Gel brought to heal in mice

Curr. Biol. 13, 1697-1703 (2003)

A new bio-active gel speeds up wound healing in mice. The treatment targets connexin 43 (Cx43), a protein that is involved in cell-to-cell communication and which is overproduced at injury sites.

Cindy Qiu *et al.* tested the gel on mice with open skin wounds. The active ingredients — so-called antisense oligonucleotides — are nucleic acid sequences that prevent cells from making Cx43, by binding to the protein-encoding RNA. A single application enhanced the normal decline of Cx43 protein levels in and around the wound. The number of neutrophils — damaging inflammatory cells that gobble up cellular debris and bacteria — was reduced. Wounds were less swollen and healed more quickly.

The antisense oligonucleotides break down rapidly within the tissues they penetrate, so the effects are transient. The gel kick-starts the healing process, the authors say, and its effects persist long after the oligonucleotides are gone. After 12 days, the scars were smaller, narrower and flatter against the skin.

The gel may prove to be an effective and safe therapy for a variety of wounds, the authors speculate.

Helen R. Pilche

Vision

In the eye of the beholder

J. Exp. Biol. 206, 3963-3977 (2003)

Cells in the retina of male flies are specialized for spotting small, fast-moving objects, giving them an edge over their quarry when pursuing females with mating

Eyes right — male flies have the edge in the mating game.

in mind. The 'lovespot' in the male's eye can detect another fast-moving fly from 76 cm away: female eyes can manage only 33 cm, Brian G. Burton and Simon B. Laughlin have found.

The lovespot's receptors fire strongly in response to small objects, and they respond more quickly, pinpointing the target's position with greater precision, than do females' receptors. They also stop responding more quickly. This reduces blurring, as it erases any after-image from the retina.

There are plenty of examples of eye adaptation for particular purposes.
Frogs' eyes, for instance, respond strongly to anything that might be a flying meal.
But in this case the relevant events occur further along the image-processing path, not in the receptor cells themselves. Burton and Laughlin's work involved using a small moving light to mimic a fly target for another fly, and recording the electrical output from single receptor cells in the retina.

John Whitfield

Fluid dynamics

Jet-bending explained

Phys. Fluids 15, 3568-3571 (2003)

Last year, James M. Chwalek *et al.* reported that liquid jets squirting through a nozzle can be deflected by a temperature gradient (*Phys. Fluids* **14**, L37–L40; 2002). Michael P. Brenner and Srinivas Paruchuri have now furnished a simple theoretical explanation for this effect. This kind of understanding is essential for putting the effect to practical use, as it identifies the key factors influencing the jet's trajectory.

Controlling the flow of a liquid jet is central to the function of, for example, ink-jet printers. In some printers it is achieved by using a cumbersome array of

electrodes to deflect individual, electrically charged droplets after the jet has broken up. Deflecting the unbroken jet would potentially be much cleaner. It might also be useful for fibre spinning of polymer fibres, where the liquid polymer is forced through a nozzle.

Chwalek et al. produced deflection angles of several degrees by heating the nozzle asymmetrically. Brenner and Paruchuri show that the main cause is the Marangoni effect — the temperature dependence of surface tension, which sets up a bending stress on a cylindrical jet. Philip Bal

Neurobiology

Alzheimer's enzyme target

Mol. Cell 12, 553-563 (2003)

Researchers have struggled to work out how fragments of amyloid- β , which accumulate into plaques in the brains of people with Alzheimer's disease, kill neurons and cause dementia. Sungmin Song *et al.* have now identified a way in which the fragments block the breakdown of certain unwanted neuronal proteins.

Using a gene chip, the team found that amyloid- β ramps up production of an enzyme, E2-25K/Hip-2, which marks redundant proteins for destruction in a cell compartment called the proteasome. It does this by adding a ubiquitin chemical tag.

Levels of E2-25K/Hip-2 are higher than normal in the brains of Alzheimer's patients and in neurons exposed to toxic amyloid- β . Excess enzyme is implicated in amyloid- β -induced neuron death, but the exact mechanism involved is unclear. It causes a build-up of tagged proteins in neurons, and activates two proteins (ASK1 and JNK) that trigger cell death. The enzyme may also tag a protein, UBB+1, that inhibits the proteasome.

The authors speculate that drugs that block E2-25K/Hip-2 might help to prevent some of the symptoms of Alzheimer's disease. But the basic biology would need to be worked out far more before help could be offered to the estimated 15 million sufferers around the world.

Reproduction

Being compatible

Genes Dev. 17, 2502-2507 (2003)

External fertilization can be a hit-and-miss affair. Sperm and egg must first find each other and then unite — a tricky process for marine animals that release their gametes into water. The sea urchin, however, has evolved a series of steps to help the process and to prevent sperm from one species fertilizing the eggs of another.

The sperm express a protein, called bindin, at the tip. This protein helps sperm bind to egg, and it differs between sea urchin species. Its discovery prompted researchers to muse that complementary species-specific egg proteins, or receptors, must also exist.

Now these elusive egg proteins have been identified. Noriko Kamei and Charles G. Glabe looked for species-specific sequences in DNA from ovaries of the sea urchins *Strongylocentrotus franciscanus* and *S. purpuratus*. One sequence that differed between the two species turned out to encode a bindin receptor on eggs, which they dubbed EBR1.

The discovery may shed light on how the species have evolved and help reveal, at the molecular level, how sperm and egg interact to form fertile offspring.

Helen R. Pilcher

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