IN THE NEWS

Looking sheepish

You might think that one sheep looks pretty much like the next, but apparently this is not the case; if you happen to be a sheep, that is. According to a report in *Nature*, a sheep can recognize over 50 different sheep faces, and remember them for up to 2 years. The research team, led by Keith Kendrick at the Babraham Institute in Cambridge, UK, even trained the sheep to recognize a human face. Their findings also indicate that sheep might form long-lasting emotional attachments with their flock-mates.

The Times (UK, 8 November) reports that "The scientists focused on parts of the temporal and frontal lobes of the brain, which … process faces and emotional response in human beings and monkeys. When the sheep were shown pictures of an unknown sheep or human face, the activity in the brain remained quiet. When they were shown a familiar face, the cells sprang into life, showing a clear emotional response". Kendrick told the *Independent* (UK, 8 November): "In humans this area is active not just when you see a face but when you think about that person when they are not around. Sheep form individual friendships with one another, which may last for a few weeks. It's possible they may think about a face even when it's not there". As the *Daily Telegraph* (UK, 8 November) puts it, "while apparently mindlessly ruminating, sheep could be thinking about long-absent flock-mates — victims of foot and mouth culls, perhaps — or even shepherds".

Although this study provides a fascinating insight into ovine intelligence, one gets the impression that perhaps the researchers gave their experimental subjects too much credit. Apparently, during the recognition tests, 'ear tags were removed to rule out the unlikely possibility that the sheep "were reading them"' (*Daily Telegraph*).

Heather Wood

NEURODEGENERATIVE DISORDERS

SOD mimetics to the rescue

Superoxide, produced from oxygen in mitochondria, is the principal source of cellular reactive oxygen species (ROS). ROS have been implicated in the pathogenesis of a wide range of neurodegenerative diseases, as well as in normal ageing, but attempts to find antioxidant treatments for such conditions have met with only limited success. Superoxide is normally scavenged by mitochondrial superoxide dismutase (SOD2); mice lacking this enzyme die prematurely, showing metabolic and tissue pathologies that include a lethal spongiform neurodegenerative disorder. Melov and co-workers now show that SOD-catalase mimetics can attenuate ROS-related defects in these animals, and extend their lifespan by threefold.

In a previous study, Melov *et al.* treated *Sod2* nullizygous mice with the catalytic antioxidant 5,10,15,20-tetrakis (4-benzoic acid) porphyrin (MnTBAP). These mice usually die in the first week of life due to the peripheral effects of mitochondrial oxidative stress. By protecting peripheral tissues, MnTBAP treatment extended the lifespan of *Sod2* null mice; however, MnTBAP cannot cross the blood–brain barrier, and improving the survival of these animals unmasked a severe neurological disorder that was associated with spongiform change, chiefly in the frontal cortex and in brainstem nuclei.

The authors shifted their focus to neurologically active ROS scavengers; could a lipophilic SOD-catalase mimetic rescue the spongiform encephalopathy in *Sod2* nullizygous mice, and further improve their survival? Melov *et al.* examined the effects of three salen manganese complexes, which mimic the active site of metalloenzymes such as SOD2, on the fate of *Sod2* null mice. These compounds

(EUK-8, EUK-134 and EUK-189) significantly extended the lifespan of nullizygous mice beyond that of untreated or MnTBAP-treated animals. The EUK-treated mice showed no clinical evidence of neurodegenerative disease at 3 weeks of age, when MnTBAP-treated mice usually express severe motor disturbances. In histopathological and electron-microscopic analyses, the authors showed that salen

DEVELOPMENT

Making tracks

Photograph by George Trevis.

In adult vertebrates, the epibranchial ganglia relay sensory information from the oropharyngeal cavity to the hindbrain. During development, the sensory neurons that populate these ganglia migrate inwards from the epibranchial placodes — thickenings of the surface ectoderm that lie in clefts between the pharyngeal arches. The signals that guide the placodal cells to their target ganglia are unknown, but as reported in *Science*, a new study by Begbie and Graham indicates a role for another migratory cell population — the neural crest.

Using the lipophilic dye DiI to label migrating epibranchial placodal cells in chick embryos, the authors noticed that the trajectories of these cells bore a striking resemblance to certain neural crest migration routes in the hindbrain. By labelling neural crest and epibranchial placodal cells with different markers, they showed that the migrating placodal cells were closely apposed to the neuroglial crest, a late-migrating population of cells that gives rise to neurons and glia.

To test whether these cells have a role in epibranchial neuronal guidance, the authors ablated the neural crest by removing the entire hindbrain neuroepithelium. In these embryos, the placodal cells failed to migrate away from the surface epithelium, although they were still able to differentiate and extend axons. Ablation of specific regions of the hindbrain had more localized effects; for example, ablation of rhombomere 4 blocked only the migration of placodal cells destined for the geniculate ganglion.

To confirm that their results were caused by loss of the neural crest, and not the neural tube itself, the authors removed the neural tube only after the neural crest had emerged. In this case, the placodal cells were able to migrate towards their usual targets, although loss of