BJPsych | The British Journal of Psychiatry (2010) 197, 332–333

# Correspondence

Edited by Kiriakos Xenitidis and Colin Campbell

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## Interpretation of ecological data

We read the debate on antidepressants and suicide[1] with interest, especially the issue concerning the importance of controlling for possible confounding variables in ecological studies and the associated difficulties in drawing conclusions from such ecological observations.

One potentially important confounder, which has been overlooked, is the size of the family of origin. Birth cohort studies from Scotland[2] and Norway[3] suggest that having elder siblings may be linked with an increased risk of suicide. As the total fertility rates (a reasonable proxy for average family size) fell across most Western countries between the 1950s and 1970s,[4] it is plausible that some of the decline in suicide rates observed from the late 1980s onwards may be, in part at least, a resultant cohort effect.


1 Isacsson G, Rich CL/Jureidini J, Raven M. The increased use of antidepressants has contributed to the worldwide reduction in suicide rates (debate). *Br J Psychiatry* 2010; **196**; 429–33.

2 Riordan DV, Selvaraj S, Stark C, Gilbert JSE. Perinatal circumstances and risk of offspring suicide. Birth cohort study. *Br J Psychiatry* 2006; **189**: 502–7.

3 Gravseth HM, Mehlum L, Bjerkedal T, Kristensen P. Suicide in young Norwegians in a life course perspective: population based cohort study. *J Epidemiol Community Health* 2010; **64**: 407–12.

4 Population Division of the Department of Economic and Social Affairs of the United Nations Secretariat. World Population Prospects: The 2008 Revision Population Database. United Nations, 2009 (http://esa.un.org/unpp).


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**doi: 10.1192/bjp.197.4.332**

**Author's reply:** We welcome Riordan & Stark's suggestion that declining family size may have contributed to declining suicide rates. I agree. As we noted, there are many potential confounding variables in the relationship between antidepressants and suicide.

Riordan & Stark cite two birth cohort studies using data linkage that both found that birth order was independently associated with suicide risk. In Riordan *et al*'s 2006 analysis of linked data from the Scottish Morbidity Record and Scottish death records, higher maternal parity, younger maternal age, non-professional parental occupations and low birth weight were all independently associated with higher suicide risk of offspring in young adulthood.[1] In Gravseth *et al*'s study using several Norwegian national registers, suicide risk factors included higher maternal parity, instability of maternal marital status during childhood, low education levels and severe mental illness.[2] Notably, maternal parity remained a significant risk factor even after adjustment for mental illness.

Data linkage studies such as Riordan *et al*'s and Gravseth *et al*'s are particularly important in suicide research because of the need for adequately large sample sizes and sufficient statistical power to investigate suicide, a relatively rare event. They are methodologically superior to ecological studies, which are the mainstay of claims that antidepressants reduce suicide. The inherently weak methodology of ecological studies is often compounded by failure to control for potential confounding factors and by biased interpretation.[3,4]

Data linkage studies generally reveal multiple significant contributory factors, many of which are linked to social adversity. Many such factors also contribute to other forms of premature mortality such as accidental death and natural death due to preventable conditions. For example, Riordan *et al* also found an association between higher maternal parity and increased risk of offspring death from causes other than suicide. The aetiological overlap means that primary prevention focusing on shared determinants has greater potential to reduce overall mortality.[5]

Data linkage studies provide valuable evidence that challenges the simplistic belief that depression is the cause of suicide and antidepressants are the solution. As emphasised by De Leo & Cerin,[4] suicide is not simply a function of depression, and suicide prevention is far more than a psychiatric enterprise.

### Declaration of interest

I am a member of Healthy Skepticism.


1 Riordan DV, Selvaraj S, Stark C, Gilbert JSE. Perinatal circumstances and risk of offspring suicide. Birth cohort study. *Br J Psychiatry* 2006; **189**: 502–7.

2 Gravseth HM, Mehlum L, Bjerkedal T, Kristensen P. Suicide in young Norwegians in a life course perspective: population-based cohort study. *J Epidemiol Community Health* 2010; **64**: 407–12.

3 Safer DJ, Zito JM. Do antidepressants reduce suicide rates? *Public Health* 2007; **121**: 274–7.

4 De Leo D, Cerin, E. More than antidepressants are needed to avert suicide. *BMJ* 2003; May 15 (http://www.bmj.com/cgi/eletters/326/7397/1008).

5 Neeleman J. A continuum of premature death. Meta-analysis of competing mortality in the psychosocially vulnerable. *Int J Epidemiol* 2001; **30**: 154–62.


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**doi: 10.1192/bjp.197.4.332a**

## Failure to communicate effectively or failure of feedback?

In reference to a recent study,[1] we do not agree with the use of email as a sole medium to provide feedback to website administrators. Electronic communication by an unknown, unexpected source has a high chance of ending up in the spam box and going unnoticed by the recipient. Sending the email on a letterhead or with a university logo could not have added enough authenticity to suspicious-looking mail, given that we are all wary of opening emails, let alone attachments, from unknown senders. Further, the recipient may have lacked the expertise to decipher it as genuine feedback. Lack of acknowledgement of the receipt by a large proportion of websites makes us wonder whether the results should be interpreted as a human failure or a technical failure. An alternative medium could have been a fully addressed, official communication posted or couriered personally to the administrator, with a formal acknowledgement of receipt. Another medium could have been follow-up via telephone acknowledging receipt of the email. But an essential component for feedback to be successfully conveyed is to ensure that the message reached the recipient, that the message was at the very least received, if not acknowledged or acted upon.

