Correspondence

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Hot weather and suicide: a real risk or statistical illusion?

Page et al (2007) reported an association between increased risk of suicide and hot weather. We believe it is important that this finding is compared with similar associations reported in other countries and under similar conditions, particularly for countries with hotter climates but also for those moving through a period of climatic change.

We are a little disappointed that despite the authors’ excellent statistical analyses and effective display of results, they determined the shape of their natural cubic splines ‘visually’ instead of using some model selection criterion, for example likelihood ratio tests, Akaike’s information criterion (AIC), etc. Page et al also stated that Yip et al (2000) ‘failed to show any significant seasonality with recent UK data’. This may not be entirely accurate as we believe that Yip et al (2000) showed a decreasing seasonal pattern but not that the pattern had vanished.

The ‘unexpected’ reduction in suicide during the heatwave of 2003 reported by Page et al is difficult to explain on the basis of temperature alone, particularly as there was a 13.5–33% increase in general mortality during the 2003 heatwave (Kovats et al, 2006). It is clear that the association of increased mortality with high temperature is not specific to suicide. Hajat et al (2002) reported an almost identical increase in all-cause mortality of 3.34% (95% CI 2.47–4.23) for every 1Â°C increase in mean temperature above 18Â°C compared with the 3.8% increase in suicide reported by Page et al. This raises the possibility of an unaccounted confounder linking suicide, total mortality and daily mean temperature above 18Â°C. Such factors include climatic and non-climatic factors, whether acting independently or as interaction terms, for example number of sunshine hours (Salib & Gray, 1997), relative humidity, rainfall, unusual weather conditions, stress resulting in changes in the hypothalamic–pituitary–adrenal axis or even changes in the solar wind as measured by satellites (Richardson et al, 1994). Chronomics of suicides (Halberg et al, 2005) which do not rely on calendar year but on periodicity of solar wind (Richardson et al, 1994) may provide a plausible and alternative explanation to the findings of Page et al.

Perhaps the only conclusion that can be drawn from reading Page et al’s paper is that high temperature may be associated with increased all-cause mortality. Given the very similar rate of increase in all-cause mortality and in suicide, the mechanism by which high temperature affects the rate of suicide should not be expected to differ from that operating for other causes of death.

Although high daily mean temperature may increase suicide risk, this is not an independent risk factor and may not have the implications for public health policy in relation to global warming that Page et al indicated.


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Authors’ reply: We agree that our findings need to be replicated in other populations and climates. Salib et al may have misinterpreted our analysis of suicides during the 2003 heatwave, as our finding of –1.8% (95% CI −17.8 to 18.4) change in suicides from expected is consistent with no change rather than a reduction. We discuss reasons for this lack of effect and point out that the lack of power in this calculation leads to an imprecise estimate.

We disagree with Salib et al’s assertion that the effect of high temperature on all-cause mortality (rather than suicide specifically) is a reasonable explanation for our findings. We only examined deaths from suicide and undetermined intent, so it is not possible for other causes of death to have ‘confounded’ our results. We considered carefully which confounders to include in our models. Individual-level confounders, for example the effect of individual stress on the hypothalamic–pituitary–adrenal axis, are irrelevant in a time-series analysis as they do not vary day to day across a population. Sunshine hours were sufficiently accounted for by including a term for hours of daylight. We think it unlikely that any of the other potential confounders mentioned by Salib et al could be sufficiently associated with both temperature and suicide to explain our findings. Also, humidity, rainfall and unusual weather conditions (e.g. thunderstorms) tend to vary regionally more than temperature, meaning that exposure misclassification would be a problem in a countrywide analysis. The role of solar winds in the aetiology of suicide is highly speculative.

Higher temperatures affect mortality through a range of mechanisms (Bouchama & Knochel, 2002). Cardiovascular and respiratory deaths during periods of high temperature are caused by physiological changes, including increased coagulation,
dehydration and increased cardiovascular output – particularly important in the elderly or those with pre-existing disease. A range of antipsychotic drugs are known to inhibit sweating and therefore thermo-regulation. Recent work has shown that deaths from respiratory and external causes are particularly increased at high temperatures (Hajat et al., 2007). Further research is needed on the pathophysiology of heat, but it is clear that persons with mental illness remain a high-risk group for heatwave mortality (Kovats & Ebi, 2006).

