We are grateful to J. de Villiers and J. N. Theron for their criticism of the manuscript.

M. R. COOPER

*South African Museum,*

R. OOSTHUIZEN

*Zwartkraal,*
*Prince Albert*

Received September 11, 1973.

1 McLachlan, I. R., and Anderson, A., *Palaeont. afr.*, **15**, 37 (1973).
2 McLachlan, I. R., and Anderson, A., *IUGS, Gondwana Symp., Abstr.*, **3**, 42 (1973).
3 Martin, H., *Trans. geol. Soc. S. Afr.*, **56**, 37 (1953).
4 Martin, H., and Wilczewski, N., *IUGS, Gondwana Symp.*, **2** 225, (1970).
5 Martin, H., Walliser, O. H., and Wilczewski, N., *IUGS, Gondwana Symp.*, **2**, 621 (1970).
6 Martin, H., *Trans. geol. Soc. S. Afr. Annex.*, **64**, 1 (1961).
7 Germs, G. J. B., *Bull. P.R.U., Univ. Cape Town*, **12**, 1 (1972).
8 Martin, H., *The Precambrian Geology of South West Africa and Namaqualand* (University of Cape Town, 1965).
9 Theron, J. N., *The Stratigraphy and Sedimentation of the Bokkeveld Group* (Thesis, Stellenbosch, 1972).
10 Brunton, C. H. C., Cocks, L. R. M., Rowell, A. J., and Rust, I. C. *Q. Jl. geol. Soc. Lond.*, **125**, 583 (1970).
11 Visser, H. N., *Ann. Geol. Surv. S. Afr.*, **6**, 31 (1967).
12 Stratten, T., *Palaeont. afr.*, **11**, 127 (1968).
13 Theron, J. N., and Blignault, H. J., *IUGS, Gondwana Symp.*, **3** (in the press).
14 Ahmad, F., *IUGS, Gondwana Symp.*, **2**, 179 (1970).
15 Lindsay, J. F., *IUGS, Gondwana Symp.*, **2**, 477 (1970).
16 Hill, D., *Trans. R. Soc. N.Z., Geol.*, **2**, 137 (1964).
17 Palmer, A. R., and Gatehouse, C. G., *Prof. Pap. U.S. geol. Surv.*, **456** D, 1 (1972).

## Discrimination learning in ascorbic acid-deficient guinea pigs

PAULING[1] has suggested that a high intake of vitamin C may be necessary for optimum cerebral function including learning and memory. We have tested this hypothesis in a preliminary way by comparing maze learning in two groups of guinea pigs: those receiving large supplements of ascorbic acid (the 'control' group) and those receiving a daily quantity of the vitamin sufficient to maintain a concentration in the brain at approximately 25% that of the controls (the 'deficient' group).

Thirteen male guinea pigs were reared on a diet well supplemented with ascorbic acid. When the animals were 9 weeks old (day 0) they were randomly assigned to one of two groups ('control' or 'deficient') and given an ascorbic acid-free diet[2]. Drinking water was changed daily. That of the 'controls' contained 1% ascorbic acid throughout the experiment. From day 16, both groups received a daily oral dose of ascorbic acid (6 mg $kg^{-1}$ body weight) just sufficient to maintain good growth in deficient animals[3,4]. Water consumption measurements indicated that controls received at least 500 mg $kg^{-1}$ body weight of ascorbic acid daily. This intake is much in excess of that required to saturate guinea pig tissues with vitamin C[3].

From day 46 to day 67 animals were tested for spatial discrimination in a water T-maze (J. L. Smart, in preparation). On the first two days (days 46 and 47) escape was possible from both arms and animals were given five trials a day to test for left or right preferences. There followed 20 successive days of six trials per day during which escape was possible from only one arm of the maze. Animals were required to reverse previous preferred or learned responses. A learning criterion (successful 'reversal') was said to have been met when no errors (entries into arms other than the escape arm) were recorded during the last five of the six trials. The day after meeting this criterion the escape route was reversed.

TABLE 1 Body weight, tissue ascorbic acid and maze performance in control (saturated) and chronically ascorbic acid-deficient guinea pigs

| | | Control ($n$ = 6) | Deficient ($n$ = 7) |
|---|---|---|---|
| Body weight (g) | day 0 | 413 ± 61 | 420 ± 63† |
| | day 68 | 651 ± 40 | 626 ± 62† |
| Tissue ascorbic acid on day 68 ($\mu$mol $g^{-1}$ wet wt) | Forebrain | 1.11 ± 0.05 | 0.30 ± 0.05* |
| | Brainstem | 0.70 ± 0.05 | 0.20 ± 0.04* |
| | Liver | 1.67 ± 0.20 | 0.28 ± 0.17* |
| | Adrenals | 6.49 ± 1.28 | 0.91 ± 0.15* |
| T-Maze performance (days 48 to 67) | Total reversals | 7.0 ± 1.1 | 6.1 ± 2.9† |
| | Total errors | 50.0 ± 11.0 | 48.7 ± 13.4† |

Results are mean ± s.d.
* $P < 0.001$.
† Not significant (Student's $t$ test) when compared with corresponding control value.

On day 68 animals were killed by decapitation and ascorbic acid was determined[5] in brain, liver and adrenal glands.

In a parallel experiment mean forebrain ascorbic acid in deficient animals was, on day 15, 0.44 ± 0.05 $\mu$mol $g^{-1}$ ($n$ = 4), and, on day 42, 0.26 ± 0.25 $\mu$mol $g^{-1}$ ($n$ = 4). These values represent deficits, compared with controls (Table 1), of 60% and 77%, respectively. Brainstem showed deficits of 69% at 15 d and 63% at 42 d. On day 68 (Table 1) the deficits were 73% in forebrain, 71% in brainstem, 83% in liver and 86% in adrenals. It can be inferred that the brains of the deficient group were severely lacking in ascorbic acid both during maze learning and for at least 30 d before.

The chronic, severe brain vitamin C deficiency had no effect on T-maze performance, as assessed by either reversals or errors during 20 d of testing (Table 1). This indicates that learning is not enhanced by having saturating levels of ascorbic acid in the brain.

This work was supported by grants to Dr John Dobbing from the Medical Research Council and the National Fund for Research into Crippling Diseases. We thank Dr R. E. Hughes for advice.

B. P. F. ADLARD
SUSAN MOON
J. L. SMART

*Department of Child Health,*
*University of Manchester,*
*Clinical Sciences Building,*
*York Place,*
*Manchester M13 OJJ.*

Received November 29, 1973.

1 Pauling, L., *Science N.Y.*, **160**, 265 (1968).
2 Hughes, R. E., and Hurley, R. J., *Br. J. Nutr.*, **23**, 211 (1969).
3 Burch, H. B., *Ann. N.Y. Acad. Sci.*, **92**, 268 (1961).
4 Williams, R. S., and Hughes, R. E., *Br. J. Nutr.*, **28**, 167 (1972).
5 Roe, J. H., and Kuether, C. A., *J. biol. Chem.*, **147**, 399 (1943).

## Similarity of morphine abstinence signs to thermoregulatory behaviour

THE precipitated abstinence syndrome consists of a series of behavioural events which appear in morphine-dependent organisms after the administration of narcotic antagonists. Martin[1] has suggested that some precipitated abstinence signs arise because an error force is generated when a narcotic