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SHARED MEMORY

If one side-steps the potential health benefits—it reads like the stuff of nightmares: ‘Biologists ‘transfer’ a memory through RNA injection.’ A team from UCLA have published their study where a ‘memory’ has been transferred from a sensitised (trained) marine snail to a naïve (untrained) marine snail. There was a demonstrable increase in excitability in sensory (but not motor neurones) in the recipient molluscs. But more importantly they also exhibited the same sensitisation response to electric shock as the donor snails.

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DO NOT MENTION YOUR DEMENTIA

Big claims from big data need big responsibility and as A Fo Ben mutters often to himself—correlation does not equal causality. So how to navigate the hyped link that prior use of antiepileptic drugs is linked to

an increased risk of dementia? An increased ‘risk’ was seen with those taking drugs with prominent cognitive side effects. Authors could not correct for Berkson’s bias—a form of selection bias where those who are seen in medical clinics for one condition are more likely to be diagnosed with another condition through dint of being under the care of a medic. The authors did not consider that the drugs were unmasking the dementia earlier as the drugs made people decompensate sooner. We await a better study before we claim that antiepilepsy drugs cause dementia.

J Am Geriatr Soc. 2018 [Epub ahead of print]

THE HEART RULES THE HEAD

A Fo Ben was always told that there was a certain type of patient (older, multimorbidity) that ‘*just didn’t do well*’ following cardiac procedures. A study from China may shed some light on this clinical observation. Myocardial ischaemia leads to increased levels of a microRNA miR-1 in the hippocampus and in blood and subsequent neuronal microtubule damage. The authors went on to demonstrate that this damage could be prevented, in mice, if miR-1 was knocked down with a viral vector. This work suggests that silent hypoxia or catch-all decompensation may not be the cause of cognitive decline following cardiac procedures after all.

J Mol Cell Cardiol. 2018;120:12-27.

FIFTY SHADES OF GREY

I rightly receive criticism (criticism that of course I acknowledge and then dismiss) that the

Carphology-Neurology curriculum is too narrow. Affirming this stereotype, A Fo Ben will continue to pick at the ageing literature as I rage against the dying of the light. How to avoid going grey? A study (again in mice) suggests that the process involves both the immune system, viral infection and a transcription factor called MIFT. MIFT suppression of innate immunity genes in hair follicle melanocytes is important for preventing the process of greying.

PLoS Biol. 2018;16(5):e2003648.

LOWEST FORM OF WHAT?

Anyone who has seen a new-fangled ‘compliant’ rolling rota for junior trainees knows that they are a fiendish tool for inflicting sleep deprivation on the young. What might be the impact of this on our trainees? Metabolic syndrome, increased road traffic accidents and mood disorders all pale in to insignificance when one considers this Belgian study. An ambiguous voicemail message left by a friend was then presented, and participants had to decide whether the recipient would perceive the message as sincere or as sarcastic. Reaction times were blunted in excess of the generalised slowing expected from sleep deprivation. The authors conclude that sleep deprivation might damage social interactions by slowing the perspective-taking processes.

PLoS One. 2015;10(11):e0140527.

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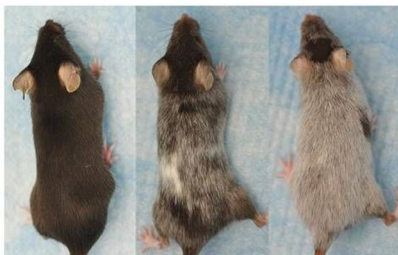


Figure 1 Dorsal images of female litter mates taken at postnatal day 110 demonstrating the variation in greying. Heterozygous transgene mouse (left), homozygous transgene mouse (middle). Heterozygous transgene also haplonsufficient for *Mitf*^{mi-vga9/+} (right).