


Suicide rates in people of South Asian origin in England and Wales

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Acceptance, grief and meaning

Prigerson & Maciejewski[1] assert that the resolution of grief coincides with increasing acceptance of loss, mainly cognitive and emotional acceptance. The role of spiritual acceptance has not been mentioned directly, although experiences like inner peace, tranquility and letting go, or regaining what is lost or being taken away, are more spiritual rather than emotional or intellectual. Moreover, some of the features which can be considered spiritual are included as criteria for prolonged grief disorder,[2] such as confusion about one's identity and feeling that life is empty and meaningless since the loss. Issues related to culture and the meaning and value of death[3] are relevant to both grief and acceptance, and I wonder whether these should also be considered.

Patients diagnosed with terminal cancer often confront existential issues. Experiences with patients with advanced or terminal cancers indicate that not only is cognitive and emotional acceptance essential, but that spiritual aspects are equally important. Spiritual acceptance of grief will help the grieved to understand the meaning and purpose of the loss. As Frankl[4] states 'suffering ceases to be a suffering as soon as it finds a meaning'. Longitudinal studies should clarify not only the way in which grief resolution relates to acceptance of dying and death, but also whether grief relates differentially to cognitive, emotional and spiritual acceptance. Prigerson & Maciejewski[1] conclude that decline in grief-related distress appears to correspond with an increase in peaceful acceptance of loss, which I feel could be enhanced by addressing issues related to purpose and meaning of the loss.

There is some small change besides the two sides of the coin!

1 Prigerson HG, Maciejewski PK. Grief and acceptance as opposite sides of the same coin: setting a research agenda to study peaceful acceptance of loss. *Br J Psychiatry* 2008; **193**: 435–7.

2 Prigerson HG, Vanderwerker LC, Maciejewski PK. Prolonged grief disorder: a case for inclusion in DSM–V. In *Handbook of Bereavement Research and Practice: 21st Century Perspectives* (eds M Stroebe, R Hansson, H Schut, et al): 165–86. American Psychological Association Press, 2008.

3 Byock I. The meaning and value of death. *J Palliat Med* 2002; **5**: 279–88.

4 Frankl VE. *Man's Search for Meaning* (4th edn). Washington Square Press, 1985.

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Authors' reply: We thank Dr Chaturvedi for highlighting the potentially important role that spirituality plays in the acceptance of loss. Recent research attests to the powerful influence of spirituality and religious beliefs in shaping patients' cognitive acceptance of terminal illness, treatment preferences, and even in determining the receipt of intensive, life-prolonging care in the last week of life.[1]

Nevertheless, we wish to differentiate between components of grief (e.g. yearning) and factors affecting the intensity and course of grief (e.g. spirituality). We posit that grief is on the same continuum as emotional acceptance – opposite poles of a unitary dimension. We contend that both spirituality and cognitive acceptance are distinct from, but related to, emotional acceptance and grief. Spirituality might foster emotional acceptance; cognitive acceptance might exacerbate grief. Identifying factors affecting grief and emotional acceptance may suggest ways to enhance an individual's mental health and well-being in the face of death, and offer ways to minimise loss-related suffering.

As a further distinction, we consider the loss of meaning in the context of prolonged grief disorder[2] to represent the emptiness experienced by the absence of an attachment figure. It is not intended to refer to a broader existential crisis. The sense of emptiness felt in grief may well lead a person to question the meaning of life. It may heighten an individual's sense of anomie (i.e. a feeling of disorientation and alienation from society caused by the perceived absence of a supporting social or moral framework) and affect a person's will to live. The meaning derived from spiritual beliefs may buffer individuals from the emptiness that follows a major interpersonal loss. Still, we do not consider spiritual beliefs to be components of grief. Rather spirituality may be a powerful antidote (perhaps, social support and social integration are others) to the pain of grief and elixir promoting emotional acceptance.

1 Phelps AC, Maciejewski PK, Nilsson M, Balboni TA, Wright AA, Paulk ME, et al. Coping with cancer: religious coping predicts use of intensive life-prolonging care near death. *JAMA* 2009; **301**: 1140–7.

2 Prigerson HG, Vanderwerker LC, Maciejewski PK. Prolonged grief disorder: a case for inclusion in DSM–V. In *Handbook of Bereavement Research and Practice: 21st Century Perspectives* (eds M Stroebe, R Hansson, H Schut, et al): 165–86. American Psychological Association Press, 2008.

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Taking an internet history

Cooney & Morris[1] argue that we should consider taking an 'internet history' to help assess young people's risk of self-harm, suicide and presumed psychopathologies such as 'internet addiction'. Although an understanding of how a client uses the internet may be important, the authors caricature what we know about the risks of the internet.

Although information on suicide methods is available online,[2] there is currently no clear evidence that the risk of self-harm or

suicidal behaviour is raised by 'pro-suicide' internet sites, as we lack all but the most preliminary studies in this area. Those studies that have been completed, in line with earlier research on 'pro-anorexia' sites, reported that 'pro-self-injury' boards relay mixed messages – clearly providing social support, coping methods and understanding, but also tending to minimise the significance of self-harming behaviour.[3] On the basis of current evidence, we might hypothesise that the use of such websites could equally be a protective factor or a risk factor.

The authors also mention internet addiction but seem unaware that the existing research is based on inconsistent criteria, is subject to widespread sample bias, relies almost entirely on correlative studies,[4] and that the concept itself lacks conceptual validity.[5] I challenge the authors to find any empirical studies to support their claim that in Asia 'cardiopulmonary-related deaths and even game-related murders in internet cafes are now regarded as serious public health issues'.

I wholeheartedly support the authors' contention that clinicians should consider the role of the internet in the lives of patients, but I would stress that this needs to be done with an understanding of the relevant research literature and a working knowledge of both the technology and culture of the medium.

We ask no less in other areas of clinical work and this is particularly important in a time when fears about the internet are amplified by the media with little regard to the evidence base.

1 Cooney GM, Morris J. Time to start taking an internet history? *Br J Psychiatry* 2009; **194**: 185.

2 Recupero PR, Harms SE, Noble JM. Googling suicide: surfing for suicide information on the Internet. *J Clin Psychiatry* 2008; **69**: 878–88.

