

such as this using well-designed RCTs that will inform clinical practice.

#### Declaration of interest

G.L. has received payments for lectures from the pharmaceutical industry.

**Bell, A. J., Cole, A., Eccleston, D., et al (1993)** Lithium neurotoxicity at normal therapeutic levels. *British Journal of Psychiatry*, **162**, 689–692.

**G. Lewis** Division of Psychiatry, University of Bristol, Cotham House, Cotham Hill, Bristol BS6 6JL, UK

**N. Stimpson** University of Wales College of Medicine, Cardiff, UK

**N. Agrawal** Chelsea and Westminster Hospital, London, UK

#### Getting closer to suicide prevention

We would like to offer a slightly different perspective from De Leo (2002) on the progress of suicide prevention. There is no argument against suicide representing a complex set of variables. The general method of science, however, is to analyse phenomena in order to find the most simple explanation – the principle formulated by William of Occam in the early 14th century. In the medical paradigm, death results from a disease process. Studying people with heart attacks led to the identification of atherosclerosis as the underlying disease process for the vast majority. Treating myocardial infarctions is important. The development of various approaches to prevention and treatment of atherosclerosis has, however, prevented more premature deaths from heart attacks. Why must one conclude that suicide is a more complicated medical problem than myocardial infarction?

A fundamental discovery was made in the late 1950s (Robins *et al*, 1959): the majority of suicides were committed by people with clinical depression. This finding has been replicated over and over again and we believe that many, like us, have concluded that this connection has been replicated enough to be proven. We have also presented evidence that suicides occur infrequently in people with depression taking antidepressant medication (Isacsson *et al*, 1994).

Thus, in spite of the ‘extreme complexity’ of the phenomenon of suicide, a simple and testable hypothesis can be stated: depression is a necessary cause of most suicides. Based on this proposition, it has

been suggested that effective suicide prevention must focus on improving identification and treatment of depression in the population (Isacsson, 2000). When we look at the declining suicide rates over the past decade or so, we see a great deal of support for that theory. Since the introduction of the new generation of antidepressants during the past 10–15 years, the use of antidepressants has increased up to 5-fold. Concurrently, suicide rates have decreased considerably in many Western countries (e.g. Joyce, 2001). It appears to us that we are getting closer to suicide prevention.

We believe that a lack of focus on depression as the basic disease leading to suicide is most likely the reason why the current decline in suicide rates ‘seems reasonably unrelated to the existence of any national plan’.

#### Declaration of interest

Both authors have delivered lectures at scientific meetings sponsored by pharmaceutical companies.

**De Leo, D. (2002)** Why are we not getting any closer to preventing suicide? *British Journal of Psychiatry*, **181**, 372–374.

**Isacsson, G. (2000)** Suicide prevention – a medical breakthrough? *Acta Psychiatrica Scandinavica*, **102**, 113–117.

—, **Bergman, U. & Rich, C. L. (1994)** Antidepressants, depression, and suicide: an analysis of the San Diego Study. *Journal of Affective Disorders*, **32**, 277–286.

**Joyce, P. R. (2001)** Improvements in the recognition and treatment of depression and decreasing suicide rates. *New Zealand Medical Journal*, **114**, 535–536.

**Robins, E., Murphy, G. E., Wilkinson, R. H., et al (1959)** Some clinical considerations in the prevention of suicide based on a study of 134 successful suicides. *American Journal of Public Health*, **49**, 888–899.

**G. Isacsson** Neurotec, Division of Psychiatry, Karolinska Institute, Huddinge University Hospital, S141 86 Stockholm, Sweden

**C. L. Rich** Department of Psychiatry, University of South Alabama, Mobile, Alabama, USA

**Author’s reply:** There is little doubt that depression has a major role in suicide, being identifiable in approximately 50% of cases (Andersen *et al*, 2001). For this reason, depression is a target in all the national plans that I am aware of.

