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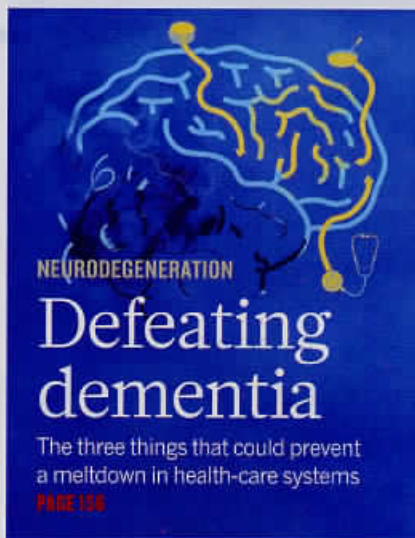
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Cliff erosion on Britain's southern coast

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173 CANCER

Bad neighbours cause bad blood

Ptpn11 mutations in the bone marrow alone can cause blood cancer in mice

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Eighty years of stress

A description of the stress response sparked a wealth of research

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A strange kind of liquid

Observations of self-bound quantum droplets

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177 SPINAL-CORD INJURY

Neural interfaces take another step forward

A neural interface enables monkeys paralysed in one leg to walk

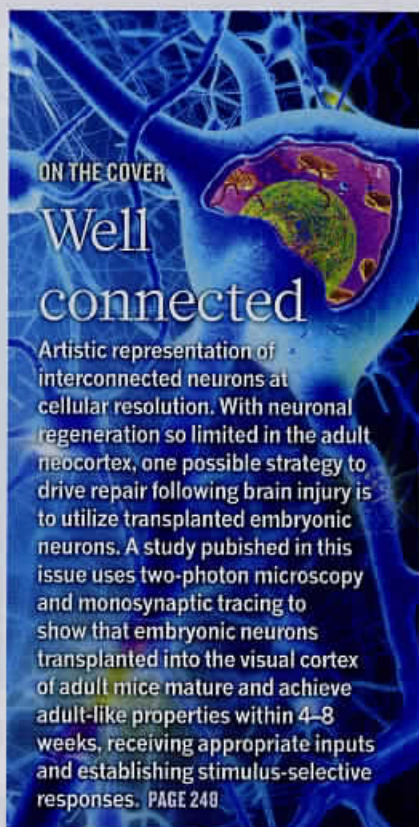
Andrew Jackson **SEE LETTER P.284**

ARTICLES

237 EVOLUTIONARY BIOLOGY The stem osteichthyan *Andreolepis* and the origin of tooth replacement

D Chen, H Blom, S Sanchez, P Tafforeau & P E Ahlberg

242 EVOLUTIONARY GENETICS Evolution of Osteocrin as an activity-regulated factor in the primate brain



Artistic representation of interconnected neurons at cellular resolution. With neuronal regeneration so limited in the adult neocortex, one possible strategy to drive repair following brain injury is to utilize transplanted embryonic neurons. A study published in this issue uses two-photon microscopy and monosynaptic tracing to show that embryonic neurons transplanted into the visual cortex of adult mice mature and achieve adult-like properties within 4–8 weeks, receiving appropriate inputs and establishing stimulus-selective responses. **PAGE 248**

B Ataman et al. **SEE N&V P.171**

248 NEUROSCIENCE Transplanted embryonic neurons integrate into adult neocortical circuits

S Falkner et al.

254 SOCIAL BEHAVIOUR Balancing selection shapes density-dependent foraging behaviour

J S Greene et al.

LETTERS

259 QUANTUM PHYSICS Self-bound droplets of a dilute magnetic quantum liquid

M Schmitt, M Wenzel, F Böttcher, I Ferrier-Barbut & T Pfau

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263 OPTICAL PHYSICS Tracking the ultrafast motion of a single molecule by femtosecond orbital imaging

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268 CHEMISTRY Catalytic alkylation of remote C–H bonds enabled by

proton-coupled electron transfer
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272 CHEMISTRY Amide-directed photoredox-catalysed C–C bond formation at unactivated sp^3 C–H bonds

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276 CLIMATE SCIENCES Fluvial sediment supply to a mega-delta reduced by shifting tropical-cyclone activity

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280 ENVIRONMENTAL SCIENCES Cultural innovation and megafauna interaction in the early settlement of arid Australia

G Hamm et al.

284 NEUROLOGICAL DISORDERS A brain–spine interface alleviating gait deficits after spinal cord injury in primates

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289 NEUROSCIENCE A basal ganglia circuit for evaluating action outcomes

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294 CELL BIOLOGY Fatty acid synthesis configures the plasma membrane for inflammation in diabetes

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299 STEM CELLS Reconstitution *in vitro* of the entire cycle of the mouse female germline

O Hikabe et al.