3 Whitlock JL, Powers JL, Eckenrode J (2006) The virtual cutting edge: the internet and adolescent self-injury. *Dev Psychol* 2006; **42**: 407–17.

4 Byun S, Ruffini C, Mills JE, Douglas AC, Niang M, Stepchenkova S, et al (2008) Internet addiction: metasynthesis of 1996–2006 quantitative research. *Cyberpsychol Behav* 2008; Epub ahead of print.

5 Bell V. Online information, extreme communities and internet therapy: Is the internet good for our mental health? *J Ment Health* 2007; **16**: 445–57.

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Authors' reply: We welcome Dr Bell's interest in our letter and would be happy to debate the issue – but find ourselves entirely in agreement with him. He makes some crucial points which we too would emphasise. In particular, we all share the 'contention that clinicians should consider the role of the internet in the lives of patients'. We too 'would stress that this needs to be done with an understanding of the relevant research literature and a working knowledge of both the technology and culture of the medium.' Sadly, there is too little sound evidence to inform our attitudes.

Bell argues rightly that internet use could 'equally be a protective factor' and indeed one of us (J.M.) has participated in research exploiting the potential for delivering therapy via the web.

Bell is right in suggesting that until we have a better understanding of the complex and subtle influences which may be disseminated by the medium of the internet – and indeed by other communication media too – we and our colleagues are likely to fall into the trap of caricaturing both risks and benefits of internet use.

We are certainly aware that the term 'internet addiction' is itself a caricature of a diagnosis rather than a well-explored entity, but in the absence of empirical studies we are obliged to rely on anecdotal evidence. It has been as a result of some distressing clinical experiences, as well as concerns raised sensationally rather than scientifically in the media, that we have been moved to highlight the issue and to embark on our own preliminary studies.

Our letter does not aim to re-ignite a debate on whether the internet is helpful or harmful. As Dr Bell has observed, such a reductionist approach belies the complexity and variety of internet-based activities, any of which may have an influence in either direction.[1] We instead reflect that without empirical data to inform us, and where there is the possibility of either risk or benefit, careful and sensitive questioning of patients with high internet use may be a valuable component of a full psychiatric assessment.

The internet has taken a central place in modern culture particularly among younger people. Although we may not fully understand the complex interactions of the web and mental health, and while we await research to enlighten us, we are left with the choice to either ignore or engage with this phenomenon. Legislators, mental health advocates,[2] concerned parents and media journalists have all focused their efforts. It is time for scientists and clinicians to follow suit. In our view, this begins with the careful taking of an internet history.

1 Bell V. Online information, extreme communities and internet therapy: Is the internet good for our mental health? *J Ment Health* 2007; **16**: 445–57.

2 Papyrus: Prevention of Young Suicide. 2008 (http://www.papyrus-uk.org/news.html).

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Dementia: suicide by drowning

Purandare *et al*'s article on suicide in dementia is a valuable contribution to suicide research in the elderly, particularly in those with dementia.[1] The authors have already dealt with a number of methodological limitations quite succinctly. One important limitation in particular is the choice of controls. As the authors rightly stated, a control group of patients with dementia who had not died by suicide would have been more appropriate.

In the Method section, the authors referred to ICD–10 only and not ICD–9. As far as I am aware from my own experience dealing with the Office for National Statistics (ONS), ICD–10 has been used by ONS only since 2001. Prior to this date and for the first 5 years of Purandare *et al*'s study period (1996–2000), the ONS used ICD–9. If the authors applied the same criteria in their selection of suicide and open verdicts in cases reported between April 1996 and December 2000, then I assume they would have selected: ICD–9 E950–E959 for suicide and E980–989 excluding E988.8 for open verdicts respectively in a similar manner as they did with ICD–10 (p. 175). However, this very relevant fact does not appear to have been mentioned or explained by the authors, and was quite possibly omitted from the manuscript in error. However, this omission, which covers 5 years of a 9-year study, ought to be acknowledged and duly corrected.

I am grateful that the paper provides the opportunity to make one or two comments on some issues relating to drowning as a method of suicide in the elderly. Suicide by drowning accounted for 13.5% of total elderly suicide, being the third commonest cause of death in elderly suicide in England and Wales during 1979–2001 (16% for women as the second commonest cause of

death after overdose in elderly female suicide and 12% for men) and the fourth commonest cause of death by suicide in elderly males.[2] Interestingly, Purandare *et al*'s findings clearly show that drowning is the second commonest method of suicide by elderly psychiatric patients, with or without a dementia diagnosis.

The diagnosis of drowning itself may be difficult as there are no specific features at autopsy.[3] During the investigation of a body found in water, a wide range of possibilities other than downing have to be considered: accident, suicide, misadventure or homicide. Even when the diagnosis of drowning is confirmed (or excluded) the manner of death may remain undetermined.[3] It is also difficult to determine whether underlying natural diseases play a role in the death, wrongly regarded as self-harm.[4] There are some examples of this; 'sudden death in bathroom' has been reported,[5] occurring mostly in winter and 80% of cases were elderly persons who died while bathing. Cardiac arrest and subsequent drowning in bathtubs were attributed to the sudden reduction in blood pressure and cardiac arrhythmia, and not due to fatal self-harm.[5] Drowning has also been attributed to sudden unexpected death in epilepsy either subsequent to a seizure or occurring suddenly without explanation.[6] It may be relevant that in a local study in Cheshire, UK, bathroom drowning accounted for 7% of all elderly drowning.[7] Niacin deficiency, similar to pellagra, has been reported in high-income countries in people with alcohol dependency with poor eating habits and self-neglect, malabsorption, malignancy and nutritional deficiencies.[8] It may be reasonable to assume that some elderly people with dementia, especially those who live alone, develop mental and physical changes due to an easily overlooked nicotinamide deficiency which could result in accidental drowning in an attempt to alleviate skin irritation as is the case in some pellagra sufferers.[2] It is interesting to note that Lunetta *et al*[3] reported that 2.6% of bodies found in water were found to have died of natural causes after an initially suspected suicide. A review of trends in elderly suicide by drowning in England and Wales 1979–2001 revealed that suicide by drowning in the elderly attracted only 38% verdicts of suicide but 62% open verdict.[2] The high rate of open verdicts in death by drowning compared with any other method of fatal self-harm in England and Wales simply confirms the difficulties in reaching a firm conclusion in drowning death. Combining suicide and all undetermined deaths in suicide by drowning as a matter of course, especially in nationally collected statistics and consequently in research, may result in grossly exaggerated rates and misleading trends. Suicide by drowning is probably not amenable to prevention and although the elderly are often thought to benefit from suicide prevention more than younger adults, this is not likely to be the case regarding drowning, perhaps sadly more so in those with dementia.

