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Highlights of this issue

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I may not have a lot to give/But what I got I'll give to you[1]

Linking the strength of one's handgrip with anything mental-health-esque felt one of the more confusing ideas the first time I read it some years ago. But over time, data on the topic, and in particular links with depression, have proven quite robust. Of course, we're primarily talking about associations rather than weakness of brachioradialis or flexor digitorum profundis causing low mood. Sarcopenia and dynapenia (age-related loss of muscle mass and strength, respectively) are linked with frailty, which is strongly associated with multiple poor environmental and health outcomes including depression. Further, exercise, which would counter such weakness, has a contrasting wealth of counter-benefits. Fascinatingly, there is also a plausible causative model, with skeletal tissue having a regulatory role in brain functioning via the release of neurotrophic factors upon tissue contraction. López-Bueno et al (pp. 135–142) extend our understanding of the epidemiology in older adults via work on over 115 000 individuals. As well as the large number of participants across 24 countries, their work is enhanced by long follow-up with a median of 7 years, which could account for time-varying changes. They found an inverse dose–response relationship between grip and risk of depression, though with a ceiling, for both men and women. Whether this represents an indirect association, a direct cause or both, it's certainly grist to the mill in the promotion of physical exercise in older adults. In another seemingly implausible tale on exercise, Kaleidoscope (pp. 147–148) discusses a recent paper from *Nature* suggesting that certain gut bacteria *cause* exercise motivation.

We move on to two papers on endocrine dysfunction and depression. The first, by Rasmussen et al (pp. 119–124), evaluated the underexplored area of whether a history of endocrine disease – specifically thyroid disease, pre-pregnancy diabetes and polycystic ovary syndrome – predisposes women to postpartum depression (PPD). It's an interesting question, asking whether prior and/or existing hormonal dysfunction and feedback loops risk creating broader imbalances, including, for example, via steroid hormones influencing hippocampal functioning. Taking data from almost a million women from Danish national registers, they found that those with such a history in the 10 years before childbirth had a significant (42% greater) risk of subsequently developing PPD. Interestingly, the relationship was bidirectional, with those with PPD having a 50% greater chance of later developing an endocrine condition. The authors reasonably argue that we need to improve our history-taking to explore such underlying risk factors. Jeffery et al (pp. 112–118) finish us off, looking at that common yet complex group of individuals with both depression and type 2 diabetes: once again an area where there are bidirectional associations and causes. In particular, the authors wanted to better understand any links among polypharmacy, duration of antidepressant drug treatment and propensity to illness relapse. Using a UK primary care cohort population, they included 48 000 individuals who started and discontinued antidepressants during follow-up. Both polypharmacy and longer duration of past pharmacological management of depression were associated with greater risk of relapse once medications were discontinued. The authors note that more work is needed to determine whether the association is with the actual polypharmacy, or confounded by indication of depression severity or multimorbidity.

I don't care too much for money/Money can't buy me love[1]

The 'social motivation deficit hypothesis' posits that aberrant reward processing underpins difficulties in social learning and skills in autism spectrum disorder (ASD). Attempts to elucidate more detailed neurobiological elements of this have produced mixed findings, potentially owing to the heterogeneous nature of ASD, typically limited study sample sizes, and failure to delineate anticipation and delivery phases of reward processing. Baumeister et al (pp. 100–111) report on a neuroimaging study with distinct social and non-social (monetary) reward tasks that were applied to over 200 individuals with ASD, aged 7 to 30 years, and a similar number of matched controls, as part of the well-defined Longitudinal European Autism Project. Whereas those with ASD showed relative reduction of ventral striatal activity during reward anticipation, there were no differences between task type, inferring a more general hypoactivity as opposed to one more specific to social motivation. Interestingly, categorical analyses showed that ASD effects were strongest in those without concomitant attention-deficit hyperactivity disorder (ADHD). The authors note the inadequate measurement of ADHD symptoms in such work in general, which might affect some of the aforementioned heterogeneity in some other studies.

Moving to a condition about which one might imagine we have better, longer-standing models: depression. Clark and Watson (pp. 125–134) update us on how in contemporary computational psychiatry, mood is the outcome of expectations (prior beliefs) interfacing with likely interoceptive consequences of action. The backdrop is a so-called 'Markov blanket', the statistical boundarying of a system – here, the brain from the outside world – and how our minds must make Bayesian inferences about the likely causes of inputs, based on these priors, to create predictions that can be tested and update priors and/or expectations. Their paper describes an observable decision process that seeks to minimise free energy with regard to such priors in response to a range of stressful and non-stressful induced environmental changes and anticipated outcome actions. The work allowed exploration of altering mood in depression, mania and anxiety. Notably, the depression simulation showed that environmental stress induced resistance to the ordinarily belief-changing impact of pleasure signals. Intriguingly, it also opens up avenues for future experimental work including illness prediction in single individuals. I've written elsewhere about the growing need for improved digital literacy as a core competency in psychiatrists; clearly this work is taking this to a new level. Malhi et al (pp. 97–99) bring us back to perhaps more comfortable territory with regards to modelling depression, as they contrast national guidelines for treatment from the UK with those from Australia and New Zealand. It's always stimulating to learn how colleagues work elsewhere, but as you might expect we have far more in common than in difference. Making a robust diagnosis, and keeping that appropriately reviewed and evaluated, and wrapping personalised care around the individual's needs sit at the heart of both.

Reference

1 The Beatles. *Can't Buy Me Love*. Parlophone Records, 1964.

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