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Kaleidoscope

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March 20th was the United Nations International Day of Happiness (http://www.dayofhappiness.net). What makes you happy; as you ponder pending election choices, does your political persuasion affect your affect? The story goes that politically conservative people are happier – at least on self-reports. Wojcik *et al*[1] use big data to try and understand what the real differences between liberals and conservatives are. They show that, in fact, self-deceptive enhancement mediates the association between happiness (or, at least, life satisfaction) and political ideology. Taking samples of text from the US Congressional Record, content analysis for negative and positive language (using the PANAS-X scale) determined a small but significant effect between conservatism (by voting record and party membership) and a decrease in positive-affect words ($\beta = -0.16$, $P < 0.001$). Perhaps, however, your face really betrays your true happiness. Duchenne smiling is the facial expression where the corners of the mouth (zygomatic major) lift in tandem with those around the eyes (orbicularis oculi) and supposedly represents 'genuine' smiling; non-Duchenne smiling is where only the mouth corners rise, and is proposed to be essentially fake (the quintessential 'Pan Am smile'). When facial images from the US Congress equivalent of a yearbook (the Congressional Pictorial Directory of the 113th US Congress) were analysed, it transpired that that conservatives used the eye muscles (i.e. Duchenne smiling) significantly less than liberals, but did not differ in the mouth corners. Kaleidoscope did its own facial expression analysis of the main UK party leaders to help inform our voting; something that readers may wish to try for themselves, as scrupulous impartiality forbids us from releasing our results.

The first comprehensive meta-analysis quantifying mortality across mental health disorders[2] has confirmed what many of us will have suspected, but lacked evidence for – that they are among the most substantial causes of death worldwide. Unpicking morbidity and mortality data is difficult as most individuals do not die as a direct consequence of their mental illness, and present with mediators such as greater rates of smoking and lack of exercise. In this work the overall mortality pooled relative risk was 2.2: a median 10 years of potential life was lost, with approximately 8 million deaths per annum attributable to mental ill-health. The mortality risk for psychoses was significantly higher than for major affective, bipolar affective, and anxiety disorders, and in-patient samples had worse outcomes than community samples; no differences were determined for sample source, diagnostic system utilised, or global geographical location. 17.5% of deaths were due to 'unnatural' causes, including suicide; this is striking and it also reinforces the comorbid physical health needs of those with mental health problems, with 67.3% of the *additional* deaths being due to 'natural' causes such as cardiovascular disease.

Within medicine, the stresses of medical training and clinical life are well recognised, and will resonate with the readership of the *BJPsych*; data from the US show that an average of one doctor dies by suicide every day, with increased rates compared with the general population (by 1.41 and 2.27 times, in men and women respectively). Despite an active discussion on the roles of stress and a machismo culture in medicine as contributors to suicide, this has not led to a concerted advocacy for proactive screening for physician burn-out. An editorial in *JAMA Psychiatry*[3] draws an interesting parallel with the US Air Force, which determined in the 1990s that suicide was an institutional problem and introduced a programme that included awareness and prevention training, champions to identify and funnel at-risk individuals, and induction and follow-up mental-health screening questionnaires: suicide rates fell from 16.4 to 9.4/100 000 within 4 years. The authors compellingly argue that our profession – not limited to psychiatry – needs similar, nationally driven, models, and make recommendations for a targeted response to this issue, incorporating appropriate education, screening and treatment.