1 Purandare N, Oude Voshaar RC, Rodway C, Bickley H, Burns A, Kapur N. Suicide in dementia: 9-year national clinical survey in England and Wales. *Br J Psychiatry* 2009; **194**: 175–80.

2 Salib E. Trends in suicide by drowning in the elderly in England and Wales 1979–2001. *Int J Geriatr Psychiatry* 2005; **19**: 175–81.

3 Lunetta P, Smith G, Pentilla A, Sajanntila A. Undetermined drowning. *Med Sci Law* 2003; **43**: 207–14.

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5 Yoshioka N, Chiba T, Yamauchi M, Monma T, Yoshioki K. Forensic consideration of death in the bathtub. *Leg Med* 2003; **5** (Supp 1): 375–81.

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7 Salib E, Rahim S, El-Nimr G, Habeeb B. Elderly suicide: an analysis of coroner's inquests into two hundred cases in Cheshire 1989–2001. *Med Sci Law* 2005; **45**: 71–80.

8 Kumar P, Clark ML. Nutrition. In *Clinical Medicine*: 163–4. Baillière Tindall, 1994.

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Purandare *et al*[1] used National Confidential Inquiry data to compare the characteristics of dementia patients who died by suicide with those of age-matched suicides with other diagnoses. They say empirical data on suicide in dementia are scarce and largely based upon case reports. Although the literature in this area contains a number of interesting, albeit highly atypical, case reports of patients with less common subtypes of dementia who died by suicide, there are also a substantial number of reports on suicide in older adults with various psychiatric diagnoses. The real problem with this literature is the quality of the studies; the majority are methodologically flawed, for example not employing a sensitive method for detecting mild cognitive impairment or absence of a control group, also use of coroner's records or death certificates to determine psychiatric diagnoses, sources known to underreport cases of dementia. The overall finding from this literature is that suicide appears to be uncommon in dementia, although the risk in Huntington's disease is in the region of threefold compared with the general population.[2] However, in a recent cohort study based on Danish case registers, it was found that for younger patients (50–69 years) diagnosed with dementia during psychiatric hospitalisation the risk of suicide was over eight times that of the age-matched general population, and the risk was threefold for patients aged over 70.[3]

Purandare *et al*[1] report suicide in dementia to be uncommon in the first year following diagnosis and highlight 1–5 years after first contact with services as the high-risk period. The case–control design of their study used a convenient but not very informative control group, namely age- and gender-matched suicides with other psychiatric disorders, and consequently the findings do not shed much new light on the association between suicide and dementia. No information is provided about severity of dementia or subtype (dementia subtype is of interest since fronto-temporal dementia would be expected to be associated with impulsive acts of suicide and self-harm, as frontal lobe impairment is associated with impulsiveness). The finding that suicide is less common soon after diagnosis is counter-intuitive, and contrary to the findings of Erlangsen *et al*[3] where suicide was most common in the first 6 months after diagnosis. There is also evidence that when attempted suicide occurs in dementia it is more common in early, mild disease and when accompanied by depression.[4,5]

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2 Harris EC, Barraclough, BM. Suicide as an outcome for medical disorders. *Medicine* 1994; **73**: 281–96.

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5 Osvath P, Kovacs A, Voros V, Fekete S. Risk factors of attempted suicide in the elderly: the role of cognitive impairment. *Int J Psychiatry Clin Practice* 2005; **9**: 221–5.

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Authors' reply: We welcome the interest in our study of suicide in patients with dementia in England and Wales. We found relatively lower risk of suicide during the first year of illness in dementia. Dr Haw writes that our findings are contrary to findings by Erlangsen *et al*.[1] However, such comparison is inaccurate. Erlangsen *et al* compared the risk of suicide in patients who were diagnosed with dementia during hospitalisation for physical or psychiatric illness with the risk of suicide in the general population. The authors point out that 'the findings cannot be generalised to persons with dementia who have not received the diagnosis while hospitalised'. The risk of suicide is known to be increased around the time of psychiatric hospitalisation.[2] Psychiatric in-patients would be expected to have more psychiatric disturbances. The study by Tsai *et al*,[3] which Haw quotes to support the association with mild dementia, found that delusions were present in all seven of those who later died by suicide. Haw also seems to compare the literature on increased risk of attempted suicide in those with mild cognitive impairment with our study of completed suicide in patients with diagnosed dementia. One consideration during disclosure of the diagnosis of dementia is the potential for adverse reactions. Our findings suggest that unless the risk assessment, which should be done in any patient being given a diagnosis of a major physical or mental illness, identifies a specific suicide risk, the 'fear of suicide' should not be a major factor in the decision to not disclose the diagnosis of dementia.

We thank Salib who correctly points out that our Method omitted ICD–9 which was indeed the classification system in use by the Office of National Statistics in the earlier part of the study. The relevant ICD–9 codes were E950–E959 and E980–989 (excluding E988.8).

Our findings are based on National Confidential Inquiry data, so include individuals who died by suicide within 12 months of contact with specialist health services. When we examined general population deaths (suicide and undetermined verdicts) in older people during the period covered by this study, drowning was the third most common method of suicide overall after hanging and self-poisoning (National Confidential Inquiry into Suicide and Homicide, personal communication, 2009). This is consistent with Salib's findings. We agree that the method of suicide may be an important determinant of verdict and there are difficulties in establishing suicide as a cause in drowning. However, this does not affect our main findings, which are based on the conventional definitions of suicide used in previous research and national statistics.

Suicide prevention requires a variety of strategies.[4] Although we agree that restricting access to drowning as a method of suicide may not be feasible, we do not agree that suicide prevention is futile in this group. Other strategies, for example the improved assessment and treatment of mental disorders, are likely to be worthwhile. We do not accept that younger individuals may be less amenable to prevention. However, different age groups may require a different preventive emphasis.[5]

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4 Department of Health. *National Suicide Prevention Strategy for England*. Department of Health, 2002.

