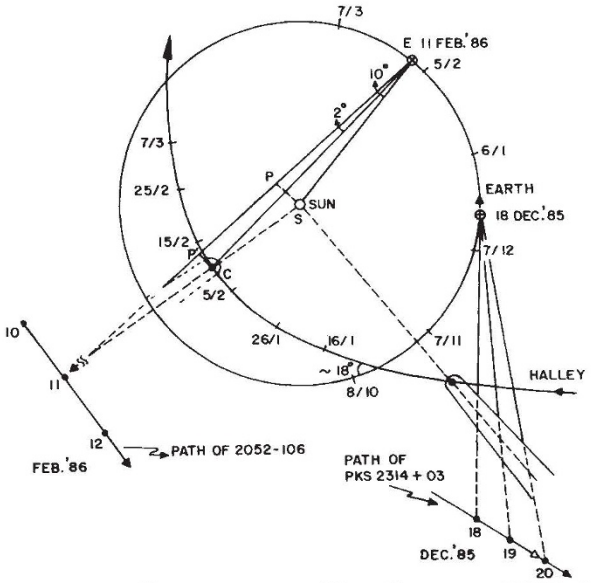

Relative positions of the Sun, the Earth and Halley's comet during the observations reported in refs 1 (December 1985) and 3 (February 1986). The paths of the quasar sources are also shown.

compact radio sources resulting from occultation by strong cometary ion-tails can be identified only for favourable alignments of the comet, Sun and observer. Such conditions existed during the observations reported in ref. 1 and it is possible, therefore, that the scintillations were of cometary origin. Unambiguous confirmation could be obtained by using more control sources close to the line of sight[4].

S. K. ALURKAR
A. K. SHARMA
P. JANARDHAN
R. V. BHONSLE

Physical Research Laboratory, Ahmedabad-380009, India

ANANTHAKRISHNAN *ET AL.* REPLY — The importance of our letter[3] lies in its demonstration that control sources are essential in investigations of scintillation through the cometary plasma tail. Alurkar *et al.* also agree that control observations are essential. Without such observations it is difficult to determine whether or not the observed[1] enhancement in scintillation is caused by the plasma tail.

We disagree with the suggestion that because our observations of 2052−106 on 11 February 1986 were made at a solar elongation of about 10° (actually 11.2°), the circumstances were unsuitable for studying the enhancement of turbulence. On the contrary, the outflow of plasma from the comet would be an order of magnitude higher for our observations than for those of Alurkar *et al.*[1], made at a solar elongation of 85° and before perihelion. Furthermore, angular broadening effects are unimportant unless the source is very much closer to the Sun. Therefore, the 60% enhancement seen by us (Fig. 2 of ref. 1) could have arisen either from the comet or from the solar wind. However, a similar increase in the control source implies that a solar-wind origin is most probable. In addition, observations far from the Sun, of the sources 1817−391 and 1921−293 along with control sources, also show no enhancement resulting from the plasma tail[3].

We agree that geometry of occultation is important. Unfortunately, cometary occultations of the required geometry are extremely rare, and consequently there is a need to take every available future opportunity for making occultation observations of radio sources by cometary tails (along with suitable control sources) in order to resolve the present conflict of results.

S. ANANTHAKRISHNAN
P. K. MANOHARAN
V. R. VENUGOPAL

Radio Astronomy Centre, Tata Institute of Fundamental Research, PO Box 8, Udhagamandalam 643 001, India

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Zinc and LTP

SIR—Recent studies have implicated *N*-methyl-D-aspartate (NMDA) receptor activation and concurrent postsynaptic depolarization in the induction of long-term potentiation (LTP)[1]. Kauer *et al.*[2] recently showed that NMDA receptor activation produced only a transient potentiation in the CA1 region of the hippocampus and speculated that some additional factor, perhaps co-released with glutamate from presynaptic terminals, was essential for the later, long-lasting phase of LTP. We suggest that synaptic Zn^{2+} is the factor. Zn^{2+} is systematically localized in synaptic vesicles of excitatory synapses throughout the mammalian telencephalon[3] and probably is co-released with glutamate into the synaptic cleft with neuronal activity[4,5], especially at higher rates of stimulation[6]. Zn^{2+} decreases NMDA receptor-mediated responses but increases quisqualate receptor-mediated responses[7,8], and may acutely modulate the postsynaptic response to glutamate. If this synaptically released Zn^{2+} can gain entry into postsynaptic neurons, it is possible also that it could induce LTP. It has been shown *in vitro*, that Zn^{2+} can activate protein kinase C and induce its translocation to membranes[9], both associated with establishing LTP[1,2]. Entry of Zn^{2+} into neurons has not been clearly documented but it could pass through the NMDA channels. Zn^{2+} can block the NMDA channel (C. W. Christine and D. W. Choi, manuscript in preparation). Mg^{2+}, and perhaps divalent cations in general, may permeate the NMDA channel at a rate determined by the ease with which surrounding water molecules can be lost[10,11]. Zinc can shed its water molecules at a rate between that of Ca^{2+} and magnesium[12], suggesting that its permeability may be greater than that of Mg^{2+}. The neurotoxicity of Zn^{2+} on cortical neurons is consistent with Zn^{2+} entry through the NMDA channel, as it is competitively blocked by the NMDA-channel blocker, MK-801, and increased by removal of extracellular Na^+ and calcium[13].

Thus the simple co-occurrence of NMDA receptor activation with postsynaptic depolarization could result in a calcium influx and transient postsynaptic potentiation. If the presynaptic neuron is firing sufficiently rapidly, Zn^{2+} could be released from presynaptic terminals and enter the postsynaptic neuron via NMDA channels, activating protein kinase C (and perhaps other intracellular processes) resulting in LTP.

JOHN H. WEISS
JAE-YOUNG KOH
CHADWICK W. CHRISTINE
DENNIS W. CHOI

Department of Neurology, Stanford University Medical School, Stanford, California 94305, USA

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