Avoiding errors about ‘margins of error’

When discussing actuarial risk assessment instruments (ARAI), Hart et al (2007) acknowledge that ‘prediction’ may refer to probabilistic statements (e.g. a ‘prediction’ that an individual ‘falls in a category for which the estimated risk of violence was 52%’: p. s60). For unclear reasons, however, the authors seem to value only predictions with right or wrong outcomes. They therefore regard statements about future behaviour of large groups (where one can be almost certain that the fraction of persons who act a certain way will fall within a narrow range of proportions) as potentially ‘credible’, but predictions for individuals as meaningless.

If the purpose of risk assessment is to make choices, then well-grounded probabilistic predictions about single events help us. Suppose we conclude that it is legally and ethically acceptable to impose preventive confinement upon individuals in ARAI categories with estimated recidivism rates above a specified threshold. This policy entails making ‘false-negative’ and ‘false-positive’ decision errors. We recognise, however, that unless we are omniscient perfection is not an option and ARAs simply help us make better decisions than we otherwise could.

How do ‘margins of error’ in estimated recidivism rates affect our decision process? Hart et al believe their ‘group risk’ and ‘individual risk’ 95% confidence intervals speak to this problem. Their group intervals are standard confidence intervals for estimated population proportions based on random samples. If the threshold lies outside the group risk confidence interval for a category, then we can be reasonably certain that a decision we make concerning someone in that category is the same decision we would make if we knew the true recidivism rate for that category. If the threshold falls within a category’s risk confidence interval, then our estimate quite possibly might lead to the ‘wrong’ decision. Statistical decision theory (Berger, 1985) shows, however, that it is still a sensible strategy to choose whether to confine a member of a category based on which side of the threshold our estimated risk falls.

Hart et al talk about ‘individual risk’ as though it is something different from category (or ‘group’) risk. Yet if all one knows about an individual is his membership of a risk group, what can ‘individual risk’ mean? The authors do not say. If ‘individual risk’ refers to believed-to-exist-but-unspecified differences between individuals within a category, such differences should not affect choices by a rational decision-maker. The 95% CIs for ‘individual risk’ pile nonsense on top of meaninglessness. Hart et al describe the replacement of ‘n’ by ‘1’ in the Wilson (1927) formulae as ‘ad hoc’, but this substitution makes no sense when the basis for the estimated proportion is an n-member sample. With ‘1’ in place of ‘n’, the formulae just don’t mean anything.

Using ARAs raises serious moral problems as well as the valid scientific questions that Hart et al mention. But in faulting the capacity of ARAs to address an unspecified quantity called ‘individual risk’, and in dressing up this notion with misapplied formulae for confidence intervals, Hart et al ultimately create a muddle.


Authors’ reply: Actuarial risk assessment instruments (ARAs), constructed using data from known groups, are used to make life-and-death decisions about individuals. How precisely do they estimate risk in individual cases? The 95% CI for proportions, which evaluates the precision of risk estimates for ARAI groups, cannot be used for individual risk estimates unless one makes a very strong assumption of heterogeneity – that ARAs carve nature at its joints, separating people with perfect accuracy into non-overlapping categories. No one, not even those who construct ARAs, makes this assumption. So, we ask again, what is the precision of individual risk estimates made using ARAs?

Mossman & Sellke criticise us for inadequately defining ‘individual risk’ and for using an ad hoc procedure to estimate the margin of error for individual risk estimates, which they opine served only to ‘pile nonsense on top of meaninglessness’. We must plead guilty to some of the charges levelled by Mossman & Sellke – indeed, we did so in our paper, acknowledging the conceptual and statistical problems with the approach we used. In our defence, we claimed duress: because developers used inappropriate statistical methods to construct ARAs, we could not use appropriate methods to evaluate them. Violent recidivism was measured in the ARAI development samples as a dichotomous, time-dependent outcome, and so the developers ought to have used logistic regression or survival analysis to build models; if they had, one could directly calculate logistic regression or survival scores for individuals and their associated 95% CIs.

But we also plead that these charges are irrelevant to our conclusion. As we discussed, to reject our findings that the
Austrian firearms: data require cautious approach

We note with interest Kapusta et al’s (2007) report on firearm suicide and homicide following legislative reform in Austria. However, a note of caution must be applied to statements concerning apparent consistency between Australian and Austrian experiences with firearm legislation.

Recent work demonstrates that Australia’s 1996 gun laws had no significant impact on firearm homicide but that the pre-existing decline in firearm suicide accelerated post-reforms (Chapman et al., 2006; Baker & McPhedran, 2007). There has been an accompanying decline in non-firearm suicides beginning in the late 1990s.

