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Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

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'1 in 4' prevalence for psychiatric disorder…or should that really be '1 in 3'? Parity of esteem in statistical headlines

Bebbington & McManus are to be thanked and congratulated for keeping this important and popular, but slippery, statistic under regular review and close-examination.[1] Their summary is likely to lead most readers to continue using the now well-known and oft-quoted '1 in 4' headline statistic for the overall prevalence of psychiatric disorders. They also rightly point out the twin dangers of over- and under-egging the statistic, risking lack of credibility and lack of impact, respectively.

However, I would suggest that the data presented comfortably allows for a new, revised '1 in 3' headline. This would not be overstating the case, but would simply accurately describe their findings. Their current '1 in 4' summary headline explicitly excludes 'personality and other disorders', as well as 'substance use disorders' and 'developmental disorders' …including them takes the true statistic to (very nearly) '1 in 3' (31.6%, to be precise).

There is now a long history of evidence and campaigning to have personality disorders recognised as 'bona-fide' mental disorders, with services developed and provided to match. Calls range from the 2003 National Institute for Mental Health in England 'Personality Disorder: no longer a diagnosis of exclusion',[2] through to the more recent 2018 consensus statement on personality disorder[3] and the freshly released 2020 Royal College of Psychiatrists Position statement, 'Services for People Diagnosable with Personality Disorder'.[4] They have always been in the ICD-10. Similar arguments and evidence could be made (ethically, on the grounds of stigma/parity, and scientifically) for the inclusion of the substance use disorders and developmental disorders that bring the final statistic to 1 in 3.

Presenting a new '1 in 3' headline would not be over-egging the evidence, but simply presenting the full findings of carefully conducted up-to-date research, thus promoting accurate, evidence-based societal perceptions of mental disorder, and subsequent policy decision-making. This is especially important given the limited traction so far gained in closing the gap between rhetoric and action with regard to 'parity of esteem for mental health'.[5] The NHS Long Term Plan for Mental Health[6] carries the potential for hope, but nothing should be taken for granted until it materialises.

In the meantime, we should advocate not excluding people with personality disorder (or substance use and developmental disorders) from the headline statistics generated by good-quality research; statistical parity of esteem for all those with mental disorders would justify a new, revised, evidence-based and accurate '1 in 3' summary headline, which would be neither under-egged, nor over-egged, but 'just(-ly) right'.

Declaration of interest

I am an elected member of the Executive Committee of the General Adult Faculty, Royal College of Psychiatrists, and also work in an NHS general adult community mental health team that treats and supports people with a range of mental health problems, including personality disorders.

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Further evidence for a role for the locus coeruleus in the aetiopathogenesis of dementia

We read with great interest the article by Peters et al, which provides a systematic review of changes in blood pressure, body mass index (BMI) and cholesterol levels in individuals that go on to develop all-cause dementia.[1] The authors find that a decrease in BMI and, in turn, blood pressure occurs well before the onset of dementia.[1] This finding, based on 13 longitudinal studies, adds to a confluence of evidence indicating that the locus coeruleus (meaning 'blue place' in Latin) plays a key role in the aetiopathogenesis of dementia.

The locus coeruleus serves as the major noradrenaline supplier to the brain. Via a ubiquitous network of projections, the locus coeruleus critically influences cognitive and affective processes together with physiological parameters such as heart rate, blood pressure, pupil size, sleep pattern, inflammation and stress. Neuropathologically, locus coeruleus degeneration is a hallmark of dementia, especially of those subtypes that are characterised by prion-like protein aggregates such as Alzheimer's disease, Down syndrome and Lewy body dementia, but not typically vascular dementia.[2] Importantly, neurofibrillary degeneration of locus coeruleus neurons seems to be an early event, and is closely linked to mild cognitive impairment and its progression to Alzheimer's disease.[3] It is easy to envisage how cytopathology in the locus coeruleus might affect sympathetic output to the cardiovascular system via coeruleo-vasomotor and coeruleo-spinal pathways and, thus, cause a decrease in blood pressure.

Placing the findings of Peters et al[1] in the context of the neuropathology of neurodegenerative diseases raises several interesting issues. If the decrease in blood pressure is a risk factor and/or precursor for dementia, should we monitor blood pressure in our

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elderly patients more carefully and, in particular, pay more attention to a decline in blood pressure? Furthermore, an exciting study in amyloid precursor protein transgenic mice found that noradrenaline depletion results in microglia dysfunction together with an increase in extracellular β-amyloid deposition, which can be rescued pharmacologically.[4] It remains to be seen whether this finding can be translated into noradrenaline-based therapies for patients with dementia (for example noradrenaline reuptake inhibitors). Conceivably, the neuroprotective effects of noradrenaline may even extend beyond a single cellular mechanism or disease entity. Understanding the alterations in central noradrenaline signalling preceding overt dementia may create a powerful new window of opportunity for identifying both preclinical dementia stages and developing novel treatments targeting the locus coeruleus circuitry.[5]

Declaration of interest

None declared.

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Sarcosine in the management of schizophrenia

I read with interest the editorial in December 2019 on 'A possible role for sarcosine in the management of schizophrenia'. Professor David Curtis did suggest that 'it seems to be universally well tolerated with an absence of significant side-effects'.[1] I wonder if addition of sarcosine to medication for schizophrenia is actually safe for every patient. It is well accepted that sarcosine level increases in many cases of carcinoma of the prostate gland. Indeed, it may well be a marker for carcinoma of the prostate.[2–4] It is thought that this elevated level of sarcosine is produced by the prostatic cancer cells. This does not mean that it causes the cancer. However, there are at least two important studies in the literature that comment on this issue. Sreekumar et al[5] in *Nature* in 2009 found metabolomic profiles delineating a potential role for sarcosine in prostatic cancer progression and Khan et al[6] in *Neoplasia* in 2013 found increased alteration of benign prostatic epithelial cells upon the addition of sarcosine to prostatic cells. I wondered therefore if a note of caution should be sounded about the use of sarcosine supplement in older men with schizophrenia, especially those with signs of prostatic hypertrophy. Prostate cancer is the most common cancer in males over 70 and the second most common cause of cancer deaths in men.

Declaration of interest

None.

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Author's reply

Dr Brennan is quite right to draw attention to the theoretical possibility that sarcosine might have unrecognised side-effects along the lines he draws attention to. However, it is worth stating that there is no empirical evidence at all that sarcosine does in fact increase risk of prostatic hypertrophy or carcinoma. This possibility could be investigated using animal studies and in the context of properly resourced, large-scale clinical trials. As sarcosine cannot be patented, these would have to be funded by research councils or charitable bodies since no pharmaceutical company is likely to be interested. At present, the evidence strongly suggests that sarcosine is effective in at least some patients with schizophrenia and is well tolerated and probably safe.

Declaration of interest

None declared

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Comment on 'The vulnerability paradox in global mental health and its applicability to suicide'

Michel Dückers et al, present a fascinating paper that aims to confirm an inverse association between country vulnerability and mental health.[1] They cite studies indicating that the higher levels of individualism, more equal distribution of power, low masculinity and greater indulgence within more affluent societies can increase the sensitivity of individuals to social failure and hence increase the risk of suicide. This is a very persuasive argument that harkens back to Émile Durkheim's work on anomie as cause of suicide.[2] It could be argued that modern technology attacks two presumed protective factors of traditional societies – community and the limited mobility that partially restricts contact to a small group of individuals of similar socioeconomic background. Social media would seem to do this by increasing personal isolation and increasing exposure to a wider network of 'more successful

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