

Hotopf *et al* recalculated the post-randomisation effect for longer-term OPC in what they refer to as our ITT sample, rather than the sample we actually used. They say the effect is not significant but their calculation excludes the historically violent subgroup.

For hospital outcomes, unlike violence, we obtained follow-up information on the entire ITT sample through admission records. Here we found a statistically significant experimental result. For any month during the study year, the randomly assigned OPC group had a lower risk of readmission than the control group (OR=0.64, $P < 0.01$). Hotopf *et al* do not mention this finding.

About one-third of the OPC group had their court orders expire very early in the study – during the first or second month – and more of these individuals were rehospitalised than those remaining on OPC, which explains the early separation of the lines in the figures from Swartz *et al* (1999).

Swanson, J. W., Swartz, M. S., Wagner, H. R., et al (2000) Involuntary out-patient commitment and reduction of violent behaviour in persons with severe mental illness. *British Journal of Psychiatry*, **176**, 324–331.

Swartz, M. S., Swanson, J. W., Wagner, H. R., et al (1999) Can involuntary outpatient commitment reduce hospital recidivism? Findings from a randomized trial with severely mentally ill individuals. *American Journal of Psychiatry*, **156**, 1968–1975.

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Psychosocial interventions for self-harm

Crawford *et al* (2007) conclude that the results of their meta-analysis ‘do not provide evidence that additional psychosocial interventions following self-harm have a marked effect on the likelihood of subsequent suicide’. This conclusion is far too bold considering the weaknesses inherent in the analytical approach employed. In my opinion Crawford *et al* have not allowed adequate weight for several methodological problems, the most prominent being the rationale for including studies in the analysis.

They acknowledge the ‘lack of statistical power’ in the meta-analysis but offer a definitive and sweeping conclusion.

The lack of statistical power is only one reason not to conduct the meta-analysis. The central rationale for clustering the included studies is seriously flawed. Not only have they mixed simple interventions and treatments, the target populations range from latency-age children (some as young as 12 years) to older adults (>50 years), intervention methods and theoretical orientations vary considerably (employing individual, group, case-management and home-based care), samples include those making suicide attempts as well as those engaging in self-harm (non-suicidal) behaviour, and they have also included studies that employed questionable intervention or treatment protocols for suicidality. A review of the intervention and treatment protocols of the studies included reveals wide variability in the nature, oversight and fidelity of the services being offered. I have serious concerns about at least 8 of the 19 study protocols. Some of the interventions cannot realistically be described as appropriate for suicidality, at least from the perspective that they have a serious chance of reducing subsequent pathology of suicide attempts, much less actual deaths. For example, Harrington *et al* (1998) employed four home visits by a social worker. Similarly, Guthrie *et al* (2001) included four sessions delivered in the patient’s home. Cedereke *et al* (2002) explored the utility of random telephone interventions and Clarke *et al* (2002) included ‘management enhanced by nurse-led case management’. As these examples illustrate, not all psychosocial interventions are the same, something Crawford *et al* (2007) failed to clarify in their article. Why would we expect that a meta-analysis of randomised trials of interventions or treatments that are this broadly disparate (with samples equally disparate) would actually provide evidence of effective reduction of subsequent suicides?

Meta-analyses have become increasingly popular and increasingly misleading in their findings. Prior to inclusion in a meta-analysis of intervention or treatment outcome, I would suggest a thorough review of the intervention/treatment approach and related fidelity. Only those studies meeting strict and predefined criteria should be included. When considering strategies for including and clustering treatment studies for meta-analysis, it is

particularly important to consider the targeted problem or disorder. Many, if not most problems targeted by psychosocial interventions and treatments are recurrent, persistent and potentially chronic in nature. Hence, the need for careful scrutiny of studies included.

Compounding the problems noted above, the follow-up periods for all of the studies included by Crawford *et al* ranged from 6 to 12 months. The efficacy of treatment or interventions for suicide will only be known after 5, 10 or 20 years. In shorter-term studies even if the results did show a reduction in subsequent suicides, we would not know whether the interventions or treatments were ‘delaying’ suicide or actually preventing it without longitudinal data.

There are many other factors that need to be scrutinised prior to inclusion of studies in a meta-analysis (e.g. sample size, categorisation of attempt status and suicide intent, fidelity/oversight of intervention or treatment) but space does not allow a full discussion. The point is that identifying appropriate inclusion criteria for such a study is a complex process which is far more complicated than simply taking all randomised controlled trials.

The definitive nature of the conclusion offered by Crawford *et al* belies the current state of the science in this area. In an age when legislators and funding agencies rely on science for direction, studies like this one generate ill-informed conclusions on what interventions, treatments and approaches to suicide prevention offer the most promise. Many readers will sadly and mistakenly carry away the message that psychosocial interventions offer no promise to reduce suicide rates.

Cedereke, M., Monti, K. & Ojehagen, A. (2002)

Telephone contact with patients in the year after a suicide attempt: does it affect treatment attendance and outcome? *European Psychiatry*, **17**, 82–91.

Clarke, T., Baker, P., Watts, C. J., et al (2002)

Self-harm in adults: a randomized controlled trial of nurse-led management versus routine care only. *Journal of Mental Health*, **11**, 167–176.

Crawford, M. J., Thomas, O., Khan, N., et al (2007)

Psychosocial interventions following self-harm: systematic review of their efficacy in preventing suicide. *British Journal of Psychiatry*, **190**, 11–17.