Unfortunately, these findings may require re-evaluation owing to issues of data quality. The Australian Bureau of Statistics (ABS), a primary data source for researchers in the field, appear to be ‘over-counting’ unintentional deaths and ‘under-counting’ suicides. De Leo (2007) showed that ABS data ‘under-counted’ the total number of suicides (all methods) in one Australian State (Queensland) by 127 cases in 2004 alone. Re-analysis of the updated data reduced the apparent downward trend in suicides that had emerged from previous analyses. This finding has significant implications for assessment of suicide prevention initiatives in Australia, given that most assessments are based on ABS data.

Consequently, it has been suggested that the incidence of firearm suicide in Australia may be higher than thought, and, if so, then studies using ABS suicide figures merit re-evaluation (McPhedran & Baker, 2007). In addition, the National Injury Surveillance Unit has questioned the accuracy of homicide data, which suggests that firearm homicides may also be higher than ABS data show. There are growing calls for ABS data to be cross-checked against coronial records and for ABS records to be updated where discrepancies are found.

Although this situation does not bear directly upon the findings of the Austrian study, other than reinforcing the importance of quality control, it demonstrates that drawing conclusions about the impact or otherwise of restrictive firearm legislation in Australia may be premature.

Effective public health initiatives need to be built on accurate information. We therefore caution researchers against citing Australian figures during wider discussions of the possible role of firearm legislation in public health strategies, until and unless full data accuracy can be guaranteed.


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In Australia, the 1996 National Firearms Agreement (NFA) was introduced following the Port Arthur massacre, in which 35 people were killed. The NFA introduced access restrictions (particularly of assault weapons), storage regulations and a gun buy-back scheme to reduce firearms in the community. The recent killings at Virginia Tech have fuelled the debate on the causal impact of the NFA, with rates of homicides virtually unchanged but substantial reductions in numbers and rates of firearm suicide (Chapman et al., 2006). However, the dramatic decrease in suicide deaths by firearms in Australia began prior to 1996.

In Queensland, on the basis of the Queensland Suicide Register (QSR), rates of firearm suicide in 1994 were more than 30% less than those recorded in 1990 (approximately 10 in 100 000). In addition, in 1994 there was a crossing-over between declining rates of firearm suicide and increasing rates of hanging suicide. Both trends between 1990 (year of constitution of the QSR) and 2004 showed statistically significant variations (R^2=0.88 for firearms and R^2=0.70 for hanging), with firearm suicide being more than 5 times less frequent than hanging suicide in 2004 (it was 2 times more frequent in 1990). Most firearm suicides involved hunting rifles, the use of which started to appear strongly reduced by early 1990s. Minor declines were recorded in the use of other weapons.

Kapusta et al. (2007) underline the successful effect of the Austrian reform on firearm use on both homicide and suicide rates; moreover, they did not witness any increase in suicide with other methods. We believe this has not happened in Queensland, where the current legislation has not restricted firearms within the community (around 500 000 in four million inhabitants) and there has not been a reduction in male suicide rates (De Leo et al., 2006). However, a big shift in the choice of suicide methods has occurred, with younger males increasingly choosing hanging. As pointed out by Kapusta et al., causality remains speculative in this type of observation. Although controlling access to means remains of paramount importance in suicide prevention (De Leo, 2002), it seems that a change in societal and cultural views towards firearms has played a bigger role than the NFA. To verify this, we are currently checking if those who died by suicide through other methods were also in possession (and/or had availability) of a firearm at the time of their death.

We agree with McPhedran & Baker that Australian firearm laws should be re-evaluated on the basis of more reliable data, but as long as sufficient evidence is not available, theoretical assumptions that Australian firearm laws had no life-saving effects remain speculative. This applies also to Europe where independent scientific evaluations of firearm law are still rare.

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Cardiovascular risk with antipsychotics: case–control study or survey?

Mackin et al (2007) highlighted the significantly higher risk of cardiovascular and metabolic diseases in people with severe mental illness. Rates of metabolic syndrome and cardiovascular risk similar to those in schizophrenia have been reported in bipolar disorders, and atypical antipsychotics have been approved for the treatment of the latter (Fagiolini et al, 2003; Birkenaes et al, 2007). This implies that all such populations should be studied for putative long-term adverse outcomes, as in the timely study of Mackin et al (2007).

However, some methodological issues need clarification. Mackin et al state that their study is a case–control study, but by definition a case–control study starts with an outcome and investigates exposure to putative risk factors in groups with and without the outcome (Lewallen & Courtright, 1998), generating a measure of relative risk with regard to a given risk factor. Mackin et al started with a group with mental illness on antipsychotics and studied the prevalence of metabolic disease and cardiovascular risk compared with controls. Thus the study has really used a survey design with a control group. The use of a control group alone does not justify the label of a ‘case–control study’.