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Adolescent-onset anorexia nervosa – missing half of the story?

As a psychiatrist working in an eating disorder service, I am always intrigued by the stability of the eating disorder diagnosis over time. As quoted by Treasure *et al*,[1] 'when long-term prognosis is considered, the overlap between anorexia nervosa and bulimia nervosa becomes more striking'. We all know that patients diagnosed with anorexia later switch over to other eating disorders and vice versa.[2] Hence, the paper about long-term outcome of anorexia nervosa[3] attracted my attention. The methodology of recruiting is vital in a population-based study. Wentz *et al* have taken extreme steps to be as rigorous as possible. But I consider that they might have overlooked some of the aspects. The authors have described how the diagnosis of individual patients has changed over time from anorexia nervosa to bulimia nervosa to no eating disorder to eating disorder not otherwise specified (EDNOS) (their Fig.1). This highlights the diagnostic instability of these groups of illnesses. The authors have assessed individuals cross-sectionally and asserted them to have anorexia nervosa. The important information missing here is whether these individuals had symptoms of other eating disorders such as EDNOS before having symptoms of anorexia. Since the study is about long-term outcome of anorexia nervosa, Wentz *et al* should have taken adequate care to ascertain that the cohort they were following did in fact belong to the anorexia nervosa group. This drawback is further highlighted in the exclusion criteria of the study. Excluding patients at the initial stage (Study 1) of individuals with a history of eating disturbances could have excluded individuals who might have been suffering from a non-anorexic type of eating disorder. The authors assert that in the subsequent study they did not exclude patients who crossed over to other eating disorders (thereby promptly registering changes prospectively), but what they did by excluding certain people is to exclude these potential participants who could have shown crossing over from another type of eating disorder to anorexia nervosa.

There are other minor points that are worth mentioning. Comprehensive screening, that would have included patients of all severity, of individuals who were born in a particular year (1970), identified 24 cases of anorexia nervosa. Combining this with a less comprehensively assessed group of individuals (and thereby potentially picking up only very severe cases) could have resulted in heterogeneous populations being mixed. Instead of mixing these two cohorts with potential difference in their severity with a possible impact over their outcome including complications,[4] the authors could have treated them as two

groups. I was also wondering about the validity of making a personality disorder diagnosis in such young individuals. Overall, if the diagnosis of anorexia could become bulimia, EDNOS or no eating disorder, the authors failed to consider the reverse being true (with the relative exception of bulimia to anorexia) at the important initial stage of this study.

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4 Bulik CM, Thornton L, Pinheiro AP, Plotnicov K, Klump KL, Brandt H, et al. Suicide attempts in anorexia nervosa. *Psychosom Med* 2008; **70**: 378–83.

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Authors' reply: Dr Sekar has concerns that the individuals in our study had other eating disorders before the onset of anorexia in adolescence. The aim of the original study, that took place in 1985, was to investigate the prevalence of adolescent-onset anorexia (and to examine background factors in this sample), not the prevalence of bulimia nervosa or eating disorder not otherwise specified. The mothers of the individuals, who were diagnosed with anorexia at the time of the original study, were interviewed thoroughly regarding premorbid eating disturbances.[1] Furthermore, the individuals themselves were interviewed regarding the same topic. No individual in the anorexia group (or the comparison group) had another eating disorder before the onset of anorexia. The school nurses at the schools in Göteborg continued to follow all pupils born in 1970 regarding weight and height until leaving school, usually after age 18 years. In the process, individuals with a later adolescent-onset of anorexia were also found. We believe that we have missed no cases of anorexia born in 1970 with anorexia onset before age 18 years. Since the original study focused on adolescent-onset anorexia we have not continued the screening of individuals born in 1970 after leaving school. Mean age of anorexia onset in our sample was 14.3 years. Bulimia typically presents during or after late adolescence and it is rare for onset to occur before the age of 14 years.[2,3]

The study has a prospective and not a cross-sectional design, i.e. we have examined all individuals at four occasions, but we have interviewed them both regarding current eating disorders (and other psychiatric disorders) as well as eating disorders during the follow-up period.[4,5] Data regarding eating disorders during the last follow-up period, between Study III and Study IV, are available from the first author.

Dr Sekar is also worried about the two subgroups being too diverse; the birth cohort with individuals born in 1970 was pooled together with a group of individuals with adolescent-onset anorexia born in adjacent years (in most cases 1971–1973). In the original study, the two groups were compared using several hundred background parameters and found to be similar in virtually all key respects.[1]

The use of personality disorder diagnoses with teenagers is arguable, but we considered (and still consider) it justifiable in cases persistently (over a period of several years) showing the essential characteristics of a personality disorder described in the DSM–III–R (the diagnostic manual used at the time of the original study). This is explicitly suggested by DSM–III–R guidelines. In the original study, apart from the age criterion, all DSM–III–R criteria had to be fulfilled for a diagnosis of personality disorder to be made. All individuals receiving a diagnosis of personality disorder showed significant impairment in social functioning and/or subjective distress.[1]

To conclude, since the aim of the original study was to investigate prevalence of adolescent-onset anorexia, we did not screen for other eating disorders. Nevertheless, from the time of entering our study, all participants (anorexia group and comparison group) were examined in great detail regarding eating disorders (past, present, and longitudinally at several follow-up occasions). We believe that we can safely say that there were no individuals who had crossed-over from another eating disorder to anorexia before the onset of anorexia in adolescence.

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Fallacies in standardised mortality ratios in anorexia nervosa

The article by Papadopoulos *et al*[1] adds to the evidence of high mortality rates in anorexia nervosa. An impressively large cohort was obtained through the Swedish Cause-of-Death Register which includes all Swedish persons who died since 1952. The crude mortality rate for 6009 females with at least one hospital admission for anorexia nervosa was 4.41% over a mean follow-up of 13.4 years (averaging 0.33% per annum). This rate compares favourably with other studies (0.5–2.2% per annum),[2] yet the authors, after much manipulation of their data, conclude that the mortality rate in Swedish women was 'astonishingly' high.