The late film critic Roger Ebert wrote that 'for most gamers, video games represent a loss of those precious hours we have available to make ourselves more cultured, civilized and empathetic' – a sentiment anyone who has personally, or by proxy through their family, lost valuable days to Tetris or Angry Birds may endorse. Today's multi-million dollar immersive virtual worlds make the video games of the early 1980s look like primitive cave paintings. Back in 1983, when Demon Attack was game of the year on the generation-defining Atari 2600, one of the first academic studies on gaming showed improvement in skilled visuo-motor performance in 31 exposed individuals. Now, 49 Atari 2600 console games are the testing battery for a new biologically inspired method for visuo-motor skilled learning by computers. In *Nature* Mnih *et al*[4] show how combining hierarchies of artificial neural networks (in the style of Hinton & Salakhutdinov's deep learning networks[5]) can take raw visual input (from video capture of the Atari console's screen) and generate motor output in such a way that it wins the games. Sounds trivial, but as the futurist Hans Morovec noted, we can emulate the 'thinnest veneer' of human performance such as high-level deliberate reasoning in playing chess or automatically proving theorems in algebras, but cannot engineer anything with even the most elementary sensorimotor skill of an infant. The hierarchical architecture of human cortex (which these models emulate) facilitates iterative levels of coding and recoding of sensory information leading to a more compressed but task-oriented representation with irrelevant 'nuisance' information filtered out. These networks learn to approximate a variant of a reinforcement learning model so well, that on 29 of the 49 games, the algorithm outperforms expert human players – including on the classic Demon Attack.

James Joyce captured it in a single word: m'm'ry. More prosaically, mild cognitive impairment (MCI) incorporates modest but discernible decay in memory functioning, and can herald the beginning of Alzheimer's disease. However, such progression is far from inevitable, and there has been much work trying to elucidate which factors in MCI might be prognostic of this outcome. Currently, there are three major sets of classification guidance for MCI cohorts, with varying criteria and biomarker usage, which stratify individuals into a likelihood of having Alzheimer's disease. Direct evaluation between them had been lacking, but a large international collaboration[6] has now compared their utility in over 1600 individuals with MCI from 13 cohorts (766 of whom had both amyloid and neuronal injury markers). While there were minor variations between the guidelines, all were effective in delineating populations into higher- and lower-likelihood groups for progression to Alzheimer's disease. The authors propose that their findings support the use of these research criteria in identifying Alzheimer's disease at the MCI stage, and that clinically the use

of both amyloid and neuronal injury markers offered the most accurate prognosis.

Once Alzheimer's disease is present, can symptom profile predict the rate of further progression? Yes, according to Peters *et al*[7] who followed up 335 patients with incident Alzheimer's disease; psychosis, agitation/aggression and any one clinically significant neuropsychiatric symptom were associated with a more rapid progression to severe dementia, while the same factors and affective symptoms were also associated with earlier death. Most of the healthcare costs of Alzheimer's disease are due to the long-term care needs of those at the severe end of the illness spectrum; any delay in progression would have huge economic benefit, as well as the more important aspect of giving more meaningful time to sufferers and their loved ones. It remains to be seen whether the treatment of specific neuropsychiatric symptoms in earlier illness phases can delay the progression of this disease.

What makes you 'believe' scientific evidence and which factors might sway your opinions? Are candidates the journal impact factor, study methodology, or prior knowledge of the leading authors? The inclusion of brain imagery or a nonsense mathematical statement has been shown to improve the perception of the quality of work (though Stephen Hawking's editor famously warned him that each (genuine) equation halved the number who would actually read it). Now a fascinating experiment by Fernandez-Duque *et al*[8] suggests that superfluous neuroscientific information enhances the judged explanations of psychological phenomena, regardless of the quality of the underlying data or argument. In the 385 tested college students – most of whom were majoring in psychology – the effects were not due to the length of explanation and were specific to neuroscience; additional irrelevant social science data had significantly less impact, as, interestingly, did unnecessary information from 'hard sciences' such as maths, computer science and physics. An analytical thinking style did not protect against this 'allure of neuroscience' bias, and the authors offer the argument – slightly paraphrased here – that perhaps the brain may be prone to the predisposition that it is the best explainer of mental phenomena, a so-called brain-as-engine-of-mind hypothesis.