The authors state 'this trial can be seen as an effectiveness rather than an efficacy trial, because it evaluated feedback under realistic conditions'. We wish to say that, generally speaking, the effectiveness of an intervention is meaningful after the efficacy has been established. Although there was an attempt to provide feedback, we felt that the one-time sending of an electronic communication is neither complete nor strong enough an effort at feedback and, realistically speaking, is likely to go unnoticed. The study, however, highlights an important point regarding the poor quality of most websites concerning serious medical or public health matters. Although quacks or uncertified self-claimed experts can be prosecuted under law, there are a number of websites promising help for people who are suicidal, but which fail to deliver on the quality or extent of information available to individuals seeking help.[2] There is a need for regulation or a mandatory professional certification of the content of websites, especially in such matters where life can be at stake. Short of that, interventions need to be planned so that they are readily acceptable and effective in ensuring a positive change in the content of suicide prevention websites.


1 Jorm AF, Fischer JA, Oh E. Effect of feedback on the quality of suicide prevention websites: randomised controlled trial. *Br J Psychiatry* 2010; **197**: 73–4.

2 Van Ballegooijen W, van Spijker BA, Kerkhof AJ. The quality of online suicide prevention in the Netherlands and Flanders in 2007. [In Dutch.] *Tijdschr Psychiatr* 2009; **51**: 117–22.



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**doi: 10.1192/bjp.197.4.332b**


**Author's reply:** The point of our study was to see whether a very simple, cheap feedback intervention might work to improve quality of website information. Clearly, it either did not work or the effect was small. It is quite possible that more elaborate feedback interventions might work. This needs to be tested. However, if these were to work, would they be of any practical use? Is anyone going to go to the trouble of routinely monitoring website quality and personally contacting website developers to give them feedback? Who would fund this sort of work? There is also the related issue of who would resource website owners to carry out substantial revisions. In this regard, it is interesting that after our trial was over, one website administrator wrote to us saying that they had now revised their website in response to our feedback. The reason they cited for the delay is the limited resources they had as a non-government organisation.

Readers of our article may be interested in another study on feedback which only came to our attention after our trial was completed. This was a much larger randomised controlled trial ($n = 299$ URLs) from the field of pharmacology and gave feedback on quality of information on the drug sildenafil. Like our trial, this one found no effect of emailed feedback letters.


1 Martin-Facklam M, Kostrzewa M, Martin P, Haefeli WE. Quality of drug information on the World Wide Web and strategies to improve pages with poor information quality. An intervention study on pages about sildenafil. *Br J Clin Pharmacol* 2003; **57**: 80–5.



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**doi: 10.1192/bjp.197.4.333**


## Cannabis and psychosis

We read with interest the recent study by Henquet and colleagues.[1] As well as providing further support for the well-established theory that cannabis may worsen or re-awaken psychosis in vulnerable adults, this study reports the fascinating and novel finding that cannabis appears to differentially affect mood – with patients with a psychotic disorder, but not controls, reporting improvements in negative affect following cannabis use. On the other hand, cannabis enhanced positive affect in patients and controls alike.

Previous studies have been contradictory regarding the effects of regular cannabis use on mood. Denson & Earleywine found that regular users reported less depressed mood and more positive affect than non-users,[2] whereas Degenhardt and colleagues reported that heavy cannabis use and depression were associated.[3] The reason for these differences is not clear, but may be due to differences in cannabis composition, as pure delta-9-tetrahydrocannabinol is anxiogenic when given acutely, whereas cannabidiol appears to ameliorate these effects.[4]

The finding that patients derived more benefit from cannabis use in terms of mood suggests that the association of early cannabis use with subsequent onset of psychosis may not, in fact, be a causative relationship as previously reported.[5] Rather, early cannabis use in these (already vulnerable) individuals may be more likely as they derive more benefit – in terms of mood enhancement – than individuals who are not at risk of psychosis. Henquet and colleagues also report that the effects on mood are acute, whereas effects on psychosis are subacute. It would be interesting to determine whether the effects on mood and psychosis occur with equal frequency earlier in the illness, because if psychosis emerges only with repeated dosing, this may be a further maintaining factor in early use.

Regardless of the aetiological relationship of cannabis use to psychosis onset, this study highlights an important point – people take cannabis because they feel that they derive benefit from it, and patients with psychosis are no different in this respect. In terms of clinical practice, this paper highlights one reason why service users may continue to smoke cannabis, despite the fact that it clearly worsens their psychotic symptoms. This awareness can add to our understanding and attitude towards the service user, and enable us more creatively to help the service user find alternative ways to boost their mood.


1 Henquet C, van Os J, Kuepper R, Delespaul P, Smits M, à Campo J, et al. Psychosis reactivity to cannabis use in daily life: an experience sampling study. *Br J Psychiatry* 2010; **196**: 447–53.

2 Degenhardt L, Hall W, Lynskey M. Exploring the association between cannabis use and depression. *Addiction* 2003; **98**: 1493–504.

3 Denson TF, Earleywine M. Decreased depression in marijuana users. *Addict Behav* 2006; **31**: 738–42.

4 Bhattacharyya S, Morrison PD, Fusar-Poli P, Martin-Santos R, Borgwardt S, Winton-Brown T, et al. Opposite effects of delta-9-tetrahydrocannabinol and cannabidiol on human brain function and psychopathology. *Neuropsychopharmacology* 2010; **35**: 764–74.

5 Moore TH, Zammit S, Lingford-Hughes A, Barnes TR, Jones PB, Burke M, et al. Cannabis use and risk of psychotic or affective mental health outcomes: a systematic review. *Lancet* 2007; **370**: 319–28.



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**doi: 10.1192/bjp.197.4.333a**