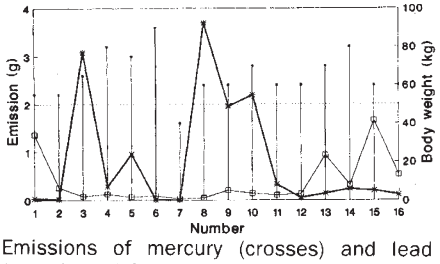
material on a quartz filter. Vapour-phase mercury was absorbed in a solution of 1% $KMnO_4$ in 10% sulphuric acid, and lead was absorbed in 5% HNO_3 in a solution of 3% peroxide. The quantity of metal in each sample was determined by cold-vapour and graphite-tube atomic absorption spectroscopy for mercury and lead, respectively. In addition, the temperature of each incineration and information about age, sex, weight, nationality, medication, where the person died and the type of coffin were provided.

We show the emissions of mercury and lead for each cremation in the figure. (Errors were estimated to be 25 and 30% for mercury and lead, respectively.) Clearly, emissions of both these elements vary considerably between cremations.



Emissions of mercury (crosses) and lead (open boxes) from a crematorium incinerator. Smaller, solid boxes, body weight.

The maximal emissions of mercury are in good agreement with the estimations of Mills, and the emissions of lead are somewhat smaller than those of mercury.

We found no correlation between emissions of these elements, between body weight and emissions, or between emissions and temperature of the cremation, incinerator-model, type of coffin, medical history, age or sex. We conclude that the mercury emissions are probably due to amalgam fillings, but we have no explanation for the origin of the lead emission peaks.

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SIR—Two points may be added to Arnold P. Wendroff's letter (*Nature* **347**, 623; 1990). First, only about 10% of inhaled mercury enters the circulation through the lung alveoli, the remainder is absorbed by the bronchial secretions which are usually ingested. The result is that it tends to be mixed with food before digestion. Because it is toxic by virtue of its combination with protein, a diet rich in protein is likely to be protective. It is therefore difficult to predict the danger from heavy metal ingestion unless the amount of protein present in the diet is taken into account.

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No pulsar in SN1987A

SIR—Our previously reported observation (*Nature* **338**, 234–236; 1989) of what appeared to be an optical pulsar in the remnant from supervova 1987A turns out to be spurious. Despite repeated searches by us and others following the original observation in January 1989, the putative pulsar was not confirmed. In new data obtained in February 1990, however, we again detected the signal, in data from the supernova and, in comparison data taken on the Crab pulsar at the same time. From this, it is clear that the signal did not originate in the supernova, and we retract our reported discovery of the pulsar.

We believe that the observed signal is electrical noise from a closed-circuit television camera used for acquisition and guiding at the Cassegrain focus of the 4-metre telescope at Cerro Tololo Interamerican Observatory, where both sets of data were taken. The observed frequency of 1968.627 Hz is close to the 16th subharmonic of the nominal horizontal sync pulse of the camera (1968.75 Hz); the coincidence seems too close to be accidental.

We do not know how the signal coupled into our detector, and have not been able fully to reconstruct events during the observations. But we think there are plausible explanations for some of these things that made the original observation appear real. The most important are: (1) The signal disappeared when the telescope was pointed at a different object immediately after observing the supernova. It is likely that the offending guider camera was turned off between the two sets of observations to prevent damage from the increasing light of twilight.

(2) The signal was not observed with the same equipment on the same telescope, either before or after the detection run, until the run in February 1990. Several plausible explanations for this come to mind, any one or more of which might have occurred: there may have been subtle differences in the mounting of the detector system, changes in the adjustment of components, or a different guider camera (there are two such cameras at the telescope, which are used interchangeably). The system is extremely sensitive, and the detected signal was very small by normal standards: the interference current was equivalent to about 10^{-15} amps.

(3) Several highly suggestive properties of the observed frequency were totally fortuitous. The stability of the frequency over the 7-hour run was high (10^{-6}), a factor of 100 better than the stability specification for the camera. Furthermore, the observed small frequency change was quite smooth, and when the data were corrected for the Earth's motion, they were well fit by a sine wave, which suggested an external origin. (When a sine wave was subtracted from the data, the frequency residuals decreased by an order of magnitude. This was the basis for speculation that, for example, the pulsar might be in a binary orbit.) Similarly, the fact that the first two higher harmonics were in the same proportions as those seen in the Crab pulsar was a coincidence. All this was undoubtedly a combination of bad luck (or divine malice), misplaced pattern-finding skills, and the common human tendency to overinterpret a limited amount of data.

Searches for the pulsar continue, so far unsuccessfully.

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Scientific Correspondence

Scientific Correspondence is intended to provide a forum in which readers may raise points of a scientific character. They need not arise out of anything published in *Nature*. In any case, priority will be given to letters of fewer than 500 words and five references. □

Signalling and superinduction

SIR—The process by which protein synthesis inhibitors accentuate and prolong the induction of specific genes that are normally only transiently induced in response to polypeptide growth factors, cytokines, hormones, interferons and phorbol esters is termed superinduction. This process is thought to be attributable to the loss of labile transcriptional repressors and messenger RNA-degrading enzymes, and is usually regarded as a direct consequence of the inhibition of protein synthesis[1–6]. Subramaniam *et al.*[6], for example, used this phenomenon to conclude that the serum response element is subject to negative regulation by labile repressors. Here we present evidence that supports an alternative explanation: some protein synthesis inhibitors may act positively to activate transcription through an intrinsic ability to interact with molecules involved in intracellular signalling.

When quiescent cells are stimulated with epidermal growth factor (EGF) or the tumour promoter tetradecanoyl phorbol acetate (TPA), the proto-oncogenes c-*fos* and c-*jun* are rapidly and transiently