The role of depression in suicide has been well known since antiquity (Van Hooff, 2000) and this understanding has been largely responsible for the decline in a punitive attitude towards those exhibiting

suicidal behaviour since the Enlightenment. Consequently, the ‘fundamental discovery’ at the end of the 1950s of the role of affective disorders in suicide was far from revolutionary. It is worth remembering that in the vast majority of cases, fortunately, depression does not culminate in suicide. The relative risk for suicide across the lifespan has been recently revised downwards (see, for example, Bostwick & Pankratz, 2000). In addition, a significant percentage of patients who die by suicide appear to have been adequately treated (25% in the experience of Andersen *et al*, 2001). A World Health Organization (1998) technical report has pointed out that optimal treatment of clinical depression would have little impact on global suicide rates, leaving the field open to speculations around more powerful factors in suicide prevention. In any case, the ‘medical paradigm’ is, in my view, only one of many possible perspectives, and needs to be integrated with other disciplines. Clearly, it is not the different prevalence of depression among countries that helps to explain the enormous diversity in rates of suicide that I mentioned in my editorial. Religious, cultural and social factors play very relevant roles in suicidal behaviour. It is in this light that the World Health Organization has correctly endorsed an ecological model, to help both understand and prevent/intervene in suicidal behaviours.

I am aware that Isacsson and Rich, through their research, strongly support the role of the newer antidepressants in preventing suicide. But others are a bit more hesitant in accepting this hypothesis (see, for example, Van Praag, 2002), and maybe lithium has shown more consistent (and convincing) effects, so far, on suicidal behaviour (Tondo *et al*, 2001).

With regard to the comments about a possible overemphasis on the complexities of suicidal behaviour, I am afraid that the philosopher Albert Camus, if he came back to life, would die again on hearing that!

**Andersen, U. A., Andersen, M., Rosholm, J. U., et al (2001)** Psychopharmacological treatment and psychiatric morbidity in 390 cases of suicide with special focus on affective disorders. *Acta Psychiatrica Scandinavica*, **104**, 458–465.

**Bostwick, J. M. & Pankratz, V. S. (2000)** Affective disorders and suicide risk: a re-examination. *American Journal of Psychiatry*, **157**, 1925–1932.

**Tondo, L., Ghiani, C. & Albert, M. (2001)** Pharmacologic intervention in suicide prevention. *Journal of Clinical Psychiatry*, **62** (suppl), 51–55.

**Van Hooff, A. J. L. (2000)** A historical perspective on suicide. In *Comprehensive Textbook of Suicidology* (eds R.W. Maris, A. L. Berman & M. M. Silverman), pp. 96–126. New York: Guilford.

**Van Praag, H. M. (2002)** Why has the antidepressant era not shown a significant drop in suicide rates? *Crisis*, **23**, 77–82.

**World Health Organization (1998)** *Primary Prevention of Mental, Neurological, and Psychosocial Disorders. Suicide*, pp. 75–90. Geneva: WHO.

**D. De Leo** Australian Institute for Suicide Research and Prevention, Griffith University, Mt Gravatt Campus, Queensland 4111, Australia

### Concepts of social capital

McKenzie *et al* (2002) illustrate how emerging conceptions of social capital can help psychiatric researchers study links between social context and the prevalence, course and outcome of psychiatric conditions. Two further considerations deserve a place in this discussion. First, the premise that social capital is ‘a property of groups rather than of individuals’ (McKenzie *et al*, 2002: p. 280) does not enjoy an unqualified consensus. Work by Princeton sociologist Alejandro Portes (1998) summarises the case against insisting that social capital be treated as a group attribute. A more individualist approach draws attention to the important distinction between the social relationships that allow a person to make claims on resources held by others and the resources themselves. A family’s struggle to find a job for a recently hospitalised relative may be eased somewhat when they live in a community with trusting social relationships, but this effect is more limited in a resource-poor community. (For example, Portes (2000) found that alleged effects of social capital on the academic achievement of immigrant children in the USA are drastically reduced when proper controls are used for parental socio-economic status.)

