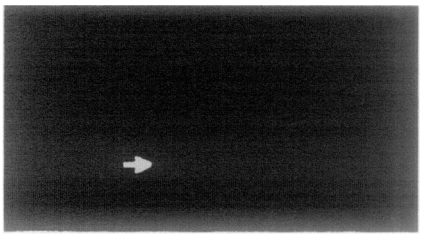
A selection of interesting papers published last month in *Chemistry & Biology*'s sister journals, *Current Biology*, *Folding & Design* and *Structure*.

Electronic Identifier: **1074-5521-004-00159**

Chemistry & Biology February 1997, 4:159–163

☐ **Differential effect of components of the extracellular matrix on differentiation and apoptosis.** Dorit Aharoni, Iris Meiri, Ruth Atzmon, Israel Vlodavsky and Abraham Amsterdam (1996). *Curr. Biol.* **7**, 43–51.

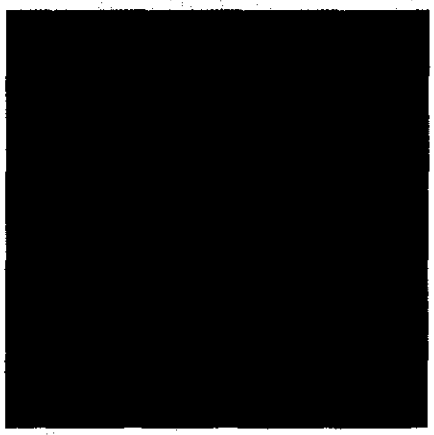
Basement membranes and extracellular matrices (ECMs) are the natural substances upon which cells migrate, proliferate and differentiate. Cellular responses to the ECM seem to be mediated by the combined action of basement membrane macromolecules, such as collagen IV, laminin, nidogen/entactin and heparan sulfate-containing proteoglycans, and regulatory molecules, such as growth factors and enzymes, that are immobilized and stored in the ECM by attachment to its macromolecular constituents, primarily to heparan sulfate proteoglycans (HSPG). To investigate the effect of the ECM on granulosa cell differentiation and death, primary granulosa cells were cultured on ECMs that lacked or contained bFGF (basic fibroblast growth factor). These otherwise identical ECMs were deposited by HR9 mouse endodermal cells, which do not synthesize bFGF, or by HR9 cells transfected with the bFGF gene. Both ECMs provided protection against apoptosis in serum-free medium, but only the bFGF-containing ECM maintained expression of the steroidogenic P450scc enzyme system and the production of progesterone. Laminin, but not fibronectin, was able to replace the ECM in protecting the cells from apoptosis; but not in maintaining steroidogenesis, whereas bFGF enhanced steroidogenesis but did not protect the cells against apoptosis. Cellular responses to ECM are therefore mediated by the combined action of macromolecular constituents and regulatory molecules, such as bFGF, that are sequestered and stored in the ECM.



18 December 1996*, Research Paper, Current Biology

☐ **Gravin, an autoantigen recognized by serum from myasthenia gravis patients, is a kinase scaffold protein.** J Brian Nauert, Theresa M Klauck, Lorene K Langeberg and John D Scott (1996). *Curr. Biol.* **7**, 52–62.

The subcellular targeting of protein kinases and phosphatases provides a mechanism for directing them to their preferred substrates. A recently identified mammalian scaffold protein, AKAP79, controls the location of two broad-specificity kinases and a phosphatase. The authors have identified and characterized another mammalian scaffold protein which coordinates the location of protein kinase A and protein kinase C. The protein turned out to be gravin, originally identified as a cytoplasmic antigen recognized by myasthenia gravis (a disease of neuromuscular transmission) sera. Residues 1526–1780 of gravin bind the regulatory subunit (RII) of protein kinase A with high affinity, and residues 265–556 bind protein kinase C. Gravin expression in human erythroleukemia cells can be induced with phorbol ester. Immunolocalization experiments show that gravin is concentrated at the cell periphery and is enriched in filopodia. These results indicate that gravin forms part of a signaling scaffold, and suggest that protein kinases A and C may participate in the coordination of signal transduction events in the filopodia of human erythroleukemia cells.



20 December 1996*, Research Paper, *Current Biology*

☐ **R-Ras can activate the phosphoinositide 3-kinase but not the MAP kinase arm of the Ras effector pathways.** Barbara M Marte, Pablo Rodriguez-Viciana, Stefan Wennström, Patricia H Warne and Julian Downward (1996). *Curr. Biol.* **7**, 63–70.

Although the small GTPase R-Ras has transforming activity, it is less potent than the closely related Ras oncogene products, and the pathways by which it exerts its effects on cellular proliferation have been unclear. The authors report that both Ras and R-Ras interact with phosphoinositide (PI) 3-kinase *in vitro*, and induce elevation of the levels of PI 3-kinase lipid products in intact cells. Unlike Ras, R-Ras does not activate Raf or mitogen-activated protein (MAP) kinase in cells. In co-transfection assays, the serine/threonine protein kinase PKB (or Akt) is effectively stimulated by R-Ras, Ras, mutants of Ras that activate PI 3-kinase but not other effectors, and activated forms

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