304 CANCER STEM CELLS Leukaemogenic effects of *Ptpn11* activating mutations in the stem cell microenvironment

L Dong et al. **SEE N&V P.173**

309 CANCER STEM CELLS Single cell RNA-seq supports a developmental hierarchy in human oligodendroglioma

I Tirosh et al.

314 ERRATUM Structural basis of potent Zika–dengue virus antibody cross-neutralization

G Barba-Spaeth et al.

314 CORRIGENDUM Human commensals producing a novel antibiotic impair pathogen colonization

A Zipperer et al.

314 CORRIGENDUM Age-dependent modulation of vascular niches for haematopoietic stem cells

A P Kusumbe et al.

NEURODEGENERATIVE DISEASES

REVIEWS

180 Ageing, neurodegeneration and brain rejuvenation

Ageing affects every organ in the body and plays a considerable part in neurodegeneration and associated brain disorders such as dementia. Indeed, the ageing brain becomes increasingly prone to neurodegenerative diseases. Tony Wyss-Coray presents current knowledge on brain ageing and neurodegeneration. He explains how protein factors in the blood circulation can modulate ageing and provide rejuvenating effects in the brain and other organs. Such discoveries raise the prospect of stalling or even reversing age-related neurodegenerative processes.

Tony Wyss-Coray

187 The road to restoring neural circuits for the treatment of Alzheimer's disease

Alzheimer's disease, a progressive loss of memory and cognition, is accompanied by cellular and cognitive hallmarks. These include the accumulation of extracellular aggregates of amyloid- β , a peptide implicated as having a primary role in the disease. Li-Huei Tsai, Rebecca Canter and Jay Penney explore why targeting amyloid- β might not be sufficient to treat cognitive decline in Alzheimer's disease. They propose that multipronged approaches incorporating genetic, cellular and neural circuit components might provide new or improved therapeutic strategies.

Rebecca G Canter, Jay Penney & Li-Huei Tsai

197 Decoding ALS: from genes to mechanism

Amyotrophic lateral sclerosis, also known as Lou Gehrig's disease or motor neuron disease, is characterized by a progressive degeneration of motor neurons in the brain and spinal cord. Genetic factors are known to drive this neurodegeneration as well as to increase susceptibility to the disease or to influence its progression. Paul Taylor, Robert Brown and Don Cleveland discuss advances in our understanding of the molecular



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mechanisms that underlie amyotrophic lateral sclerosis. They also describe how such knowledge might translate into therapies that can attenuate the path of this lethal disease.

J Paul Taylor, Robert H Brown Jr & Don W Cleveland

207 Defects in trafficking bridge Parkinson's disease pathology and genetics

The progressive loss of midbrain dopamine neurons represents the core pathophysiology of Parkinson's disease — a debilitating, age-related movement disorder. Yet the factors that underpin this loss of neurons are still unclear. Asa Abeliovich and Aaron Gitler use evidence from genetics and model systems to suggest that defective intracellular transport pathways have a large role in the development of Parkinson's disease. Indeed, the accumulation of proteins and cellular components probably exceeds the degradative capacity of the neurons involved. Other mechanisms such as inflammation and prion-like spreading might also contribute to the pathogenesis of the disease.

Asa Abeliovich & Aaron D. Gitler

217 Mammalian prions and their wider relevance in neurodegenerative diseases

Prions are infectious, protein-based agents that cause fatal brain diseases

such as Creutzfeldt-Jakob disease in humans and bovine spongiform encephalopathy in cattle. Composed of self-propagating assemblies of misfolded protein, prions can lead to neurotoxicity and are able to evolve *in vivo*. John Collinge highlights the structure and biological properties of prions. He also investigates how best this knowledge might be applied to understanding the transmissibility of other neurodegenerative diseases in which the deposition of misfolded proteins is a common theme.

John Collinge

227 The activities of amyloids from a structural perspective

Amyloids, the hallmarks of various neurodegenerative disorders, are formed from aggregates of proteins. Their structure is based on pairs of many-stranded and repetitive intermolecular β -sheets that interact to form the cross- β -sheet motif. Roland Riek and David Eisenberg discuss the configuration of several amyloids, revealing how their features enable them to self-replicate and to adapt to their surroundings. In turn, these resulting properties facilitate the transmission of amyloids between cells, as well as their infectivity and toxicity.

Roland Riek & David S Eisenberg