

## Kaleidoscope

Derek K. Tracy, Dan W. Joyce,  
Sukhwinder S. Shergill

**Assortative mating is the non-random selection of a partner: do we all choose our significant other for their combination of qualities – and their ‘fit’ with our own – and if so, does one’s mental health influence this decision?** Nordsletten and colleagues<sup>1</sup> report on the first study looking at assortative mating and psychiatric disorders in a representative population sample (of over 700 000). Compared with the general population, the odds of having a partner who also suffers from a mental health problem – both within and across disorders – were significantly elevated: cross-assortative mating was approximately 0.15 for bipolar disorder, 0.36 for substance misuse, and over 0.40 for attention-deficit hyperactivity disorder, autism spectrum disorder (ASD) and schizophrenia. Overall, having one of these disorders was associated with an approximately two- to threefold increase in the odds of having a mate with the same or an alternative mental health condition. Interestingly and importantly, such cross-assortative mating was *not* found for a range of *physical* health conditions, including diabetes, rheumatoid arthritis and multiple sclerosis.

We can all picture couples who look alike and share personality characteristics; indeed, this attracts (if that’s the correct word) a curious voyeuristic quality in us all. Yet in a linked editorial Plomin *et al*<sup>2</sup> note that compared with the above data, the correlations for physical and personality traits are relatively low at 0.20 and 0.10 respectively (IQ level remains the characteristic a couple are most likely to share). These findings may help explain the generally high heritability (and comorbidity) of mental ill health, despite an often negative impact on fertility, as well as some of the variation between conditions.

**Mental health difficulties in childhood are predicted by both nature and nurture.** Most work on parental depression has evaluated women in the post-partum period, with less interest in the fathers’ symptoms or prospective monitoring of longitudinal changes in mental state as predictors of child outcomes. Narayanan & Nærde<sup>3</sup> challenge this, monitoring depressive symptoms in both parents over the 48 months after their child’s birth, and evaluating the child’s behaviour at this 4-year end-point. They found that mothers’ – but not fathers’ – depressive symptoms at 6 months predicted more emotionally reactive, anxious and withdrawn children with aggressive behaviour at 4 years. However, *changes* in fathers’ symptoms across time were a specific predictor of childhood aggression. There were few differences between boys and girls, with both more influenced by their mothers.

Stover *et al*<sup>4</sup> addressed the so-called ‘spillover hypothesis’ that states that childhood aggression can result from the ‘spillover’ from marital hostility and hostile parenting. Most existing studies have looked at cross-sections of adolescent samples, but here a longitudinal follow-up design in young children (to age 6) was utilised; uniquely, these had all been placed with non-relative adoptive families, allowing a potential disentanglement of genetic and environmental factors. They found that early marital conflict (when the child was aged 18–27 months) was associated with a spillover to a more hostile parenting style. Hostile parenting by an *adoptive father* (but not mother) was associated with children’s

aggression at aged 4 years; interestingly, antisocial traits in the birth mother did not have any effect on child aggression. Both of these studies commend appropriate support and input to parents undergoing difficulties during their children’s early years.

**Human society is not unique in its punishment of offences. However, its dispensation of justice by a ‘third party’ unaffected by the societal violation – such as by a judge – is exceptional.** This third-party punishment (TPP) undoubtedly promotes cooperation by deterring offending, but is costly to the individual – think of a juror who has to sacrifice their time – so what evolutionary advantage would compensate the price of meting out justice upon others? Jordan *et al*<sup>5</sup> argue that when people punish others via TPP they implicitly signal a ‘trustworthiness’ to other group members that increases their reputation in the wider group. In essence, if you observe someone punish selfish behaviour, you infer that this person will not be selfish in any interactions with you.

Testing this, they assigned participants – who were anonymous to and did not see each other – to one of two roles in a TPP game: ‘signaller’ or ‘chooser’. The signaller was given some money and entered into one of three conditions: playing a ‘punisher’ (where they could pay varying sums of their money to penalise the action of a digresser whose actions had not affected them); playing a ‘helper’ (giving money to another party); and playing both. Irrespective of condition the chooser passively observed the action. The protocol then moved to a trust game where the chooser had to decide how much to trust the signaller, by deciding how much money to give them; this amount was tripled and given to the signaller, before the signaller returned a proportion of their money back to the chooser. The signaller benefited from being trusted with the chooser’s money, but the chooser would only earn money if the signaller truly was trustworthy and made a choice to return their money. The amount of money given by the chooser signalled how much trust-reputation had been established. In the punishment-only condition, choosers tended to give significantly more money to signallers who had punished than those who had not. This appears to truly reflect a trust-worthiness signal, because signallers who had punished also returned more of the money to the chooser. This contrasted with the help–punish condition, where signallers punish less and choosers only trusted these punishers slightly more than non-punishers. When helping was available, the results reflected those of the punishment-only condition in terms of money invested by the chooser and returned by the signaller in the trust game: the authors argued that because helping (or not) were the only options, altruistic behaviour became the signal of trustworthiness.

