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Kaleidoscope

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Few topics seem to generate as much heat – frequently without the illumination of evidence-based light – as the heritability of intelligence. Samuel Johnson said 'Such is the delight of mental superiority that none on whom nature or study have conferred it would purchase the gifts of fortune by its loss'; for the modern reader this captures the importance of both nature and nurture, and also touches on, perhaps with slight discomfort, a contemporaneous social sensitivity to inferred elitism. An expert review by Plomin & Deary[1] notes how intelligence is one of the most heritable behavioural traits as well as being among the best predictors of occupation, mental and physical health, and mortality outcomes. Among what they label the special findings of the genetics of intelligence, they describe how the heritability of intelligence increases dramatically from approximately 20% in infancy to 60–80% in later adulthood: the authors posit that although individuals clearly have genetic stability, they select, modify and create environments that are correlated with their abilities, resulting in a so-called genetic amplification with time. Assortative mating – having a partner with similar traits to oneself – is greater for intelligence than for other behavioural traits such as personality and psychopathology, or physical traits such as weight and height. Intelligence has a normal population distribution: the authors' statement that the exceptional end of performance is a model for studying 'positive genetics' is reasoned and reasonable, but sharply cuts back to the image problem this field historically has had, echoing Johnson's 'mental superiority'. Clear heads, as well as intelligence, are needed.

The powerful influence of environmental manipulation on cognitive, language, motor and socio-emotional development in infancy is highlighted in a stimulation and nutrition study reported in the *Lancet*.[2] Children receiving nutritional enhancement demonstrated generally superior developmental scores at 12 months but not at 24 months; whereas children receiving responsive stimulation through guided developmental play activities showed superior developmental scores across the entire gamut of cognitive, language, motor and socio-emotional scales at 12 months and most of these were maintained at 24 months. Interestingly, children receiving a combination of these two interventions did not show additive benefits. The article concludes that response stimulation has a positive impact on developmental outcomes, even above nutritional enhancement, in the context of a poorer nation's healthcare system.

With larger sample sizes, are the genetics of schizophrenia becoming clearer? That may depend on which side of a recent debate you find more convincing. In a large genome-wide association study of patients with schizophrenia (characterised by clinical symptoms) and healthy controls, Arnedo *et al*,[3] identified 42 single-nucleotide polymorphism (SNP) sets associated with a $\geqslant$70% risk of schizophrenia, and found 17 networks of SNP sets that did not share any SNP or individual. The authors argue that previous genetics research was frequently weakened by a binary categorisation of individuals having/not having schizophrenia, and conclude that 'the schizophrenias' are a group of heritable disorders caused by a range of distinct genotypic networks associated with different clinical syndromes. The online critique by Breen *et al* in PubMed Commons[4] challenged this, arguing that the authors failed to adequately address a number of issues, including: the bias of stratification of population ancestry; the issue of gender (15 of 237 SNPs being on the X chromosome); linkage disequilibrium (a correlation of SNPs that are physically close together in the genome); statistically inappropriate selection of SNPs; and a lack of clarity about the replication of their findings. The discussion remains active as the authors of the original work have responded to the critique, and stand by their findings as novel data-driven analysis that affords a mechanism by which to uncover complex genotypic–phenotypic relations when they are present, without having *a priori* assumptions.

Social interactions can be difficult for many with schizophrenia, but how much attention do we, and should we, pay to such behaviour – and our reactions – in our clinical encounters? The intuitive and immediate *Praecox-Gefühl* or 'praecox feeling' of social inaccessibility coined by H. C. Rümke in the 1940s to encapsulate many psychiatrists' rapid (diagnostic) sense of the 'definitely incomprehensible' and 'impossibility of empathy' has been widely criticised as unreliable, heavily subjective and fundamentally unscientific, although it may be integrated into the clinical picture by many clinicians.[5] Lavelle *et al*[6] videoed the non-verbal communication of both patients and psychiatrists in 40 out-patient reviews, and found that patients with schizophrenia could be classified into two groups based on non-verbal behaviour that was stable across the appointment: a larger group of those displaying pro-social behaviour inviting interaction; and those demonstrating flight behaviour to avoid interaction. Psychiatrists adapted their behaviour to the patient, interacting more socially with patients who also displayed such behaviour. A pro-social patient profile was associated with a significantly reduced symptom severity, greater satisfaction with communication, and a positive therapeutic relationship. Clearly, the process of social interaction during consultations matters, gives us important clinical information, and has an impact on outcomes.

