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Kaleidoscope

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Social bonding and a need to belong are core human characteristics, with phylogenetically ancient roots from our distant past. We typically prefer companionship to solitude, even when there are no direct benefits from being in company. Wagner *et al*[1] tested the effects of shared emotional experiences between friends: pairs – one having a neuroimaging scan while the other was separated in a waiting area – were both shown emotionally negative, positive and neutral images, and were told if their companion was simultaneously seeing the same picture or not. Both subjective reports of their emotional response and activation of their brain reward circuitry were greater during shared emotional trials, whether negative or positive, even in the absence of any communication or interaction. Shared experience appears to amplify our emotional responses.

Less desirable social interactions are evident in people exhibiting a callous disregard for others through psychopathic and antisocial traits; and children with an early emergence of such behaviour have high risks of adult dysfunction. Milton said 'the childhood shows the man, as morning shows the day', but is this trajectory fixed, or amenable to modification? A recent study[2] reported on a long-term programme to develop social competencies in almost one thousand kindergarten children with conduct problems randomised by school cluster into a 10-year intervention or a control group. The active arm included social skills training, parent behaviour-management teaching with home visits, peer coaching, reading tutoring, and social-emotional curricula in the classroom. Although psychopathology and rates of crime were high in both groups at age 25, there were significant reductions in those who received the additional care. The expense of such programmes – in this instance US$58 000 per child over 10 years, with a number needed to treat of eight to reduce 'caseness' by one – must be weighed against estimated untreated lifetime opportunity costs of US$2.6–5.3 million *per individual* in terms of incarceration, criminal justice, treatment and victims' costs.

Theory of mind, or mentalising, is a crucial aspect of social bonding involving making sense of, and predicting the thoughts, emotions and actions of others, and is a critical area of deficit in autism spectrum disorders (ASD). The neural peptide oxytocin, a hormone that is ordinarily involved in parturition, lactation and parental bonding, has garnered attention as a putative therapeutic agent in ASD. Work to date has typically evaluated emotion recognition improvements in paradigms with direct cues such as facial expression or prosodic elements of language. A double-blind cross-over randomised controlled trial[3] of 20 men with ASD utilised a novel design removing such cues to distinguish the social *beliefs* of others – which individuals with higher-functioning ASD can learn – from their social *emotions*, which remain particularly problematic to interpret. Intranasal oxytocin produced a significantly increased accuracy in inferring the social emotions of others (with concomitant enhancement of activation in the right anterior insula) and the authors argue that this specific change underlies the hormone's therapeutic effects in this group.

Data are also emerging that oxytocin's therapeutic roles might go beyond those with deficits in emotion recognition. A contemporary neural model posits that anxiety disorders are due to a lack of inhibitory amygdalar tone during the acquisition of Pavlovian fear responses, combined with deficient encoding of extinction responses in amygdala–medial prefrontal cortical circuits. Eckstein *et al*[4] randomised 62 healthy male volunteers to receive either oxytocin or placebo after undergoing a fear conditioning task, and demonstrated that the hormone inhibited amygdalar and enhanced extinction responses. This has yet to be tested in a relevant clinical population, but it offers an illustration of a potentially new pharmacological route in anxiety disorders and post-traumatic stress disorder.

Hunger heightens reward sensitivity – an evolutionarily sensible adaptation – yet starvation states do not drive those with anorexia nervosa to eat. Wierenga *et al*[5] used a delay discounting monetary decision task to differentiate immediate reward processing from cognitive control in 23 women remitted from anorexia nervosa – and 17 matched healthy female controls – during hungry and satiated states. As well as hunger increasing reward salience, satiety increased cognitive control circuitry in the control group; but there were no differences noted in the remitted anorexia nervosa cohort, who showed greater cognitive control regardless of their metabolic state. These findings might underlie the capability to restrict food consumption in anorexia nervosa and, furthermore, the impaired determination of emotional salience could potentially contribute to the reduced motivation to engage with treatment, anhedonia and the lack of insight commonly seen in this condition. Weight gain is the all too familiar problem in psychotic illnesses, with numerous contributing factors, including antipsychotic medication. The STRIDE programme[6] assessed the effectiveness of an active intervention programme consisting of moderate caloric reduction, a 'DASH' (dietary approaches to stop hypertension) diet and enhanced physical activity, over 6 months. Those in the active arm lost 4.4 kg more than the treatment-as-usual group over 6 months; 2.6 kg more over a year; they experienced fewer medical hospital admissions and improved fasting glucose levels. Unlike comparable recent studies, this work was in a community setting, and should encourage us that weight gain in psychosis need not be a hopeless inevitability.

Alien control phenomena are well-recognised symptoms of schizophrenia, with notable similarities to culturally influenced dissociative states. In a recent paper in *Cortex*[7] thought insertion-like occurrences were induced by telling highly hypnotically suggestible participants to variously imagine their own sentence completions, or instructing them that an 'engineer' was inserting the completion. In another condition, to induce experiences of alien control of movement, hypnotised participants were told they should think of a sentence completion, but that an engineer would control their hand while writing it down later. Functional magnetic resonance imaging demonstrated that a network involving areas for self-monitoring, language and movement were hypoactivated during the thought insertion. In a functional connectivity analysis, the cerebellum and supplementary motor area were significantly more and less active respectively during the 'alien control' *v.* 'voluntary movement' conditions. These findings are congruent with an established cerebellar–parietal network that has been implicated in comparison of actions and their somatosensory consequences, with the supplementary motor area providing a feed-forward signal to attenuate somatosensory processing.

Finally, the change in weather and the closing of the festive season has turned the Kaleidoscope team's minds to increasing their gym attendance. We were reminded of Chuck Palahniuk's

words that 'all the effort in the world won't matter if you're not inspired' and inspiration duly arrived in the form of Agudelo *et al*'s paper[8] in *Cell*, on skeletal muscle PGC-1α1 and how it modulates kynurenine metabolism. Kynurenine is a metabolite of the tryptophan degradation pathway that can be activated by chronic stress and has been implicated in a number of psychiatric illnesses, including major depression. Aerobic exercise can reduce the plasma levels of kynurenine, potentially protecting the brain from kynurenine reactions when the organism is exposed to chronic stress, and putatively reducing the expression of depressive behaviour. In this work, control and transgenic PGC-1α1 mice were exposed to a chronic mild stress protocol for 5 weeks, at which point anhedonia (reduction in sucrose consumption) and despair (increased immobility in forced-swim tests) behaviour were measured. Surprisingly, the modified mice displayed none of the behaviours that were evident in the control mice. PGC-1α1 expression in skeletal muscle was shown to protect synaptic transmission and plasticity in the glutamatergic systems of the hippocampus, effectively protecting the transgenic mice from depression, even after direct administration of kynurenine. So exercise appears to have a physiological link to brain function and, perhaps, alleviation of some of the features of depression. Inspirational indeed.

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8 Agudelo LZ, Femenía T, Orhan F, Porsmyr-Palmertz M, Goiny M, Martinez-Redondo V, et al. Skeletal muscle PGC-1α1 modulates kynurenine metabolism and mediates resilience to stress-induced depression. *Cell* 2014; **159**: 33–45.

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