Sunshine and Suicide Incidence

TABLE 1. The Association Between Major Football Matches Played by the Dutch National Team and Deaths Attributable to Acute Myocardial Infarction and Stroke

Opponent	Event and Date	Comment	Result*	Lag 0 OR	Lag 0 95% CI	Lag 1 OR	Lag 1 95% CI
Germany	EC, 21/06/88	Semifinal; decided in 88th minute of regular time	2-1	1.03	0.82–1.29	1.19	0.96–1.48
Soviet Union	EC, 25/06/88	Final; decided early in regular time	2-0	0.98	0.77–1.24	0.90	0.70–1.15
Germany	WC, 24/06/90	2nd round loss; decided in regular time	1-2	0.82	0.63–1.07	0.92	0.72–1.17
Denmark	EC, 22/06/92	Semifinal; decided by penalty kicks after extra time	4-5‡	1.16	0.93–1.44	0.99	0.80–1.26
Brazil	WC, 09/07/94	Quarter final; decided late in regular time	2-3	0.98	0.77–1.25	1.25	1.01–1.55
All matches combined				1.00	0.90–1.11	1.05	0.95–1.16

EC = European Championship; WC = World Championship.
* Score: Dutch team *vs* opponent.
† Lag 0 refers to mortality on the day of the match; lag 1 to mortality on day after the match.
‡ Score was tied 2-2 at end of regulation time.

and low temperature days), ambient humidity, black smoke air pollution, and indicators for day of week and holidays.[2–4]

Because the football matches were played in the evening or late afternoon, we analyzed mortality of the same day as the match (lag 0) and mortality on the day after the match (lag 1) separately.

The results (Table 1) showed little or no association between these football matches and cardiovascular deaths. An analysis using the same procedures as Witte *et al.*[1] produced similar results with lag 0 relative risks of 1.08, 0.93, 0.78, 1.23, and 0.90 for the five games, respectively.

We found no evidence of increased total or cardiovascular mortality associated with five major football games played between 1988 and 1994 by the Dutch national team. Although, in this dataset, we were unable to analyze data separately for men and women, the power of our study was sufficient to detect a 10% increase in mortality attributable to acute ischemic attacks and stroke. The Witte *et al.*[1] study found a 50% increase in men, and a nonsubstantial 11% increase in women. That our analysis using the same methodology failed to show increased mortality after five major matches suggests that the original finding may have been a chance finding, or that the 1996 game against France featured peculiarities that were not shared by the games we analyzed. It should be noted that in our data, the highest odds ratio for lag 0 (1.16) was associated with a match lost to penalty kicks, as was the 1996 game against France; further inquiries should perhaps focus on such matches. However, in general, major football matches do not seem to lead to increased total or cardiovascular deaths in the population.

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Sunshine and Suicide Incidence

To the Editor:

In an ecologic study across 20 countries, Petridou *et al.*[1] found a positive relation between relative risk of suicide during the peak month of suicide incidence and same-month average sunshine duration (+0.7; Spearman correlation). They concluded that sunshine exposure, via sunshine-regulated hormones like melatonin, may have a role in the triggering of suicide. Although there is no harm in this sort of speculation, acceptance of this effect should clearly await more direct evidence. However, we suspect that the finding itself rests on misleading methods and data, and we marshal evidence for this contention as follows.

First, the authors did not show that relative risk measures for suicide peak months are more closely related to seasonal variation in sunshine than to other environmental variables, did not mention findings opposite to their own (reviewed elsewhere),[2,3] and did not address the fact that suicide peak months generally are not the ones with the most intense sun exposure.

Second, mere size of correlational findings is not evidence for actual relations. Using the relative risk estimates from Petridou *et al.*[1] (Table 1), we obtained sizable cross-national correlations with other variables[4] as well, with physician density (+0.62), tuberculosis rate (+0.81), and computer ownership density (−0.57). Does this mean there is a role for doctors or tuberculosis cases in increasing countries' amplitude of suicide seasonality, whereas computer ownership reduces the amplitude?

Third, the authors make no mention of the perhaps most startling finding in suicide seasonality research—over the past few decades, suicide seasonality has notably diminished almost everywhere. The main agent of this secular trend remains unresolved.[3,5,6] If indeed seasonal variation in sunshine triggers within-country suicide peaks, and differences in sunshine account for cross-national differences in suicide seasonality, we look forward to hearing that seasons within countries, as well as climate differences across countries, have recently decreased.

