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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 1°C increase in mean temperature above 18°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 18°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.

Hajat, S., Kovats, R. S., Atkinson, R. W., et al (2002) Impact of hot temperatures on death in London: a time series approach. *Journal of Epidemiology and Community Health*, **56**, 367–372.

Halberg, F., Cornélissen, G., Panksepp, J., et al (2005) Chronomics of autism and suicide. *Biomedicine and Pharmacotherapy*, **59** (suppl. 1), S100–S108.

Kovats, R. S., Johnson, H. & Griffith, C. (2006) Mortality in southern England during 2003 heat wave by place of death. *Health Statistics Quarterly*, **29**, 6–8.

Page, L. A., Hajat, S. & Kovats, R. S. (2007) Relationship between daily suicide counts and temperature in England and Wales. *British Journal of Psychiatry*, **191**, 106–112.

Richardson, J. D., Paularena, K. I., Belcher, J., et al (1994) Solar wind oscillation with 1.3 pear period. *Geographical Research Letters*, **21**, 1559–1560.

Salib, E. & Gray, N. (1997) Weather conditions and fatal self-harm in North Cheshire 1989–1993. *British Journal of Psychiatry*, **171**, 473–477.

Yip, P. S. F., Chao, A. & Chiu, C. W. F. (2000) Seasonal variation in suicides: diminished or vanished. Experience from England and Wales, 1982–1996. *British Journal of Psychiatry*, **177**, 366–369.

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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 thermoregulation. 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).

Bouchama, A. & Knochel, J. (2002) Heat stroke. *New England Journal of Medicine*, **346**, 1978–1988.

Hajat, S., Kovats, R. S. & Lachowycz, K. (2007) Heat-related and cold-related deaths in England and Wales: who is at risk? *Occupational and Environmental Medicine*, **64**, 93–100.

Kovats, R. S. & Ebi, K. L. (2006) Heatwaves and public health in Europe. *European Journal of Public Health*, **16**, 592–599.

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Avoiding errors about 'margins of error'

When discussing actuarial risk assessment instruments (ARAIs), 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 ARAIs 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 group 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 ARAIs raises serious moral problems as well as the valid scientific questions that Hart *et al* mention. But in faulting the capacity of ARAIs 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.

Berger, J. O. (1985) *Statistical Decision Theory and Bayesian Analysis* (2nd edn). Springer-Verlag.

Hart, S. D., Michie, C. & Cooke, D. J. (2007) Precision of actuarial risk assessment instruments. Evaluating the 'margins of error' of group *v.* individual predictions of violence. *British Journal of Psychiatry*, **190** (suppl. 49), s60–s65.

Wilson, E. B. (1927) Probable inference, the law of succession, and statistical inference. *Journal of the American Statistical Association*, **22**, 209–212.

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Authors' reply: Actuarial risk assessment instruments (ARAIs), 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 ARAIs carve nature at its joints, separating people with perfect accuracy into non-overlapping categories. No one, not even those who construct ARAIs, makes this assumption. So, we ask again, what is the precision of individual risk estimates made using ARAIs?

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 ARAIs, 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