Individual differences in memory enhancement by encoding enactment: relationships to adult age and biological factors

Lars Nyberg\textsuperscript{a,\ast}, Jonas Persson\textsuperscript{a}, Lars-Göran Nilsson\textsuperscript{b}

\textsuperscript{a}Department of Psychology, Umeå University, S-901 87 Umeå, Sweden
\textsuperscript{b}Department of Psychology, Stockholm University, S-106 91 Stockholm, Sweden

Received 14 February 2002; accepted 15 June 2002

Abstract

Numerous studies have demonstrated an age-related decline in episodic memory performance. However, both younger and older adults benefit from various kinds of encoding support, suggesting that memory functioning remains plastic in older age. The present review is concerned with encoding support in the form of enactment. Memory for simple commands is substantially higher if the commands are enacted during encoding than only read/heard. Such memory enhancement has been demonstrated for many age groups and patient groups, suggesting that it is a general effect. Analysis of the results from 1000 participants ranging in age between 35 and 80 years revealed that about 5% of the participants had low memory performance after enacted encoding and showed no enactment effect. The majority of these were older. Comparisons of participants that did or did not show an enactment effect for a select set of biological and neuropsychological factors provided tentative evidence that a failure to benefit from encoding enactment reflects a dysfunctional motor system. This is in agreement with findings from recent functional neuroimaging studies that associate the enactment effect with motor areas in the brain. Variation in the ability to benefit from encoding enactment is discussed in relation to an age-related decline in dopamine function.

Keywords: Individual differences; Brain; Encoding support; Enactment; Dopamine; Motor system

1. Introduction

Numerous studies have shown that episodic memory performance is decreased in old age [1]. Based on cross-sectional analyses, it has been argued that the decline starts already in the 20’s [2]. Longitudinal analyses suggest that the onset of decline is considerably later [3,4]. However, cross-sectional and longitudinal analyses converge in showing a continuous negative association between age and episodic memory from about age 65 [3].

Given the strong negative relationship between age and episodic memory performance it is interesting to note that the performance of younger as well as older adults can be enhanced in various ways. This has been referred to as plasticity of memory functioning [5]. One means by which episodic memory performance can be enhanced is memory training [6]. Another way to enhance performance is to provide support or guidance when information is acquired to memory. There is evidence that older adults are more impaired relative to younger adults if memory is tested after a non-guided (intentional) encoding phase compared to if it is tested after guided (deep incidental) encoding [7]. These and related findings indicate that provided a supportive encoding situation, the memory performance of older adults can be quite high. The present review is concerned with a special form of encoding support, encoding enactment.

2. Encoding enactment

Many studies have shown that memory of simple commands (e.g. roll the ball) is substantially higher if participants during the acquisition phase enact the action described by each command than if they only read or hear the commands (for a review see Refs. [8,9]). The degree of memory enhancement following encoding enactment is substantial, often in the range of 20–30\% [10]. A memory-enhancing effect of encoding enactment has been demonstrated for several different populations, including demented...

Findings from our own laboratory serve to illustrate the strong effect of encoding enactment on memory performance across the adult life span. Fig. 1 is based on data from 1000 adults ranging in age between 35 and 80 years [17]. The data come from a longitudinal study on aging, memory and dementia [18]. The participants were presented with two lists of 16 sentences at a rate of 8 s per sentence. The sentences were read by the experimenter and presented visually on cards. The presentation of each list was followed by a test of immediate free recall (2 min were allowed for the test). The only difference between the two lists was the specific encoding instruction. For one of the lists, the participants were instructed to enact according to the sentences and to try to remember as many sentences as possible. If the action described by a sentence involved an external object (e.g. read the book), the object was handed over by the experimenter. For the other list, the participants simply tried to memorize as many sentences as possible without enacting them.

As can be seen in Fig. 1, across the age span, there was a positive effect of encoding enactment on recall performance. It can be noted that the recall performance of persons in their 70’s following encoding enactment was at about the same level as the memory performance of people in their 30’s after non-enacted (verbal) encoding.