We contest this finding based on misleading calculations of standardised mortality ratios (SMRs). Standardised mortality ratios are a means of comparing mortality in a specified patient population with a standard population. The SMR value will exceed 1 in proportion to the risk of death from the disease under study.

The authors have two different usages of SMR. The first is the customary one when the calculation is applied to a cohort of persons who have been given a specific diagnosis at the outset. In Table 3 this SMR is given as 6.2 for the 6009 patients with anorexia nervosa, among whom there occurred 265 deaths whereas the expected deaths were 42.6. So far, so good.

Their second approach was to count the number of deaths according to each specific cause of death, yielding a different kind

of SMR. For example, there were 84 suicides yielding an SMR of 13.6, signifying that suicide was 13.6 times more common among the cohort of patients with anorexia nervosa than generally expected. Similarly, the SMR for deaths due to respiratory disease was 11.5. But the SMR for anorexia nervosa as a cause of death was said to be 650.0 and it is this figure which leads the authors to conclude the death rate in their sample was astonishingly high.

So it would be if it had clinical and statistical validity. The authors' errors arise from estimating the SMR for a subgroup (n=39) of the original cohort using the fraction:

$$\frac{\text{observed number of deaths}}{\text{number of expected deaths}}$$

The numerator is given as 39 patients in whom anorexia nervosa was the main cause of death on the death certificate. It is the denominator which is elusive in its estimated value. It is given as 0.1 but the authors' own data suggest this is an approximation for 0.06, a very low figure which results in an inflated value for the SMR (650) in this ambiguous subgroup of anorexia nervosa. We suggest that when an underlying cause of death (e.g. suicide, respiratory infection) was not identified, the certifier of the death entered anorexia nervosa on recognising a cachectic state, especially as malnutrition does not feature in the list of 'underlying' causes.

These objections do not apply to the first calculation of the SMR in the full cohort of patients with anorexia nervosa whose value was found to be 6.2, by no means an astonishing death rate.

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Authors' reply: Professor Russell and Dr Ward raise the issue of the suspected erroneously inflated value for SMR (650) for the subgroup of women in whom anorexia nervosa was stated as the underlying cause of death on the death certificate in our paper.[1] The expected number of deaths for this subgroup was indeed 0.06 (denominator) as they point out and it was presented with its one decimal approximation (0.1). Russell & Ward further suggest that the certifiers of the death would be prone to enter anorexia nervosa on the death certificate when a specific underlying cause of death could not be identified but a cachectic state was evident. We agree that this could be true, but we do not believe that such 'misclassification' would be problematic if those women had an active anorexia nervosa at the time of death. On the contrary, it would be worrisome if women with other diagnoses that lead to cachectic states (other than anorexia nervosa) were misclassified as anorexia nervosa on death certificates, but our inclusion criteria were specifically selected in order to reduce this possibility. In addition, we believe that the estimation of the SMR value for this specific subgroup of patients does not confer more information than what common sense dictates, namely that those with a lifetime diagnosis of anorexia have a much higher risk of dying from it.

Overall, women with anorexia nervosa in our cohort had a sixfold increased mortality compared with the general population. This excess mortality in anorexia nervosa is two to three times higher when compared with the excess mortality observed in mental disorders in general[2] and more specifically in schizophrenia,[3] bipolar and unipolar disorder.[4] Moreover, we would like to point out that we were most astonished by the persistence of this unfavourable outcome throughout the lifetime, with high SMRs for both natural and unnatural causes of death even 20 years or more after the first admission for anorexia nervosa.

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Suicide rates in people of South Asian origin in England and Wales

A notable finding in McKenzie *et al*'s study[1] of suicide rates in people of South Asian origin is that the high relative rates in younger Asian women reported in previous research studies are found in the 1993–98 data-set but not that for 1999–2003, which shows high relative rates for Asian women over 65. In discussing their results, the investigators acknowledge potential problems with the study's methodology, including the numerator (how well the SANGRA name recognition algorithm ascertains individuals of South Asian origin in more recent samples) and denominator (the validity of a linear interpolation of numbers over their period). However, perhaps cautions are required with respect to the overall robustness of the SANGRA algorithm and the issue of numerator/denominator compatibility: the numerator uses an operational definition of ethnicity (derived from name information) and the denominator is based on self-assignment by individuals to census categories.

These matters are brought into focus in the derivation of denominators. The investigators use the counts for the 1991 categories 'Bangladeshi', 'Indian', 'Pakistani' and 2001 categories 'Asian or Asian British: Bangladeshi, Indian, Pakistani'. They also include the 2001 category 'White and Asian' (numbering around 190 000 in the census) on the grounds that people in it '. . . could be identified by SANGRA if any of their names were of South Asian origin'. We have no systematic data on how offspring of these inter-ethnic unions are named, although qualitative research has revealed the complexity of the process.[2] Inclusion of the 'White and Asian' category also introduces heterogeneity into the South Asian collectivity. Evidence from the Office of National Statistics (ONS) Longitudinal Study for members having a 1991 and 2001 ethnic group showed that half (49.0%) of the 993 'White and Asian' persons identified as 'White' in 1991 and just 9.5% identified as one of the three South Asian groups.[3] Similarly, in recent research half in the 'White and Asian' group prioritised 'White' when asked to name just one ethnic group that contributes most strongly to their identity. Our collective

identities affect our ability to make an individual life and have relevance in the context of suicide risk.

The investigators exclude 'Other Asian' (the free-text 'Any other Asian background' under the 'Asian or Asian British' label, numbering around 240 000 in the 2001 census) from the denominator 'because the majority of this group are of Middle Eastern or Sri Lankan origin'. Although around one in four were born in Sri Lanka and one in six in the Middle East, 37% had a region of birth in South Asia and 31% in the UK.[4] Given that the focus is on ethnicity rather than country of birth, the ONS Longitudinal Study data are, again, informative: of members with a 1991 and 2001 ethnic group, 42% of 1285 'Other Asian' persons identified as Indian, Pakistani or Bangladeshi in 1991. In this study, none from the 'Other Asian' group are counted in the denominator.