As an important corollary of this distinction, the sample size is rather low for a community-based survey. Mackin et al mention that comparative data for physical comorbidity in people with diagnoses other than schizophrenia are sparse; unfortunately, the study fails to generate such data owing to the inadequate sample size. We feel that Mackin et al have gone beyond their brief to analyse the effect of individual factors such as diagnoses, type of antipsychotic and smoking; not surprisingly, they failed to emerge with convincing findings as the sample was underpowered to generate such data.

Finally, we wonder whether the inclusion of several patients with depression and anxiety is appropriate for a study on ‘severe mental illness’, a term traditionally reserved for psychotic and bipolar disorders. The common denominator seems to be ‘treated with antipsychotics’ rather than ‘severe mental illness’. It is interesting to note that the type of antipsychotics had no impact on the outcome measures (except serum insulin). If replicated in a much larger community sample, this so-called negative finding could have far-reaching implications regarding choice of treatment. Another important factor in the secondary analysis could have been the duration of treatment with antipsychotics and the dosages used. In a recent study, higher doses of medication were associated with increased cardiovascular risk scores (Osborn et al, 2006). Including the dosage and duration of antipsychotics in the analysis could provide important insights regarding the true impact of antipsychotics on the outcome measures.

This study, like several others, reiterates that patients treated with antipsychotics are at heightened risk for cardiovascular events and metabolic syndrome. Longitudinal studies are needed to explore the relative contribution of putative aetiological factors to physical comorbidity in severe mental illness.


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Authors’ reply

Banerjee & Basu are correct to point out that our study does not have a case–control design in the purest epidemiological sense, that is a study in which patients who have developed a disease are identified and their past exposure to aetiological factors is compared with that of controls. Our study is conceptually similar to the case–control design, although we accept that both our study and case–control studies are inherently vulnerable to methodological weaknesses as discussed recently (Lee et al., 2007). We selected individuals who had had a diagnosis of severe mental illness and antipsychotic treatment to ascertain whether this population was at increased risk for cardiovascular and metabolic disease compared with a control group.

We also accept, and acknowledge in our paper, that the sample size is relatively small. However, we found highly statistically significant differences between patients and controls across a number of outcomes. The analysis of effect of diagnosis, type of antipsychotic medication and smoking status was not a primary aim but was a secondary analysis. Increasing the sample size further might have added power to the study to detect differences in these variables. Notwithstanding, emerging evidence from studies in people with bipolar disorders points to an excess of cardiovascular and metabolic disease comparable to that in schizophrenia, suggesting that the similar rates across our diagnostic groups is a true finding.

Banerjee & Basu question the appropriateness of the term ‘severe mental illness’. The vast majority (75.6%) of patients had a diagnosis of schizophrenia, bipolar disorder or schizoaffective disorder. Many in the ‘other’ category (comprising 24.4%) had experienced psychotic depression and other severe depressive disorders requiring antipsychotic treatment. Although the use of the word ‘severe’ may be questioned, we are confident that this is appropriate.

We await larger, prospective studies designed specifically to tease out the aetiological factors that contribute to an excess of cardiovascular and metabolic risk and ultimately an excess mortality rate in this population. We hope our study has gone some way to highlighting the need for vigilant monitoring and appropriate intervention in this high-risk group.

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Psychiatry and faith-based organisations

Leavey & King (2007) provide a useful overview of the relationship between the clergy and psychiatry. Although I wholeheartedly agree that there should be partnerships between psychiatry and religious sectors, I found the discussion to be one-sided. Leavey & King say little about what religion can offer psychiatry. It is well recognised that religious states are often misdiagnosed as mental illness (Dein, 2004). Religious professionals can play a pivotal role in teaching mental health professionals about normative religious experiences and thus enable them to make better informed diagnoses. It is not just that religious professionals need to be educated about mental illness but it is also vital that psychiatrists understand religious experience.

Of course ideas about mental illness reflecting sin still exist in some religious communities, but in my own fieldwork among Orthodox Jews and Pentecostal Christians it is evident that rabbis and pastors increasingly recognise mental illness as a state independent of moral indiscretion. Even in communities where extreme religious experiences, such as hearing God’s voice, are prevalent, religious leaders are able to differentiate these experiences from the symptoms of severe mental disorder (Dein & Littlewood, 2007). Similarly they are able to differentiate psychoses from states of spirit possession (which themselves require stringent criteria for their diagnosis within the religious context).