Guthrie, E., Kapur, N., Mackway-Jones, K., et al (2001)

Randomized controlled trial of brief psychological intervention after deliberate self-poisoning. *BMJ*, **323**, 135–138.

Harrington, R., Kerfoot, M., Dyer, E., et al (1998)

Randomized trial of a home-based intervention for children who have deliberately poisoned themselves.

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Author's reply: Professor Rudd raises important questions about whether it was appropriate to undertake this meta-analysis given the nature of interventions studied and the length of follow-up periods used. We believe that it can be appropriate to synthesise data from randomised trials to examine clinically important rare outcomes that individual studies are unlikely to be able to detect. For instance, psychosocial interventions for alcohol misuse are effective in reducing alcohol consumption but a range of factors, including clinical inertia, mean that they are not widely delivered. By synthesising data from trials conducted in a range of different settings, Cuijpers *et al* (2004) demonstrated that they are associated with a 30% reduction in subsequent mortality, a finding which may help to overcome some of the barriers to their delivery.

Although none of the studies we examined set out specifically to try to reduce suicide, it seems logical that interventions that are designed to reduce the incidence of suicidal behaviour should have an impact on the likelihood of fatal as well as non-fatal self-harm. Although several studies we included involved only brief interventions, such interventions have been shown to reduce the rate of suicide in other contexts, for instance in the period following discharge from in-patient psychiatric care (Motto & Bostrom, 2001).

Most of the studies we included followed people for between 6 and 12 months after the initial episode of self-harm. Although this is a relatively short period it is also the period during which suicide is most likely to occur (Owens *et al*, 2002). By focusing on the period immediately following an episode of self-harm we maximised the likelihood of being able to demonstrate an impact on the rate of suicide.

However, we fully endorse Professor Rudd's comment that the results of our meta-analysis need to be interpreted with caution. Lack of data on suicide deaths in several of the trials that we identified meant that study power was limited. This resulted

in wide confidence intervals around the pooled difference in suicide rates and it is therefore possible that additional psychosocial interventions do lead to reductions in subsequent suicide.

Cuijpers, P., Riper, H. & Lemmers, L. (2004) The effects on mortality of brief interventions for problem drinking: a meta-analysis. *Addiction*, **99**, 839–845.

Motto, J. A. & Bostrom, A. G. (2001) A randomized controlled trial of postcrisis suicide prevention. *Psychiatric Services*, **52**, 828–833.

Owens, D., Horrocks, J. & House, A. (2002) Fatal and non-fatal repetition of self-harm. Systematic review. *British Journal of Psychiatry*, **181**, 193–199.

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Psychiatric disorder and looked after status

Ford *et al* (2007) investigated the possible explanations for the increased prevalence of psychiatric disorder in children looked after by local authorities and linked looked after status with higher levels of psychopathology, educational difficulties and neurodevelopmental disorders. They suggested that services should bear in mind that a change of environment might be appropriate in providing help, at least in some cases.

After carefully reading the article, I think that Ford *et al* have missed an important aetiological factor: the influence of genetics. Studies (e.g. Howard *et al*, 2001) have shown that children of parents with mental disorder are likely to be looked after by another person or organisation. Biological factors which caused mental illness in the parents of children currently looked after by services might operate to cause the increased prevalence of psychiatric disorder in these children. Hence by neglecting the biological component of the bio-psychosocial model of mental illnesses, Ford *et al* have failed to provide a comprehensive assessment of causative factors in these children.

The authors could have included psychiatric disorder in the parents as a variable and divided the looked after group into

children of parents with or without mental disorder. Ford *et al* have identified that neurodevelopmental disorders and learning difficulties are associated with increased prevalence of psychiatric disorder. Both are also associated with the future development of mental illnesses such as schizophrenia (Done *et al*, 1994; Lawrie *et al*, 2001) in which genetic factors play an important aetiological role (Cardno *et al*, 1999).

Cardno, A. G., Marshall, E. J., Coid, B., et al (1999) Heritability estimates for psychiatric disorders: the Maudsley twin psychosis series. *Archives of General Psychiatry*, **56**, 162–168.

Done, D. J., Crow, T. J., Johnstone, E. C., et al (1994) Childhood antecedents of schizophrenia and affective illnesses: social adjustment at ages seven and eleven *BMJ*, **309**, 699–703.

Ford, T., Vastanis, P., Meltzer, H., et al (2007) Psychiatric disorder among British children looked after by local authorities: comparison with children living in private households. *British Journal of Psychiatry*, **190**, 319–325.

Howard, L. M., Kumar, R. & Thornicroft, G. (2001) Psychosocial characteristics and needs of mothers with psychotic disorders. *British Journal of Psychiatry*, **178**, 427–432.

Lawrie, S. M., Byrne, M., Miller, P., et al (2001) Neurodevelopmental indices and the development of psychiatric symptoms in subjects at high risk of schizophrenia. *British Journal of Psychiatry*, **178**, 524–530.

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Authors' reply We totally agree with Dr Sekar's point that biological factors make an important aetiological contribution to the development of psychiatric disorder in children. We certainly did not intend to suggest that biological factors are any less important than psychological or social factors. Many childhood disorders are known to have a high level of heritability (Rutter *et al*, 2006). However, we should not forget that both our and previous studies suggest that similar risk factors operate in looked after children as in children living in private households, but that looked after children tend to have been exposed to more of them, sometimes at greater intensity (Stein *et al*, 1996; Ford *et al*, 2007). In our opinion, this includes biological as well as psychological and social factors.

Many studies have shown that parental psychiatric disorder is correlated with childhood psychiatric disorder (Rutter,