Finally, the investigators point out that SANGRA was validated against real data. However, the key data-set were London and Midlands hospital in-patient admission data from the mid- to late-90s, a period during which the quality of ethnic coding was very poor, the team itself admitting that further studies are needed to confirm whether SANGRA is able to produce valid results across Britain.[5]

Beyond the parsimonious way in which the statistical data is presented (with no measure of the precision of the rate estimates), the collective effect of potential problems with numerator/denominator compatibility and concerns about SANGRA's performance is a factor which needs to be considered in making a judgement whether to accept these findings as the accurate contemporary evidence needed to shape specific prevention strategies.

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McKenzie *et al*'s findings[1] of low suicide rates among South Asian men in both 1993–98 and 1999–2003, and of high suicide rates among young South Asian women in 1993–98, are consistent with previously reported findings.[2] The difference from previous findings lies in the absence of an excess in young South Asian women in the recent period, 1999–2003, and an excess instead in older women.

In the absence of observed numbers of deaths and confidence intervals for the rates, it is not possible to interpret the statistical significance of the findings in Tables 1 and 2 of their article (i.e. which ethnic differences by age, gender and over time are statistically significant). Likewise, although the results were 'essentially unchanged' following the sensitivity analysis, it is unclear which differences remained statistically significant after the 11% inflationary adjustment for potential underidentification of South Asian suicides arising from the use of SANGRA.

High rates of suicide and attempted suicide among young South Asian women have been a consistent and enduring finding in national and international research over decades (see Raleigh[2] for references). Research specifically commissioned to examine this issue reported high rates of attempted suicide among young South Asian women in London, including those who were UK-born.[3] A recent study found a 2.8-fold higher suicide rate among South Asian women aged 25–39 in contact with mental health services.[4] Given the evidence overall, any decline in suicide rates in this group over the past decade would therefore be welcome. However, as this finding is counter to the evidence to date, it should be kept under review to ensure it is a real trend and not an artefact, given the caveats associated with analyses based on software-assigned ethnicity, many of which are acknowledged in the paper.

The constraints to inclusion of ethnicity at death registration were established by ONS in its review of death certification some years ago. Given the growing need for epidemiological monitoring of mortality rates and trends by ethnicity and cause of death, ONS, the Department of Health and the Information Centre should consider alternative approaches for making these data available, for example through data linkage, as undertaken in Scotland and recently by ONS for deriving infant mortality rates by ethnic group.[5] This would provide sound, comprehensive epidemiological data with self-assigned ethnicity coding of numerators and population denominators on a consistent and comparable basis, thereby avoiding the potential mismatch between numerators and denominators in the use of name-recognition software. It would also obviate the need for researchers to have access to names, which is frequently not possible for data protection reasons.

In the interim, given the growing use of such proxies for epidemiological purposes, there is a strong case for these national agencies to undertake a systematic review of the available name-recognition software programs, to establish their robustness for epidemiological analyses using national data-sets and across the spectrum of morbidity and mortality. This would also be in keeping with the statutory responsibility of these national agencies for ensuring the availability of comprehensive national data to support equality monitoring.

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Authors' reply: Our paper is the first to report findings at variance with previous studies and we welcome the opportunity to discuss the findings and subject them to scientific scrutiny.[1] The findings of a decreased rate of suicide in South Asian men has not been challenged. It is reassuring that the experimental

methods of SANGRA do not produce unexpected findings for this group.

The comparison with our study of the National Confidential Inquiry should be made cautiously, as that study included suicides among people in contact with services rather than from all deaths reported by the ONS.[2] We would also suggest self-harm rates are not a proxy for comparative suicide rates.

Dr Aspinall makes important comments about ethnicity classification. There are no data that investigate self-assigned *v.* ascribed ethnic identity and variations of this relationship across geographical areas of the UK, over time, or the patterns of transmission of ethnic identity through the generations. There are often unpleasant trade-offs when using descriptors of ethnicity and culture from survey research.[3] Ethnicity is not a measure of cultural identity.[3] Perhaps nested within self-reported ethnic categories we need more complex models of identity that take account of acculturation, social stratification and their interaction.[4] This may help more precisely to disentangle specific influences on health. Unfortunately, the concepts and methods to do this are still being developed.

The information on the denominators so far is useful but incomplete to forge a new study design or recommend specific changes in routine data sets; for example, we would need a breakdown of self-reported ethnicity in the 'Asian Other' and 'White and Asian' categories by gender and age. Adding more ethnic categories which are imprecisely measured, or for which the difference between self-rated and ascribed may vary over time and place, may lead to more random misclassification; therefore, more ethnic categories may not always be helpful or explain any more precisely which specific ethnic identity groups are at greater or lesser risk.

The finding of high rates of suicide in young South Asian women in the UK are based mainly on papers sampling groups born in Southern Asia – using the same methodology would miss the 50% of South Asians currently in the UK.[1] Of the two studies that used different methodologies, one used names to ascertain South Asian suicides but the methodology was not validated or described so that it could be replicated, and the other studied parts of London, although we know that there are significant differences in South Asian suicide rates by geographical location.[5]

The main purpose of the study was to improve the accuracy of the estimate of suicide rates in all South Asian people living in the UK, irrespective of place of birth. We know that over 50% of the South Asian population was born in the UK and future studies need a way of accurately including them in rate calculations. We believe that ethnicity assigned on death certificates is likely to be the most useful way forward. We concur with the view that there is a need to assess trends over time.

We agree that there are caveats because of the SANGRA program. However, we do not think that the program would have worked less well in the 1999–2003 cohorts than the 1993–98, that it would work so differentially for men and women, or that artefact can explain the findings in both the older and the younger female population.

We would welcome attempts to replicate these findings using different methodologies and show the raw data to facilitate this and the calculation of other statistics such as confidence intervals (Table 1). However, in the meantime, this is the first contemporary attempt to ascertain the suicide rates for the whole of the UK South Asian population. The findings for older women should be viewed with concern and the drop in younger women should be replicated and followed over time. Policy has to be based on the best evidence available rather than the best evidence that is sought and might one day become available.