Beyond this, there is emerging evidence that religion can play an important role in facilitating coping with life stressors (Pargament, 1997). Mental health professionals need to be knowledgeable about the circumstances in which referrals to religious professionals are appropriate. They should be aware that for religious believers, prayer and ritual may play a central role in the healing process. Of course involvement in such activities may influence pathways to care but there is ample evidence that religious and biomedical forms of healing can work well together: biomedicine healing the body and religion healing the soul (Littlewood & Dein, 1995).

Finally I take issue with the statement that biomedical and spiritual models of illness are necessarily conflicting. Spirituality and biomedicine offer different types of explanations for patients’ problems. The art of medicine should be to learn how to combine different treatments in order to provide more holistic care to patients.


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Finally I take issue with the statement that biomedical and spiritual models of illness are necessarily conflicting. Spirituality and biomedicine offer different types of explanations for patients’ problems. The art of medicine should be to learn how to combine different treatments in order to provide more holistic care to patients.


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Faith and other religious terms are still not considered an explicit language of psychiatry. Faith is a multilayered phenomenon, involving a belief about things of which we are uncertain accompanied by an expectancy and/or conviction (Clarke, 2003). The Bible defines faith as being sure of what people hope for and certain of what they do not see (Hebrews 11:1). Faith is considered a nebulous concept and its
benefit is beyond objective scientific measurement. Despite this the World Health Organization clearly stresses the value of concepts such as faith, hope and compassion in the healing process from any illness (World Health Organization, 1998). Religions based on strong faith and beliefs have evolved and persisted over centuries, and people do turn to religion when coping with life stressors (Pargament, 1997). If this spiritual craving is to be utilised for promoting better mental health and holistic care, stronger collaboration between psychiatrists and religious professionals is important (Rattray, 2002; Dein, 2004).

It is encouraging to note, despite the constant suspicion between psychiatry and religion (Bhugra, 1997), the incorporation of religious principles based on faith into treatment strategies especially in psychotherapy, and this should be welcomed. Christian principles have been effective in helping to improve spiritual well-being (Lipsker & Oordt, 1990; Hawkins et al., 1999). The third-wave cognitive–behavioural therapies such as dialectical behaviour therapy approach, acceptance and commitment therapy, and mindfulness-based cognitive therapy are closer to religious belief systems and practices (Andersson & Asmundson, 2006). Zen Buddhist ideas have been woven into the fabric of dialectical behaviour therapy (Robins, 2002). Acceptance and commitment therapy connects with the Buddhist philosophy and practices in accepting the four noble truths and the eight-fold noble path (Hayes, 2002). The mindfulness-based therapies have stronger associations with Buddhism, its empirical database and its application for stress reduction, health promotion and improved personal functioning (Robins, 2002). This gives an early hope that mainstream psychiatry and religions with strong faith and belief systems can work together to ameliorate psychopathology and improve the well-being of patients.


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Authors’ reply: Dr Dein argues that our editorial (Leavey & King, 2007) on collaboration between psychiatry and religion is biased because we failed to discuss what religion has to offer psychiatry. We feel that many faith-based organisations and their clergy contribute much towards human welfare and healing and we would have been happy to discuss this in more detail. However, the focus of the paper was to highlight the potential barriers and dangers arising from partnerships between religion and psychiatry. Our own research with clergy has helped clarify some of these issues (Leavey, 2007; Leavey et al, 2007). Thus, clergy of all sorts find themselves perplexed by people with mental health problems and appear to be generally untrained and unsupported by both their own organisations and by mental health services. Although some clergy are able to distinguish religious from psychiatric phenomena, others are not. Dr Dein’s reference to his study of lay members of a White Pentecostal congregation does not relate to this issue. Moreover, we did not suggest that biomedical and spiritual models of illness necessarily conflict but in some instances, and among some religious groups, they do. To treat faith communities and their clergy as homogeneous entities is somewhat simplistic. Dr Dein advocates a more holistic approach in medical care, but does he intend this to extend to exorcism and deliverance rituals? This question touches on the central concern of our editorial. We like to talk about inclusivity in psychiatry but it becomes more problematic when clinicians find themselves encouraged to engage with some of the less mainstream aspects of spirituality and religion.

Although we agree with Dr Masil that religion and spirituality, or in his own terms ‘faith’, should be of greater interest to psychiatric practice and research, we cannot agree that ‘its benefit is beyond objective scientific measurement’. Although problems of definition and measurement exist, many health studies of this kind have been undertaken, particularly in the USA where there is less hostility to research on spirituality and health than in the UK.


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