In the US sitcom *Seinfeld* George Costanza explained to his friend Jerry: 'Just remember, it's not a lie *if you believe it*'. Is honesty really all social conditioning, and how might this behaviour be moderated by frontal lobe neural networks? In Zhu *et al*'s *Nature Neuroscience* paper,[7] people with acquired and circumscribed lesions to the orbitofrontal (OFC, $n=7$) and dorsolateral prefrontal (DLPFC, $n=7$) cortices, regions considered critical to decisions about honesty, were recruited along with 27 healthy, non-brain-injured controls. They undertook a task wherein a signalling participant messaged an anonymous recipient: the signaller chose to send either an honest altruistic message ('option B will earn you more than option A' – and, unbeknownst to the recipient earned the signaller less) – or a dishonest one ('option A will earn you more than option B' – which actually earned the signaller more). Importantly both were informed that only the signalling participant knew the financial consequences of the choices, and the recipient had no way of inferring their honesty. The reward amounts varied, creating two possible conditions for each trial: a 'conflict' trial, where honesty and altruism would prevail over economic self-interest; and 'no-conflict' trials where being honest benefitted both parties. People with DLPFC lesions were less concerned with honesty, favouring lower rewards for the recipient in the conflict trials; but were similar to healthy participants and those with OFC *et al.* lesions in the no-conflict trials. The authors suggest that the DLPFC is involved in controlling and curbing self-interest in the pursuit of honesty.

Finally, for some the link between mental illness and brain function remains controversial; particularly how psychosocial events causally affect the workings of an internal organ. Taking mood disorders as an example, we might see the core requirements as two-fold: first, the neural mechanism must be sensitive to specific insults (e.g. stress-provoking life events); and second, it must be implicated in a network which includes areas thought to be responsible for the behaviours seen in the syndrome (e.g. affective state evaluation in the amygdala, hypothalamic dysregulation of sleep and appetite, the hippocampal storage and retrieval of episodic memory).

The habenula is a small thalamic structure that receives input from the hippocampus, amygdala, hypothalamus and the basal ganglia – structures associated with monoaminergic neurotransmission – which makes it a candidate for a central role in mediating between reward processing, memory, emotion, endocrine and circadian systems. It is activated by negatively valenced events, and hyperactivity in the habenula, induced by excitatory input, has been linked with depression. Shabel *et al*[8] shed light on the neurochemical control of activity in the habenula. They found, in a rat model of depression, that inhibitory gamma-aminobutyric acid (GABA) is co-released with excitatory glutamate in the lateral habenula when directly stimulated from the basal ganglia. This 'dual release' mechanism provides internal regulatory control; however, when the balance shifts towards an increased excitatory state characterised by increased glutamate release, with reduced inhibitory GABA, the habenula becomes overactive (as evidenced in models of depression). Further, they showed that administration of citalopram increased the release of inhibitory GABA at the lateral habenula/basal ganglia input synapses, normalising the influence of excess excitatory glutamate. Kaleidoscope was curious about rodent models of depression: a quick Google search returned some interesting hits, notably, the ratfanclub.org, where a section 'Understanding grief when a rat dies' provided guidance for how to manage rodents' difficulties dealing with kinship loss.

1 Plomin R, Deary IJ. Genetics and intelligence differences: five special findings. *Mol Psychiatry* 16 September 2014 (doi: 10.1038/mp.2014.105).

2 Yousafzai AK, Rasheed MA, Rizvi A, Armstrong R, Bhutta ZA. Effect of integrated responsive stimulation and nutrition interventions in the Lady Health Worker programme in Pakistan on child development, growth, and health outcomes: a cluster-randomised factorial effectiveness trial. *Lancet* 2014; **384**: 1282–93.

3 Arnedo J, Svrakic DM, del Val C, Romero-Zaliz R, Hernandez-Cuervo H, Molecular Genetics of Schizophrenia Consortium, et al. Uncovering the hidden risk architecture of the schizophrenias: confirmation in three independent genome wide association studies. *Am J Psychiatry* 15 September 2014 (doi:10.1176/appi.ajp.2014.14040435).

4 http://www.ncbi.nlm.nih.gov/pubmed/25219520#cm25219520_6388 (accessed 4 Nov 2014).

5 Ungvari GS, Xiang Y-T, Hong Y, Leung HCM, Chiui HFK. Diagnosis of schizophrenia: reliability of an operationalized approach to 'praecox-feeling'. *Psychopathology* 2010; **43**: 29–9.

6 Lavelle M, Dimic S, Wildgrube C, McCabe R, Priebe S. Non-verbal communication in meetings of psychiatrists and patients with schizophrenia. *Acta Psychiatr Scand* 6 August 2014 (doi: 10.1111/acps.12319).

7 Zhu L, Jenkins AC, Set E, Scabini D, Knight RT, Chiu PH, et al. Damage to dorsolateral prefrontal cortex affects tradeoffs between honesty and self-interest. *Nat Neurosci* 2014; **17**: 1319–21.

8 Shabel SJ, Proulx CD, Piriz J, Malinow R, et al. GABA/glutamate co-release controls habenula output and is modified by antidepressant treatment. *Science* 2014; **345**: 1494–8.

poems by doctors

Aquiline

Jacob Louis Freedman

You ask me if I believe in resurrection
And I gracefully evade your question
It's not that I don't but rather that I fear you'll be upset
If I don't appreciate your centrality in the process

Dancing through the local graveyard hasn't tired you out
And you dodge my questions too
You do not wish to tell me why you perch on the hospital bed
Aquiline and fixated on some distant prey

When I ask you what you're looking at
You tell me *The Universe*
And yet this big place is just far too small
To keep the nurses from staring back at you

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