Table 1 SANGRA South Asian Suicides 1993–2003

Age range, by gender	1993–1998	1999–2003
Male		
15–24	136	72
25–34	145	158
35–44	121	109
45–54	54	72
55–64	46	39
65–74	18	18
>74	14	10
Total	534	478
Female		
15–24	60	29
25–34	75	55
35–44	54	29
45–54	28	25
55–64	12	15
65–74	13	14
>74	6	11
Total	248	178

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Risperidone for adolescent schizophrenia

We would like to make a few comments on the study by Haas *et al*[1] which compares the efficacy and safety of two dosing regimes of risperidone. First, we would like to raise concerns with regard to the design of the study. Both groups were receiving a flexible dose of risperidone in the first 4 weeks. The dose was to remain stable only during the last 4 weeks – a very short duration. Patients started showing substantial response in the first week onwards. It is not possible to rule out the placebo effect and difficult to determine the dose-related response. Surely this design cannot establish the optimal effective dose as the dose was changing very often especially in the first 4 weeks.

Second, patients in the control group were not allowed the assured effective treatment. The control group received risperidone tenfold less than the intervention group. This dose was as good as a placebo. This raises serious doubts as to whether the lower dose was also effective or whether it was a placebo effect. This is clearly evident as a substantial improvement compared with baseline was noted in both groups within 7 days. It also raises ethical issues as the authors decided to continue a presumably ineffective dose (0.15–0.6 mg/day) in the control group for 8 weeks.[2] Patients in this group had a higher discontinuation rate owing to lack of efficacy. It was unethical to continue with such

a low dose. We also wonder why the authors arbitrarily decided to have a tenfold lower dose in the control group. We question why the authors did not try to compare the intervention drug with an existing drug such as olanzapine, as Hill[3] reports that the key point is how a new treatment compares with existing treatment rather than whether it is better than nothing.

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3 Hill AB. Medical ethics and controlled trials. *BMJ* 1963; **1**: 1043–9.

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Authors' reply: Several of the limitations of our study design as mentioned by Jainer & Mahood have been addressed within the publication's discussion. The study was not designed to establish an optimal dose or evaluate efficacy *v.* placebo. Thus, as we noted, no conclusions can be made in this regard. The objective of this study was to determine whether there was a difference between two dose ranges; this goal was achieved. The use of an active comparator was not possible because there was no drug approved for use in children or adolescents suffering from this disorder at the time the study was conducted.

The dose ranges were chosen to compare the adult therapeutic dose, known to be effective in schizophrenia, with a low dose. This low dose was presumed subtherapeutic, but not known to be ineffective. Notably, in studies in children with disruptive behaviour disorder where the allowable flexible dose range included doses <0.6 mg/day, risperidone was shown to be efficacious.[1,2] Additionally, at the time this study was designed, a low-dose comparator was preferred over placebo, although thinking on the appropriateness of using placebo control in studies of antipsychotics has evolved since then.[3] A placebo effect in terms of treatment response cannot be ruled out in our study, and presumably any placebo response would have affected both dose arms similarly. Numerous safeguards were implemented to minimise risk to patients in the study from the outset. The protocol was reviewed by and received approval from an independent ethics committee and individual institutional review boards. All patients and caregivers were advised that both doses were experimental and the lower dose might be an ineffective treatment. Accordingly, all enrolled patients were initially hospitalised and only adequately stabilised patients could be discharged to continue in the trial as out-patients. Patients could discontinue treatments at any time. All patients were monitored closely throughout the duration of the trial to further ensure patient safety.

Our conclusions remain valid, as they pertain to the comparative favourable efficacy benefits achieved in this study with risperidone treatment in the 1.5–6.0 mg/day dose range compared with the lower range. Both regimens were well tolerated with low discontinuation rates due to adverse events.

Declaration of interest

The study was funded by Johnson & Johnson Pharmaceutical Research & Development, LLC. M.H. and M.E. are employees of Johnson & Johnson Research & Development, Division of Janssen Pharmaceutica, NV. S.K., J.S., I.A., J.Q., G.P. and V.K. are employees of Johnson & Johnson Pharmaceutical Research & Development, LLC.

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3 Stroup TS, Alves WM, Hamer RM, Lieberman JA. Clinical trials for antipsychotic drugs: design conventions, dilemmas and innovations. *Nature Rev* 2006; **5**: 133–46.

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Time to change concepts and terminology

The proposal by van Os to introduce 'salience dysregulation syndrome'[1] to describe the psychosis spectrum, replacing schizophrenia and bipolar disorder, represents an acceptance that such terms have outlived their usefulness. But by introducing three subcategories, 'with affective expression', 'with developmental expression' and not otherwise specified, he simply replaces outdated terms but retains the invalid and unreliable concepts – schizophrenia and bipolar disorder re-emerge with different names.

The evidence for a psychosis spectrum, as he describes, now seems irrefutable. At one end, manic symptoms 'represent the greatest diagnostic value' and this end of the continuum seems relatively recognisable and clinically relevant. Moving towards the other end takes us into Bleuler's schizophrenias and the more recently emerged area of drug-related psychosis. We have argued the case that rather than simply continuing to try to homogenise the schizophrenias, we should listen to what patients tell us led to their first episodes. Dudley *et al*[2] have recently used Q-sort methodology to elicit this and found similarities to concepts developed empirically from clinical practice.[3] We have used these concepts of drug-related, traumatic, stress-sensitivity (early-onset) and anxiety (late-onset) psychoses successfully with patients[4] and also found them to be destigmatising.[5] They are derived from work which Van Os himself has been pre-eminent in developing and we suggest to him that he has the courage of his convictions and use aetiological concepts rather than nebulous descriptive ones.

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Author's reply: In an attempt to come up with new terminology, I sought to combine scientific evidence for valid contrasts with scientific evidence for a mechanism (aberrant assignment of salience) that refers to a psychological process that the general public can recognise and relate to, although a considerable amount of explanation may be necessary (see my reply to Bill George[1]). Kingdon *et al* propose a different approach: they select possible risk factors and mechanisms associated with schizophrenia and investigate whether aetiological diagnostic constructs based on these are acceptable to patients. To the degree that their method included an analysis of acceptability to patients,[2] their proposal is certainly superior to mine. A weakness of the method may be that there is little evidence that, for example, trauma and drug use underlie discrete effects that can be separated diagnostically. If anything, research suggests that there may be interacting causes that have an impact on the same final common pathway.[3,4] Although it could certainly be argued that as long as there are established risk factors (although doubts exist[5,6]) and the terminology is acceptable to patients, this should not prevent their use as aetiological diagnostic constructs: a major problem would remain – acceptability to mental health professionals. How likely is it that these constructs would be accepted by the DSM and ICD committees currently revising diagnostic criteria? In my view, if we really want to abandon the stigmatising term of 'mind-split disease', it is important to come up with an alternative that is not only acceptable to patients, but also to mental health professionals. The reason for this is that DSM and ICD terminology is by far the most influential in how the general public attempts to understand 'madness'. Therefore, unless DSM and ICD terminology is changed, the part of the stigma that is induced by confusing and mystifying terminology will not change. Also, the continued use of the term 'psychosis' proposed by Kingdon *et al* may perpetuate the mystification of the experiences of patients, as the public cannot understand this term to make a connection to their own psychological experiences.

