as a function of their screening score to estimate the number of unascertained cases. Using this procedure, we project an additional 27 cases (3, 7 and 17, respectively), raising the overall rate to 19.7 per cent (60/305). Future references to Pitt's rate of late puerperal depression should use this adjusted figure. Hopefully, this correction will add further impetus to a research area pioneered by Pitt.

RICHARD NEUGEBAUER

G. H. Sergievsky Center,
Faculty of Medicine, Columbia University,
630 W. 168th Street,
New York, NY 10032, U.S.A.

References

PITT, B. (1968) "'Atypical' depression following childbirth". *British Journal of Psychiatry*, **114**, 1325–35.
—— (1980) Personal communication, 25 June.

CANCER AND DEPRESSION

DEAR SIR,

Brown and Paraskevas proposed (*Journal*, September 1982, **141**, 227–32) an intriguing theory that some cases of depression in cancer may be caused by immunological interference with the activity of serotonin. Tumor basic protein (TBP) appears ubiquitous among cancer cells (Caspary and Field, 1971). There appears to be no question that it contains a site which structurally is similar to the 9-residue peptide of myelin basic protein, which is responsible for experimental allergic encephalomyelitis (EAE) and which also binds serotonin. They also have shown that TBP binds serotonin.

During clinical EAE, very little circulating antibody is produced against the EAE active peptide. Therefore one must presume that the corresponding site in TBP also is a poor antibody producer. However, what antibody that is produced should *not* bind to serotonin since it and serotonin are complimentary to TBP and the EAE active peptide, which both bind serotonin. Therefore the *in vivo* interference of serotonin actions by TBP would be dependent upon large amounts of exposed TBP in areas that are sensitive to serotonin concentrations. The interference would not be due to antibody concentrations.

Another area which should be examined is the relationship of euphoria and demyelinating diseases. Contrary to what is seen in cancer, the active EAE peptide that binds serotonin is released (Cohen *et al*, 1975) in large amounts only for very short periods, e.g., during relapses in multiple sclerosis.

FRED C. WESTALL

The Salk Institute for Biological Studies,
P.O. Box 85800,
San Diego, California 92138

References

CASPARY, E. A. & FIELD, E. J. (1971) Specific lymphocyte sensitization in cancer: is there a common antigen in human malignant neoplasia? *British Medical Journal, ii*, 613–16.
COHEN, S. R., HERNON, R. M. & MCKHANN, G. M. (1976) Myelin basic protein in CSF as an indicator of active demyelination. *New England Journal of Medicine*, **295**, 1455–7.

ANTI-CHOLINERGIC DRUGS AND MEMORY

DEAR SIR,

The effects of anticholinergic drugs on memory have rarely been studied, yet knowing their nature is important. In 1982, Potamianos and Kellett reported their finding that anti-cholinergic drugs had an adverse effect on memory. In their study, geriatric patients performed significantly worse when on benzhexol than when on placebo. However, it should be noted that this applied only to three of their tasks: 'paired associated learning', 'short-story recall' and 'word list recall'. It did not apply to 'digit span'. This short term memory test was the only task they used, which did not require that the subject established mnemonic organization at encoding.

This is of particular interest in the light of recent results we obtained while studying memory in schizophrenia (Calev, 1981; Calev, Venables and Monk, 1983). We used two *long-term* memory tasks, which like many short-term memory tasks, minimized the need for the subject to use mnemonic elaboration at encoding. In the first task, the patients were instructed, before the recall test, to meaningfully sort and semantically organize the to-be-remembered words; so that the subject's spontaneous use of mnemonic organization at the encoding stage became redundant. In the second task, (recognition memory), patients were required to discriminate formerly presented target words from distractor words sampled from the same population; this task too was said to involve minimal need for mnemonic organization at encoding (e.g. Kintch, 1970; Koh, 1978). In both these tasks, we found no differences between two groups of chronic schizophrenics, of which only one was on anticholinergic medication (Disipal, procyclidine, and benzhexol). We have recently replicated these results.

Taken together, the results of these two studies seem to indicate that anti-cholinergic drugs affect memory tasks which require mnemonic organization at the encoding stage, but not all memory tasks. When mnemonic organization is either not essential (e.g. in 'digit span' and 'recognition') or artificially induced at encoding (e.g. our first task), no deficit is apparent. A literature search indicated that this conclusion also fits