Second, McKenzie *et al* note that high social capital may be found in bad groups, such as the Mafia, and in homogeneous groups that restrict the freedom of members or exclude outsiders and minorities. This analysis of negative consequences can be expanded by an individual-oriented discussion of a dilemma familiar to clinicians working with socially marginal populations. Individuals may indulge in apparently irrational spending sprees to buy food, drugs or alcohol for companions

because these allow them to make future claims for reciprocity when times are lean (Dordick, 1997). The resulting mutual obligations can make it difficult for even a highly motivated person to enter (or re-enter) the social mainstream because he or she is vulnerable to criticism for breaking ranks with compatriots (Bourgois, 1995) or to claims on cash resources saved to facilitate an exit (for tuition, a new apartment, etc.). Programmes serving these populations need to devise strategies to help patients manage this dynamic aspect of social capital, even as they focus on recovery.

**Bourgois, P. (1995)** *In Search of Respect: Selling Crack in El Barrio*. New York: Cambridge University Press.

**Dordick, G. (1997)** *Personal Relations and Survival among New York’s Homeless*. Philadelphia, PA: Temple University Press.

**McKenzie, K., Whitley, R. & Weich, S. (2002)** Social capital and mental health. *British Journal of Psychiatry*, **181**, 280–283.

**Portes, A. (1998)** Social capital: its origins and applications in modern sociology. *Annual Review of Sociology*, **24**, 1–24.

— (2000) The two meanings of social capital. *Sociological Forum*, **15**, 1–12.

**J. Walkup** Institute for Health, Health Care Policy, and Aging Research, Rutgers University, 30 College Avenue, New Brunswick, NJ 08903, USA

**Author’s reply:** The problem with the emerging concept of social capital is that it is in danger of trying to be all things to all people. Dr Walkup is correct to point to the view of Portes and others that social capital can be individual. I do not think that this approach is particularly useful. Social capital is not a thing, it is a way of trying to describe a number of social processes. It is a theory that helps us understand what is happening in a society. Although there may be analogous processes occurring at group and individual levels, conceptualising them as the same thing is problematic.

Theories of causation argue that causes at different levels are often governed by different rules and need different methods of investigation. An example would be the effects of smoking on health. This can be investigated at a number of levels; there would be the cellular level (the effects of nicotine on the cell), the individual level (physical and psychological effects of smoking and addiction) and the group level (what increases smoking levels in one group compared with another).

One would not try to employ the concept of cellular biology to investigate groups of people and one would not try to use group or systems approaches to investigate the individual. Moreover, the factors that increase the level of smoking in a group may not be the same as those that increase an individual’s risk of smoking-related disease.

Given that group social processes are likely to affect health in different ways from individual processes, it would not seem helpful to consider social capital as a single entity that works at both levels. A choice has to be made and the choice of the majority is to conceive of social capital as operating at an ecological or group level and to consider effects at an individual level as social networks.

Dr Walkup is correct to point to the differences between the social relationships that allow a person to call on resources, and the resources themselves. However, the theory of social capital as an ecological variable does allow for this. Bonding and bridging social capital describe factors at the community level, but the concept of vertical social capital attempts to describe the ability of a community to facilitate access to resources from those in power.

Clearly, in our individualised world our interventions tend towards helping people decrease their risk of illness and their risk of relapse, and improve their participation in the world. The exciting difference about ecological conceptualisations is that they are about how society decreases the risk of illness and relapse of its population and how society facilitates the participation of the individual. These approaches aim for the same outcome but they are not the same thing and will need different conceptualisations, investigations and interventions.

**K. McKenzie** Department of Psychiatry and Behavioural Sciences, Royal Free and University College Medical School, Royal Free Campus, Rowland Hill Street, London NW3 2PF, UK

### Vulnerable individuals and the Human Rights Act

With reference to the recent editorial on the Human Rights Act and mental health legislation (Bindman *et al*, 2003), the ‘steady trickle’ of human rights cases rather than a flood is not surprising when considered in context of the history of UK human rights.