The most important issue, however, is how many patients, professionals and other stakeholders want the name to change. It certainly seems that many are of the opinion that a confusing and mystifying 19th-century term should not be used to diagnose patients in the 21st century. Maybe the time has come for the DSM and ICD committees to make a decision on this topic and, in the case a name change is favoured, to develop a process through which a change that is acceptable to as many stakeholders as possible is achieved. The methodology of consulting patients developed by Kingdon *et al* should figure prominently in this endeavour.

1 van Os J. Salience dyregulation syndrome: a patient's view (author's reply). *Br J Psychiatry* 2009; **194**: 467.

2 Kingdon D, Gibson A, Kinoshita Y, Turkington D, Rathod S, Morrison A. Acceptable terminology and subgroups in schizophrenia: an exploratory study. *Soc Psychiatry Psychiatr Epidemiol* 2008; **43**: 239–43.

3 Cougnard A, Marcelis M, Myin-Germeys I, De Graaf R, Vollebergh W, Krabbendam L, et al. Does normal developmental expression of psychosis combine with environmental risk to cause persistence of psychosis? A psychosis proneness–persistence model. *Psychol Med* 2007; **37**: 513–27.

4 Houston JE, Murphy J, Adamson G, Stringer M, Shevlin M. Childhood sexual abuse, early cannabis use, and psychosis: testing an interaction model based on the National Comorbidity Survey. *Schizophr Bull* 2008; **34**: 580–5.

5 Macleod J, Davey Smith G, Hickman M, Egger M. Cannabis and psychosis. *Lancet* 2007; **370**: 1539.

6 Morgan C, Fisher H. Environment and schizophrenia: environmental factors in schizophrenia: childhood trauma – a critical review. *Schizophr Bull* 2007; **33**: 3–10.

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Abortion and mental health disorders

The paper by Fergusson *et al*,[1] accompanied by comments, is a valuable addition to knowledge on this topic, but I should like to mention two issues which limit the usefulness of what is presented.

First, neither Fergusson nor the commentators give sufficient emphasis to the fact that that the communities of the Christchurch area of New Zealand are relatively prosperous and well organised compared with those in many parts of the rest of the world. The study findings cannot be extrapolated to communities where poverty, various degrees of malnutrition, and scarce medical and social services are common. In such communities, the modest level of what Fergusson *et al* call 'mental disorders' is likely to be present in many persons whether pregnant or not, and the significance of an unwanted pregnancy is also likely to be quite different from what it might be in more prosperous settings. How these issues interact can only be examined by direct studies in different communities.

Second, one of the commentators (Professor Patricia Casey) presents herself as 'not a member of any campaigning organisation', and also lists a number of her other activities to do with abortion and related issues. But there is no mention (probably due to the never-ending search for brevity that plagues us all) of the fact that she is a sincere member of the Roman Catholic Church, and that she always takes what can be called the 'pro-life' side in debates about abortion and related issues. Professor Casey is, of course, completely entitled to her opinions, and I have no doubt that she is proud of her activities in this difficult field and would never wish to hide them. But in these debates we all start from a position determined in part by personal background, and readers will not fully understand comments unless such things are known.

1 Fergusson DM, Horwood LJ, Boden JM. Abortion and mental health disorders: evidence from a 30-year longitudinal study. *Br J Psychiatry* 2008; **193**: 444–51.

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Author's reply: Professor Cooper suggests that the findings we report may not describe the linkages between abortion and mental health in communities that are more impoverished than the relatively advantaged New Zealand community that we studied. We agree that it would be rash to generalise our findings to these contexts. We are of the view that it is important that research into this topic is conducted in communities where material and economic conditions may make unwanted pregnancy a far more serious and stressful life event than is the case for relatively

privileged developed societies. As we point out in our paper, the important implications of our research relate to the interpretation of the abortion laws in legislations such as those in the UK and New Zealand where the mental health risks of unwanted pregnancy are the principal grounds on which abortion is authorised. Our findings suggest that in the New Zealand context, at least, the mental health risks of abortion may outweigh the mental health risks of unwanted pregnancies that come to term but that, in any event, the mental health risks associated with either of these outcomes appear be relatively small.

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Editor's note: When I commissioned the commentaries on Dr Fergusson's paper I was aware that the subject of abortion tended to polarise opinions. For this reason I commissioned two reviews, one from each side of the debate, but I had confidence from my choice of authors that they would focus primarily on Dr Fergusson's paper, not the wider issues. I chose Professor Casey as someone who was a pro-life supporter and Dr Oates as a representative of the pro-choice group (with agreement for Drs Jones and Cantwell to be added later), even though I consider these terms somewhat limited and two-dimensional in the context of reviewing a scholarly paper, and believe that specific declarations of interest in this context were unnecessary. I hoped that neither commentary was viewed as tendentious by our readers and personally regard both of them as adding substance to the conclusion of Fergusson *et al* that 'the results do not support strong pro-life positions that claim that abortion has large and devastating effects on the mental health of women. Neither do the results support strong pro-choice positions that imply that abortion is without any mental health effects' (p. 450).[1]

1 Fergusson DM, Horwood LJ, Boden JM. Abortion and mental health disorders: evidence from a 30-year longitudinal study. *Br J Psychiatry* 2008; **193**: 444–51.

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Correction

Group psychoeducation for stabilised bipolar disorders: 5-year outcome of a randomised clinical trial. *BJP*, **194**, 260–265. Table 2, p. 264: the values for Depression should read: Control group 398.55 (364.16); Psychoeducation group 93.28 (165.46). This was a typographical error only and does not affect the statistical analysis presented.

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