3. Neural basis for the enactment effect

EEG- [19] and PET-findings [20,21] have associated the enactment effect with motor brain areas. Heil et al. [19] recorded event-related potentials (ERPs) during recognition following enacted and verbal encoding. They found that recognition was associated with a larger frontocentral negativity after enacted than verbal encoding. This observation was taken as an evidence that the superior memory performance following enacted encoding reflected reactivation of motor information.

In two PET studies [20,21] we have also found evidence that retrieval following the encoding enactment is associated with motor brain regions. This is shown in Fig. 2. Fig. 2 shows brain regions where activity was higher during cued recall after enacted encoding compared to cued recall after verbal encoding. The activated regions included regions in contralateral somatosensory and motor cortex. Importantly, these regions were also more active during enacted encoding compared to verbal encoding, suggesting that some of the motor areas that are engaged during enactment are subsequently reactivated during retrieval.

Taken together, the neuroimaging results suggest that the enactment effect is related to motor areas in the brain. In Section 4 this notion will be explored further.

4. Individual differences in the enactment effect

As noted earlier, a positive effect of encoding enactment on episodic memory performance has been demonstrated for many different age groups and patient groups. This pattern of results indicates that the effect is of a general nature. At the same time, to the best of our knowledge, there have been no attempts at assessing the robustness of the effect at the individual subject level. Such a lack of individual difference analysis seems to hold for many forms of encoding support, which may be due to the fact that large...
samples are required for identification of reasonably sized sub-groups that do not benefit from support.

To address the issue of how robust the enactment effect is at the individual subject level, we examined the distribution of scores from the 1000 adults that were included in the earlier described study [17]. Specifically, to identify participants who did not benefit from encoding enactment, the free recall performances following enacted and non-enacted encoding were contrasted and a difference score was computed. Of the 1000 participants, the difference score was zero or negative for 119 participants. Of the 119, 54 participants recalled four or fewer items in the enacted condition, 13 had very high performance (>10) in both the non-enacted and enacted condition, and 52 participants performed in the ‘mid-range’ (e.g. recalled seven words in both conditions). While the latter two sub-groups may have included persons who were unable to benefit from encoding enactment, their fairly high performance levels in both conditions indicate that they did not have memory problems. Instead, their absence of an enactment effect could be due to spurious factors (e.g. they may have found the particular items in the non-enacted list easy to remember, or used some kind of mnemonic to boost their performance in the non-enacted condition). Therefore, we will use a conservative criterion and focus on the 54 participants who recalled four or fewer items in the enacted condition and showed no enactment effect ($M_{enacted} = 2.44$, range $= 0–4$; $M_{non-enacted} = 3.52$, range $= 0–9$). These participants will be referred to as the no-plasticity group.

The age-distribution for the participants in the no-plasticity group is presented in Fig. 3 As can be seen, the majority of participants were older ($48$ of $54$ were from the oldest age groups; $60–80$ years). This pattern indicates that they did not have memory problems. Still, as was noted earlier, a group of participants ($N = 54$) had poor memory performance following enacted encoding and showed no enactment effect. Most of these participants were older. What may be the reason for their failure to utilize encoding support in the form of enactment? A possible answer to this question is provided by the functional neuroimaging data, showing that the enactment effect is related to activity in motor areas of the brain (see above). That is, the neuroimaging results hint that a failure to benefit from encoding enactment is related to a dysfunctional motor system.

To address this possibility, we explored whether individuals who failed to utilize encoding support had deviating scores compared to the rest of the sample on a select set of biological/neuropsychological factors. We aimed at including factors that directly or indirectly could be related to the integrity of the motor system. One of these factors was Parkinson’s disease, a neurological syndrome that implicates motor (striatal) brain areas. A second motor-related factor was performance on the Tower-of-Hanoi task. Completion of this task has been associated with premotor areas [22]. In addition to these ‘motor factors’, we also examined head trauma [23], exposure to toxic substances [24], and a family history of dementia [25]. With the exception for the Tower-of-Hanoi task, data on these factors were provided by the participants’ answers on specific questionnaire items. One item required the participants to indicate whether they had been treated for various neurological conditions, including Parkinson’s disease and head trauma. Another item probed whether the participants ever had been exposed to toxic substances (carbondioxide, organic solvents). A family history of dementia was indicated by the participants’ answers to whether their father and/or mother had some form of dementia. The Tower-of-Hanoi test was a five-disk version of the traditional puzzle [26]. Number of moves and time to completion were recorded, as well as the number of participants who did not finish the test within $20$ min.

