the frequency of mutations could not be scored because of the high degree of sterility. The relation between frequency of mutations and the degree of sterility is, therefore, to be considered as favourable. With X-rays, for example, a semi-sterility of 70 per cent can only be associated with a mutant frequency of about 12 per cent. A 47 per cent frequency of mutants, as found with 0·5 mM NG, will be reached only by very few mutagens and is far above the maximal value obtained with X-rays. A mutant frequency of 47 per cent means that on an average 2·4 lethal mutations per meristem cell have been induced. The values found indicate that NG can be classified as a mutagen of a very high efficiency and can be put on the same level as EMS, although EMS must be applied in concentrations 50 times higher. Under the same treatment conditions, therefore, NG has a much higher effectiveness than EMS (compared on a molarity basis).

Table 1

Concentration (mM)	No. of M_1 plants	m_a	m_b	m_c	Degree of sterility
0	3,419	0·7	0·4	0·11	0
0·06	148	2·0	1·0	0·17	0
0·12	164	41·3	15·2	3·88	1
0·25	243	91·6	72·4	20·81	19
0·5	192	100·0	97·8	47·38	69
1	110	—	—	—	99

m_a, Frequency of segregating plant progenies (per cent); m_b, frequency of segregating pod progenies (per cent); m_c, frequency of mutants (per cent).

Somatic effects, semi-sterility and mutation frequency were found to be dependent on the temperature during treatment time in so far as the effectiveness of the agent drastically increased from 18° to 36° C. The effectiveness is influenced by the *p*H value, too. With alkaline solutions (*p*H = 8·9, boric acid buffer) no mutagenic effects have been demonstrated. This may be explained by the fact that under alkaline conditions NG decomposes into diazomethane. (Under acid conditions it decomposes into nitrous acid.) Any clear-cut evidence on the nature of the agent inducing mutations is still lacking. There are some arguments suggesting that the breakdown products mentioned here are not responsible for the radiomimetic activity of NG[1,4]. According to Gichner *et al.*[1] a similar mode of action for NG, *N*-methylphenylnitrosamine and 'Cupferron' might be suggested. But considering the fact that diazomethane induces mutations (compare with ref. 7) a participation of diazomethane in the mutagenesis by NG cannot be excluded.

Andreas J. Müller
Tomáš Gichner

Institut für Kulturpflanzenforschung,
Gatersleben, Kr. Aschersleben
(Germany, D.D.R.),
Institute of Experimental Botany,
Praha 6 (C.S.S.R.).

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PSYCHOLOGY

Reverberatory Activity and Memory Disorder

I have recently demonstrated that a possible explanation to account for the gross memory disorder in some elderly psychiatric patients is a lowered energy of reverberatory activity in neural cells; a reduction in cerebral excitation which mitigates against short-term storage[1]. This in turn is related to the pathology associated with senile dementia and cerebral arteriosclerosis.

A recent report by Hebb[2], dealing with memory for digits, would seem to provide the basis for extending and elucidating this notion.

Briefly, Hebb presented to 40 university students 24 nine-digit numbers which they were required to recall immediately. Each of the 24 items was different with the exception that every third one was the same. That is, the third, sixth, ninth item and so on was repeated. The subjects were not told of this repetition.

If, in these circumstances, immediate memory for digits is mediated solely by an activity trace, with no structural change, then there would be no cumulative learning. Each new stimulus would set up a completely new pattern of activity and the non-repeated digit items would be as accurately recalled as the repeated digit items. If, on the other hand, cumulative learning does occur, then this would provide evidence for structural modification. It would lend support to Hebb's[3] theory that learning involves, first, some form of reverberatory neural activity which is the basis of short-term memory, and, secondly, a structural alteration, resulting from this activity which constitutes longer-term learning.

The results of the investigation showed cumulative learning, with the implication that a single presentation of a set of digits results in a structural trace which can be cumulative. This investigation would appear to be relevant to the notion that memory disorder in old age is due to a decrease in the energy of this cerebral reverberatory activity.

The work reported here tests this notion and is one of a series designed to facilitate diagnosis and treatment of this disorder.

There were two groups of subjects in this investigation. Group *A* was composed of 20 memory-disordered psychiatric patients aged 72–87. Group *B* consisted of 20 patients aged 73–85, free of memory disorder. The two groups were matched in terms of age, verbal intelligence and digit span forward.

The procedure was a duplication of Hebb's[2] as outlined here. The only differences were a reduction in each series length from 9 to 6 digits and an increase in the number of items from 24 to 30.

Table 1

Procedure	Group *A*	Group *B*
Repeated digits	0·43	0·66
Non-repeated digits	0·35	0·43

Table 1 presents the proportion of correct responses for both groups for the repeated and non-repeated items. Analysis of the results shows that there is no significant difference between the two groups for the non-repeated items. There is, however, a significant difference ($P < 0·05$) for the repeated items. In addition, there is no difference between the two procedures for Group *A*, but there is a significant difference ($P < 0·05$) between the procedures for Group *B*.

These results suggest that while cumulative learning does occur in elderly, non-memory-disordered people, it does not occur in elderly memory-disordered people. The reason it does not occur, it is suggested, is because this type of learning is dependent on reverberatory activity, and it is this function which is operating inefficiently in elderly patients suffering from gross memory deficit.

This work was supported by a Canadian National Mental Health grant (project No. 609–5–145).

W. K. Caird

Department of Psychology,
University of British Columbia,
Vancouver, B.C., Canada.

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