

*Journal of the Academy of Child and Adolescent Psychiatry*,  
37, 512–518.

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doi: 10.1192/bjp.191.4.359

**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 synthesis 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,