Starting with the motor factors, only one person in the 1000-sample suffered from Parkinson’s disease (a 75-years old person). It is noteworthy that this person was part of the no-plasticity group, and hence failed to benefit from

![Fig. 3. Age-group distribution for participants that did not show an enactment effect.](image-url)
encoding enactment. Analyses of Tower-of-Hanoi performance were based on data from the second wave of testing. Results were available for 30 of the 54 participants in the no-plasticity group. Of these, 15 (50%) did not finish the test. This proportion is substantially higher than that for participants over 50-years of age [26]. Fig. 4 shows mean time to complete the task for the remaining 15 participants and for age-matched controls (based on Ref. [26]). As can be seen from the figure, on average, it took the no-plasticity participants 1 min longer than the controls to complete the task. Together, these results show that those individuals who failed to benefit from encoding enactment had poor Tower-of-Hanoi performance. While this finding may be taken as tentative evidence for a dysfunctional motor system, it should be noted that the Tower-of-Hanoi task is associated with increased activity in many other regions than motor areas [22], and is assumed to be a sensitive measure of executive functioning and visuospatial ability [26]. This leaves open the possibility that impaired Tower-of-Hanoi performance is related to a general cognitive deficit. Therefore, to assess global level of cognitive functioning, the mini-mental state examination (MMSE) [27] was considered. The MMSE was administered according to standardized procedures with a maximum score of 30. The performance of 70–80-year old persons in the no-plasticity group \( (N = 39) \) was compared with the reference value for these age groups [28]. As can be seen from Fig. 5, the performance of the no-plasticity persons was only marginally lower than the reference value, and their mean score \( (> 26) \) did not indicate impaired general cognitive functioning.

As for the ‘non-motor factors’, of the 54 participants in the no-plasticity group, six (11%) reported that they had been exposed to toxic substances, four (7%) reported that they had suffered a head trauma, and four (7%) reported a family history of dementia. There was no indication that the occurrence of these conditions was increased in the no-plasticity group relative to the remaining 946 individuals (control values: 11% for toxic substances; 10% for head trauma; and 7% for history of dementia).

### 6. Does lack of an enactment effect signal impaired dopaminergic neurotransmission?

The analysis above yielded no evidence that a failure to benefit from enactment is related to factors such as head trauma, dementia, or exposure to toxic substances. Instead, the consideration of factors related to the integrity of the motor system (Parkinson’s disease and performance on the Tower-of-Hanoi task) provided suggestive evidence that a failure to benefit from encoding enactment relates to a dysfunctional motor system. Although these factors are indirect indicators of the integrity of the motor system, an association of the enactment effect with motor brain areas is in agreement with the neuroimaging results that were summarized earlier.

It is of interest to relate the observation that a failure to benefit from encoding enactment becomes more frequent in advanced age (about 20% of the 80-year old participants were part of the no-plasticity group) to findings of substantial age-related decreases in dopamine function (for recent reviews, see Kaasinen and Rinne, this issue and Refs. [29,30]). There is an evidence for a loss of dopamine receptors in various brain regions of about 10% per decade [29], with onset in early adulthood. Interestingly, dopamine binding in striatal regions account for most of age-related differences in episodic memory function [31], suggesting that deficient dopamine functioning is a critical factor in cognitive aging [30]. The present finding of an age-related deficit in utilizing encoding support in form of enactment may relate to dopamine function. Individual differences in dopamine function have been found to correlate with individual differences in cognitive performance [32]. An interesting task for future studies would be to see whether individuals who do not benefit from enactment tend to have lower density of dopamine receptors than individuals of the same age who can benefit from such support. Moreover, it would be of considerable interest to examine if lack of an enactment effect has a diagnostic value in detecting diseases implicating the motor system.
Acknowledgements


References