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Kaleidoscope

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Why do we gossip? Human social networks are typically small and densely interconnected, and the gossip of others within and between these unevenly distributed webs can have an impact on personal reputation. Across three studies of 2419 individuals, Wu and colleagues[1] draw upon an evolutionary framework to assess whether gossip would promote generosity and cooperation. They found that generosity increased when either a recipient or an observer was connected to and could gossip to others with whom participants might later interact. Crucially, it appeared that *reputational concern* mediated gossip-based generosity rather than any expected explicit benefits from such future interactions. Natural selection appears to have shaped psychological mechanisms to identify opportunities to promote a positive reputation, and we are all rather adept at manipulating the universal human propensity to chin-wag. Pass it on: science would suggest that when you gossip you're also being generous and enhancing societal cooperation – just don't tell anyone we told you so.

Breaking bad: are callous–unemotional characteristics in childhood an inevitable precursor of adulthood psychopathic traits? Longitudinal and intervention studies are difficult to perform but Humphreys and colleagues[2] were able to recruit 136 12-year-old Romanian children who had been abandoned at birth and raised in state-run institutions in Bucharest – and 50 matched controls – and randomised those from institutions to either care-as-normal or foster care. Those from the institutions had far greater levels of callous–unemotional traits; however, this was mitigated in boys who were thereafter placed into foster care. Interestingly, it was caregiver *responsiveness to distress*, and not caregiver warmth, which mediated this effect: the authors hypothesise that this former factor might be more specifically linked to empathy, social competence, and self-regulation. It remains unclear why these benefits were not seen in girls – who had equal initial callous–unemotional levels – though other data have highlighted the gender differences in how early-life maternal sensitivity alters the internalising of symptoms. There are extensive personal and societal costs of psychopathy and antisocial behaviour, and the observation that severe early deprivation produces greater degrees of callous–unemotional behaviour is perhaps sadly predictable. It is, however, enormously heartening that high-quality foster care – particularly, sensitive responding to distress – can yield positive change in these very vulnerable young people. No future is predetermined with regard to an individual's mental health.

'Charting the landscape of priority problems in psychiatry' is a bold initiative to undertake. In 1900 the German mathematician David Hilbert proposed an influential list[3] of unsolved priorities in his field, and Stephan *et al*[4,5] take this as their inspiration in highlighting 17 'problems' that need addressing. They argue that our dichotomous syndrome-based diagnostic systems are increasingly frail, and, not being predicated on causal mechanisms, lack the ability to guide treatments, which evolve (slowly) on a trial-and-error basis and largely miss stratification of subgroup responders. There is fundamental uncertainty as to the role and status of symptoms in our unique specialty, particularly the computational mapping between mental states and brain states. They observe the 'frustratingly slow' progress turning advances in epigenetics and neural circuit functioning into either diagnostic procedures or therapeutic approaches: we have very strong epidemiological risk data, but sparse understanding of the gene–environment interplay and systems-level impacts on the brain. In line with the recently discussed ROAMER and DCP-3 statements[6] there is an increasing recognition of the need to understand both preventive measures and the factors underpinning individuals' resilience. Perhaps most grandly, they pose the tantalising question as to whether a closed set of mathematical equations can be elucidated to describe the brain's function. Hodgkin–Huxley equations can explain the firing of a single neuron, but can we model the temporal dynamics of the entire brain? The result would be a Fokker–Planck equation prescribing the interaction of deterministic processes and stochastic fluctuations to generate a prediction of the activity at a population level. Across these two papers every brain level is addressed, from synaptic to circuit, through cognitive to social: the gauntlet thrown down is as impressive as the task is daunting.

As winter settles in, a fascinating paper in last month's *BJPsych*[7] showed that seasonal affective disorder (SAD) had a high prevalence among individuals with severe visual impairment. How effective is bright light treatment in SAD? A randomised controlled trial compared it with a SAD-specific programme of cognitive–behavioural therapy (CBT) (6 weeks' intervention in both cases), with 177 individuals followed up over two successive winters.[8] In September of each year all participants were mailed to prompt them, depending on their randomisation, either to recommence daily light treatment upon developing any depressive symptoms or to utilise the skills learned in CBT. The two interventions didn't differ over the first year, but by the second winter the CBT group had a smaller proportion of recurrence (27.3% compared with 45.6% for light therapy), less severe symptomatology, and greater levels of remission. These data suggest that the psychological and physical interventions are equally effective at managing acute episodes of SAD, but that the gains produced by CBT are more durable.