Finally, a quarter of a century after the birth of its last iteration, the 11th edition of the International Classification of Diseases (ICD) is just around the corner – due 2017. Field trials are underway on a purportedly more uniform structure aimed at eliminating some of the more uneven aspects of ICD-10, particularly with regard to differential diagnoses, that have attracted much opprobrium (compare the many possibilities of F40–48 on neurotic, stress-related and somatoform disorders with, say, F20–29 schizophrenia and related disorders).

A World Health Organization update report[9] has noted that the relative infancy of understanding of the aetiology of mental illnesses means that clinical utility will organisationally continue to supersede neuroscience in the new edition. However, more consistent 'parent' and 'child' categories will occur: an example provided is that the grouping 'body focused repetitive behaviour disorder' will have the parent category of 'obsessive–compulsive and related disorders', and constituent children of excoriation disorder, trichotillomania, and other body-focused repetitive behaviour disorder, each having functional, temporal and severity qualifiers. There will be a general removal of a 'minimum number' of diagnostically necessary symptoms, better conforming (it is argued) to the more flexible way clinicians make diagnoses. Two other interesting introductions include a 'boundary with normality' section that will guide on delineating from similar but non-pathological presentations (for example, with anxiety), and a 'boundary with other conditions', set to help consider demarcating from differential diagnoses. Further, 'course features' will outline typical illness progression, and 'culture-related', 'developmental presentation' and 'gender-related' sections will help account for potential population variances.

There is an opportunity for you to contribute; the WHO invites you to join the field study at www.globalclinicalpractice.net. Some concern has been evident in the Kaleidoscope team that this recourse to global clinical practice may be to the detriment of previous diagnoses, for example W28, 'contact with a powered lawnmower'; subcoding of .2 indicating that this had occurred in an institution such as a dancehall, court-room or opera-house[10] (with the exclusion criterion that the building could not be under construction). This level of granularity always arouses Duchenne smiles in us, and makes the wait for ICD-11 seem worthwhile.

1 Wojcik SP, Hovasapian A, Graham J, Motyl M, Ditto PH. Conservatives report, but liberals display, greater happiness. *Science* 2015; **347**: 1243–6.

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3 Goldman ML, Shah RN, Bernstein CA. Depression and suicide among physician trainees. Recommendations for a national response. *JAMA Psychiatry* 4 Mar 2015 (doi: 10.1001/jamapsychiatry.2014.3050).

4 Mnih V, Kavukcuoglu K, Silver D, Rusu AA, Veness J, Bellemare MG, et al. Human-level control through deep reinforcement learning. *Nature* 2015; **518**: 529–33.

5 Hinton GE, Salakhutdinov RR. Reducing the dimensionality of data with neural networks. *Science* 2006; **313**: 504–7.

6 Vos SJ, Verhey F, Frolich L, Kornhuber J, Wiltfang J, Maier W, et al. Prevalence and prognosis of Alzheimer's disease at the mild cognitive impairment stage. *Brain* 17 Feb 2015 (doi: 10.1093/brain/awv029).

7 Peters ME, Schwartz S, Han D, Rabins PV, Steinberg M, Tschanz JT, et al. Neuropsychiatric symptoms as predictors of progression to severe Alzheimer's dementia and death: the Cache County Dementia Progression Study. *Am J Psychiatry* 13 Jan 2015 (doi: 10.1176/appi.ajp.2014.14040480).

8 Fernandez-Duque D, Evans J, Christian C, Hodges SD. Superfluous neuroscience information makes explanations of psychological phenomena more appealing. *J Cogn Neurosci* 12 Nov 2014 (doi: 10.1162/jocn_a_00750).

9 First MB, Reed GM, Hyman SE, Saxena S. The development of the ICD-11 Clinical Descriptions and Diagnostic Guidelines for Mental and Behavioural Disorders. *World Psychiatry* 2015; **14**: 82–90.

10 http://apps.who.int/classifications/icd10/browse/2015/en#/XX.

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