Fourth, we doubt the accuracy of countries' suicide peak months as determined by Petridou *et al.*[1] Other peak months have been identified for Australia,[7] Finland,[8] Ireland,[9] Japan,[10] New Zealand,[7] Sweden,[11] and Austria (1970–1999 data: May, not June). Some of these findings stem from time series considerably longer (Sweden: 1911–1993)[11] than that of Petridou *et al.*; others indicate either gender differences in peak months[7,8] (including Austrian data) or biseasonality in suicide incidence.[7,8,10] The Petridou *et al.*[1] time-series data vary greatly in length (4 to 24 years), which obviously led to misidentification of suicide peak months, because there is evidence for them shifting from spring to summer with increasing latitude[8] (*ie*, a positive relation). Conversely, in the Petridou *et al.*[1] data, this relation is negative (−0.28; correlation between peak month number, recoded for southern hemisphere, and capitals' latitude).

Fifth, we question the accuracy of the Petridou *et al.*[1] relative risk estimates for countries' peak suicide months. Monthly variation in suicide is still strong in the United States,[12] although, in the Petriodou *et al.* table, the smallest estimate is for the United States. The relative risk estimate is exceptionally large in Japan,[10] although not presented as such in the table; rather, in the table, the relative risk estimate for Japan is identical to that for Australia, where seasonality is weak.[7] Again, high cross-country variation in time-series' length, in concert with the statistical method used, obviously led to erroneous estimation of seasonality effects. The circular normal distribution method used by Petridou *et al.*[1] tests for one-cycle seasonality only, thus missing seasonality increments attributable to within-year cycles, and it is sensitive to outliers in the data that gain influence in short time series.[3] The accuracy of suicide seasonality estimates can be tested using their positive relation to latitude, as has been found both within the United States[13] and internationally[3,5] (*ie*, seasonality increases with increasing equatorial distance). Conversely, in the Petridou *et al.*[1] data, the correlation is negative (−0.35).

It is more parsimonious to assume misidentification of peak months and mistaken estimation of seasonality effects in the Petridou *et al.*[1] study, attributable to factors unique to their database and the statistical method used, than to suggest that a great many established findings on suicide seasonality are incorrect. Their finding of a relation between sunshine duration and suicide incidence rests heavily on correctly identified suicide peak months and correctly estimated suicide seasonality effects. Because the data are demonstrably odd, the authors' conjecture might be unwarranted.

On a final note, we express our irritation regarding the claim by Petridou and colleagues[1] for scientific priority regarding the cross-national documentation of seasonal suicide peaks. Actually, the Chew and McCleary[5] study deserves such priority—it was not merely about "several" countries, but was, rather, a large-scale (28-country) investigation covering 16 of the 20 countries sampled by Petridou *et al.*[1]

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The Authors Respond:

We are responding to the letter by Voracek and Fisher[1] concerning our paper[2] for the benefit of the readers. Neither the tone nor the arguments of the letter would deserve an answer otherwise. We take the points one by one.

1. Dr. Hakko's thesis, which is relevant to our paper, was not readily accessible, and the insinuation that we have intentionally ignored it is just a reflection of the general tone of the letter.
2. Cross-country correlations are not relevant to consistent seasonal patterns, and the argument that correlation does not necessarily imply causation is a truism for which no senior, or indeed a beginner, epidemiologist would need advice.
3. Seasons within countries have not changed, but introduction of additional causes of suicide without seasonality can obscure a seasonal pattern. Moreover, exposure to

environmental seasonal variables, such as through urbanization, has indeed changed.
4. We have indicated the origin of our data and we are aware that all empirical data have limitations. However, sample size is not a problem in our study and we have not argued that there may not be superimposing patterns on the underlying seasonality.
5. The authors of the letter do not seem to realize that: (i) suicide, as well as most manifestation-defined health outcomes, has multiple causes, which in turn have their own descriptive epidemiologic characteristics; and (ii) because it is based on a likelihood function, the method of analysis used is statistically more, rather than less, principled for estimating seasonality compared with the usual alternative of Edward's test.

Last, we did not claim priority for a phenomenon that has been well known for over 100 years. In contrast, as we have also pointed out in the paper, the advantage of our study is in having looked simultaneously at the given data from various angles, all of which have provided results consistent with an association of suicide and sun exposure. We believe that it is accumulation and consistency of evidence, and not expression of personal feelings, that advance scientific knowledge. The "irritation" of Voracek and Fisher is not relevant to a scientific discourse.

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Sexual Activity and the Risk of Prostate Cancer

To the Editor:

Dennis and Dawson[1] reported on a meta-analysis of the relation between measures of sexual activity and prostate cancer. They found elevated risks among men with (1) a history of sexually transmitted infections, (2) high coital rates, and (3) high number of sexual partners. The authors write that "the mechanism through which frequency of sexual activity may be related to prostate cancer is unclear" although they acknowledge that "the increased relative risks seen with increased sexual frequency, particularly before 60 years of age, suggest a possible link with sexual hormone levels." I suggest that this is correct. The same androgen, dihydrotestosterone (DHT), has been implicated in men's coital rates[2] and in men's risk of prostatic cancer.[3,4]

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