In an editorial in the *New York Times*<sup>6</sup> the same authors describe how we are all third-party punishers in everyday life, where this can manifest as *moral outrage*: we seemingly selflessly denounce bad behaviour and advocate justice in instances that don’t necessarily affect us (they give as an example the widespread indignation at Donald Trump’s egregious call to ‘ban Muslims’ from entering the United States). They propose that in such instances we are actually really advertising our own trustworthiness to others, though they note that it’s an evolutionary hypothesis rather than a test of conscious motivation. The authors conclude their paper in *Nature* with the observation that ‘sometimes punishing wrong-doers is the best way to show that you care’.

**Neuroinflammation is a core feature of Alzheimer’s disease, although this often attracts less attention than amyloid-beta plaques and tau neurofibrillary tangles.** Such inflammation is typified by activated microglia and reactive astrocytes accumulating around the plaques in an attempt to remove them,

but their persistence leads to a chronic and toxic immune response. There are data from healthy adults demonstrating that the colony-stimulating factor 1 receptor (CSF1R) on microglia is essential for these cells' survival, and pharmacological inhibition of the receptor leads to elimination of most central nervous system microglia. Spangenberg *et al*<sup>7</sup> tested the role of CSF1R in a mouse model of dementia. One month of receptor inhibition did not affect amyloid-beta levels or plaque load – or significantly alter astrocyte numbers – but it did lead to a reduction in inflammation, and 80% of the microglia were eliminated. Crucially, this prevented dendritic spine and overall neuronal loss, and behavioural testing showed improvements in memory. The involvement of microglia adds to interesting data noted in last month's Kaleidoscope<sup>8</sup> that showed they had a role in pathological synaptic pruning in schizophrenia. The current findings support inflammation and microglial activation being behind at least some of the neuronal loss seen in Alzheimer's dementia – fascinatingly, apparently independent of the plaque and tangle pathology – and offer up a potential novel therapeutic target for humans.

**The 'extreme male brain' (EMB) hypothesis of ASD has attracted debate but also empirical support. What about an 'extreme female brain' (EFB)?** Baron-Cohen's empathising–systematising model posited that we have evolved complimentary affective–cognitive systems, with ASD an excess (the EMB) of the typical male preference for systematising. This also allowed for a possible EFB that would manifest as more challenged by scientific and logic systems, but with an above average ability to empathise – it was suggested that an EFB was far less likely to appear 'disabled' in contemporary Western societies. Dinsdale and colleagues<sup>9</sup> (a mixed-gender team) meta-analysed studies evaluating the Reading the Mind in the Eyes test, an advanced evaluation of theory of mind, across a range of psychiatric disorders. Their findings indicated that increased empathising mediated the risk and expression of borderline personality disorder and non-clinical depression, with a clear female bias. Such individuals showed increased attention to social stimuli, with greater degrees of social and emotional sensitivity, negative emotion biases, and overdeveloped mentalising behaviour. There is a jarring quality to the findings, perhaps as they run counter to our desire for gender equality and seem to tread dangerously close to the arena of stereotype. As with the extreme male brain, the debate is far from over.

**Great literature expands our minds: Willems & Jacobs provocatively propose<sup>10</sup> that neuroscientists should therefore 'care about Dostoyevsky'.** Evaluation of high art has remained the preserve of the humanities; can a world of neuronal depolarisation and neuroimaging really add to our cultural ennoblement and scientific understanding? Is the neurocognitive study of literature even possible, let alone desirable? The authors say yes, and put forward several arguments in favour of this. In neuroimaging, mental simulation of emotions and motion taken from an entire fictional piece has demonstrated an apparent ecological validity through greater activation of relevant brain regions than their constituent sentences taken in isolation; in children, functional near infra-red spectroscopy (fNIRS) and short fiction narratives have been utilised to evaluate mentalisation and empathy. Immersion, the vicarious feelings that fictional characters produce in us (and the reason we read), has proven amenable to objective evaluation, demonstrating activation of the mid-cingulate cortex that is implicated in affective empathy.

There is something about the joining of the two cultures that pleases (and pleases long) – arguably why we became psychiatrists rather than training in surgery. But to paraphrase the aforementioned empathising–systematising model, is this a paradoxical scientific attempt to systematise empathy, with a Wildean danger of knowing the price of everything and the value of nothing? As for the master, Dostoyevsky counselled that 'It is not the brains that matter most, but that which guides them – the character, the heart, generous qualities, progressive ideas'.

**Finally, concentration camp syndrome ('KZ-syndrome' from the German *Konzentrationslagersyndrom*) is the name applied to the somatic and psychological sequelae seen in many Holocaust survivors.** Originally labelled 'Buchenwald syndrome' by the psychiatrist Paul Friedman in 1948,<sup>11</sup> it encompasses a range of symptomatology similar to that seen in the severest multi-trauma post-traumatic stress disorder, including very complex survivor guilt. Jabłoński and colleagues<sup>12</sup> undertook a retrospective assessment of such ex-prisoners' health 5 ( $n=250$ ) and 30 ( $n=120$ ) years after their liberation: rates of KZ-syndrome were 58.8 and 77.5% at the respective time-points. Not only had most victims suffered from this, the rate *increased* with time, with a growth in the number of psychological and physical morbidities and the intensity of the symptoms. Sadly, such atrocities are not restricted to the Nazis: forced population detentions, slavery, and ethnicity-based killings have all too contemporary a feel in 2016. We remember the words of Auschwitz survivor Primo Levi on 'who dies because of a yes or a no': 'meditate that this came about'.

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