

The effects of aging on time reproduction in delayed free-recall[☆]

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Abstract

The experiments presented here demonstrate that normal aging amplifies differences in time production occurring in delayed free-recall testing. Experiment 1 compared the time production ability of two healthy aged groups as well as college-aged participants. During the test session, which followed a 24-h delay and omitted all feedback and examples of the two target intervals, the two groups of aged participants' over-produced a 6 s interval. This effect is similar in form to errors shown by young participants, but twice the magnitude. Productions of a 17 s interval were generally accurate overall. However, further analysis indicated that the majority of aged participants over-produced the 17 s interval while a minority greatly under-produced the 17 s interval. Furthermore, aged participants showed violations of the scalar property of timing variability in the training session, while in the test session, only those who under-produced the 17 s interval showed this tendency. In contrast, training session performance was good for all participants. Experiments 2 and 3 investigated the ability of the participants in Experiment 1 to reproduce the length of a line from memory, under conditions analogous to those of the time production experiments. These experiments provided tests of the specificity of the errors observed in Experiment 1. Performance in the older participants was accurate, if more variable, compared to the young participants, in contrast to the time production results, indicating that production inaccuracy in free-recall is specific to interval timing in the current context.

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1. Introduction

Interval timing requires several cognitive processes such as an internal time basis, as well as attention, memory, and decision processes (Gibbon, 1977; Gibbon, Church, & Meck, 1984; Zakay & Block, 1997). Some of these processes are also mental functions that decline

with age, including attention, working memory, and reaction time (see Raz, 2000 for review). Likewise, the ability to time short intervals in the seconds to minutes range is served by the same brain structures and neurotransmitter systems that underlie higher cognitive functions, such as attention and working memory, and are most sensitive to age (Meck, 1996; Meck & Benson, 2002; Raz, 2000; Rao, Mayer, & Harrington, 2001). These factors may make timing tasks very sensitive measures of age-related changes in cognition.

Information-processing models, such as scalar expectancy theory (SET; Gibbon, Church, & Meck, 1984; see also Treisman, 1963), attempt to describe the cognitive processes involved in interval timing. In SET, as well as related models like the Attention Gate Model (AGM; Zakay & Block, 1997), attention to time allows pulses from a pacemaker to pass into an accumulator, and the accumulation of such pulses marks the passage of time.

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Judgments about the current duration versus previously relevant durations are made by comparing current accumulator values to samples from a reference memory distribution of accumulator values that were previously associated with the relevant event (e.g., in previous trials, how many pulses were accumulated before reinforcement occurred?). The distinguishing feature of SET is that in its computational form, variability in clock and memory processes is used to explain the common finding that standard deviations in timing performance are a constant proportion of (i.e., “scaled to”) the timed duration (Gibbon, Malapani, Dale, & Gallistel, 1997). This is referred to as the scalar property of timing variability, and is considered a domain specific example of Weber’s law, which describes the psychophysical relationship between stimulus magnitude and thresholds in a variety of stimulus domains (Gibbon, 1977). Experimental manipulations that lead to violations of the scalar property typically are thought to affect non-temporal aspects of performance (e.g., decision processes, motor output; Gibbon et al., 1997; Ivry & Hazeltine, 1995). Thus, measures of variability as well as accuracy are essential for understanding effects on interval timing.

Several previous studies of older adults’ interval timing accuracy have focused on age effects on attention’s role in SET and related models (Block, Zakay, & Hancock, 1998; Craik & Hay, 1999; Lustig & Meck, 2001; Vanneste & Pouthas, 1999). According to a recent meta-analysis (Block et al., 1998), verbal estimations grew shorter with age, verbal productions grew longer with age, and temporal production (most akin to the task used here) was unaffected by age. These effects were independent of target interval duration. AGM and SET explain attention effects in general by means of the switch or gate between the pacemaker and accumulator. When attention is directed away from time some proportion of pacemaker pulses do not increment the accumulator. As a result, a mismatch occurs between the estimate of current subjective time and the value of subjective remembered time because the direction of the effect depends on whether distraction occurred during acquisition or testing.

Although less heavily researched, there is also a potential link between the age-associated changes in interval timing and memory. A recent study (Perbal, Droit-Volet, Isingrini, & Pouthas, 2002) found time reproductions under distraction shortened with age, independent of interval duration, and were associated with poorer working memory performance. Other studies have found progressively noisy encoding of temporal values with increasing age (McCormack, Brown, Maylor, Darby, & Green, 1999; Wearden, Wearden, & Rabbitt, 1997), also largely independent of target interval duration.

The common findings that older participants’ timing errors, due to either memory or attention effects, are

independent of target interval duration are of particular interest because existing timing theories such as SET and AGM predict that if two durations were tested within the same session, older adults’ errors for both durations would be in the same direction. In the case of attention effects predicted by AGM (Zakay & Block, 1997) and SET (Meck & Church, 1983), the magnitude of the effects of attention stem from the degree to which a particular manipulation causes the switch between the pacemaker and accumulator to open. The direction of the attention effects is a function of when in the task distraction occurs or changes, and whether the timing task is prospective or retrospective (Block et al., 1998; Zakay & Block, 1997). As a net result of these factors, any given attention manipulation included during a given phase of a timing experiment should produce effects of uniform direction and magnitude regardless of the target interval duration. Similarly, studies conducted within the SET framework have identified a number of parameters that determine the function of the various temporal memory systems (Gibbon et al., 1984), all of which operate independently of the target interval duration. For example, “K*” multiplies all temporal values prior to storage in memory, resulting in a mismatch between currently elapsing and remembered time values (Meck, 1983, 2002). While the direction of both attention and memory effects predicted by SET is independent of the target interval duration, the magnitude of memory effects can grow with the target interval duration.

While studies of normal aging have produced evidence of duration-independent changes in timing accuracy, studies of Parkinson’s disease (PD), a neurodegenerative disease of aging (Fahn, 1995; Rakitin & Stern, 2002), have produced evidence of duration-dependent temporal memory effects. When taken off dopamine-replacement therapy, PD patients’ productions of the shorter of two intervals (e.g., 6 or 8 s) were longer than the standard intervals. Also, productions of the longer of two intervals (e.g., 17 or 21 s) were shorter than the standard intervals (Malapani, Deweer, & Gibbon, 2002; Malapani, Rakitin, Fairhurst, & Gibbon, 2002; Malapani et al., 1998). This “migration effect” strongly contrasted with performance by PD patients on medication, which was generally superior to that of aged controls (Malapani et al., 1998, 2002), and was determined to stem from a dopamine-dependent deficit in encoding temporal memory (Malapani et al., 2002). Thus, while current theories emphasize the likelihood that the direction of age-related changes in timing accuracy would be independent of target interval duration, there are known duration-dependent timing changes in pathological aging that must be considered as an alternative hypothesis.

In contrast to the many findings regarding timing inaccuracy in aging, the findings regarding timing variability are more mixed. Studies using diverse methods

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