How specific is bright light treatment to SAD? Lam *et al*[9] have now shown that it had a therapeutic effect on non-seasonal major depressive disorder (MDD). Participants with MDD ($n = 122$) were randomised to one of four conditions: light therapy and placebo medication; placebo light therapy and an antidepressant; light therapy and an antidepressant; or placebo medication and placebo light treatment. Light treatment was a 10 000 lux fluorescent white light box administered for 30 minutes each morning over the 8-week study duration. Bright light treatment was efficacious and well-tolerated in this group, with its combination with medication showing the most robust group effect in both self- and interviewer-ratings. The underlying physiology of this is unproven, but disrupted circadian patterns are an established part of MDD, and light therapy has been shown to help correct this as well as to impact directly upon monoamine levels.

Disorientation in time and space are core cognitive features of Alzheimer's disease, often most vividly displayed as disorganised 'wandering' behaviours. Spatial orientation is a function of the hippocampus and entorhinal cortex with the latter acting as a hub in a wider cortical–hippocampal network. The entorhinal cortex has grid cells that display the remarkable property of firing systematically in a way that accords with a spatial-geometric map formed of equilateral triangles. Better still, aggregates of these grid cells and proxies for their spatial-representation functions can be imaged using functional magnetic resonance imaging (fMRI).

Carrying one APOE-ε4 allele confers a 3-fold risk of developing Alzheimer's disease, and tauopathy can be detected as an early

marker in the entorhinal cortex in carriers at ages less than 30 years. For this reason, Kunz *et al*[10] studied a group of 38 healthy APOE-ε4 carriers (deemed to be 'at risk' for Alzheimer's disease) and compared them with a matched control group of 37 double APOE-ε3 carriers (the low-risk group). While undergoing neuroimaging, study participants freely navigated around a virtual environment, and were presented with object cues at certain spatial locations. They later had to navigate back to the location, to place the object in the location they remembered being cued from. The acquired fMRI data were divided in two, with the first half used to derive grid-like representations in the right entorhinal cortex for each subject. The second half was then used to measure changes in entorhinal cortex representation when movements in the virtual environment where aligned *v*. misaligned to the putative grid representations. Comparing at-risk to controls, robust grid-like representations in entorhinal cortex were found in the control group but not in the at-risk group. Further, at-risk participants showed a behavioural preference for avoiding the centre of the virtual environment. This suggests that the walled edges of the environment were stronger navigational clues for APOE-ε4 carriers that, consistent with the lack of robust internal grid-like representations in the entorhinal cortex, enable compensatory navigation in spatial environments. By examining the fMRI BOLD activity of the hippocampus, the authors were able to show a compensatory increase in hippocampal activity inversely proportional to the level of entorhinal cortex grid-cell-like representations, the correlation being larger in at-risk than control individuals. The data demonstrate behaviourally relevant brain dysfunction in at-risk individuals decades before the potential onset of Alzheimer's disease.

Finally, we have been impressed by Pennycook *et al*'s treatise 'On the reception and detection of pseudo-profound bullshit'.[11] The authors note that most philosophical work to date has been concerned with the motivations of the *bullshitter*, whereas they were most interested in the factors that predispose or protect one from becoming a *bullshittee*. Undergraduate participants ($n = 280$) completed a battery that included cognitive tasks to determine their analytic style, a heuristics and biases battery of reflective thinking, and an ontological confusions scale. They were then presented with randomly arranged but syntactically correct buzzwords in what were labelled 'bullshit statements', for example 'Hidden meaning transforms unparalleled abstract beauty'. There were three follow-on arms, including a second study that tested real tweets from Deepak Chopra's Twitter account on approximately 200 members of the public. Overall, the authors found that a susceptibility to bullshit was reliably associated with an intuitive cognitive style and supernatural beliefs; interestingly, the protection against bullshit was the discernment of deceptive vagueness – rather than an indiscriminate scepticism per se. Our particular bullshit bête noire has been the proliferation of 'inspirational' and 'motivational' postings (would 'motspirational' count as neologistic bullshit?) on LinkedIn and Facebook; and closer to home, the scientific literature is also not immune from this influence. Perhaps we shouldn't worry too much – we ended up having fun with one of the online bullshit generators[12] – and, after all, as Pennycook and colleagues remind us, 'A wet person does not fear the rain'.

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7 Madsen HO, Dam H, Hageman I. High prevalence of seasonal affective disorder among persons with severe visual impairment. *Br J Psychiatry* 2016; **208**: 56–61.

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12 http://sebpearce.com/bullshit/

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