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Memory Processing of Serial Lists by Pigeons, Monkeys, and People

Abstract. *List memory of pigeons, monkeys, and humans was tested with lists of four visual items (travel slides for animals and kaleidoscope patterns for humans). Retention interval increases for list-item memory revealed a consistent modification of the serial-position function shape: a monotonically increasing function at the shortest interval, a U-shaped function at intermediate intervals, and a monotonically decreasing function at the longest interval. The time course of these changes was fastest for pigeons, intermediate for monkeys, and slowest for humans.*

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The U-shaped serial-position function is a prominent benchmark of our understanding of memory processing. Typically recognition or recall memory is better for the first list items (primacy effect) and the last list items (recency effect) than it is for the middle items (*1*). The primacy effect has been traditionally thought to index long-term memory and the recency effect to index short-term memory. The form of the serial-position function along with the analyses of its primacy and recency effects has contributed to the support or demise of many theories of memory, from the early association network theories (*2*) to the more recent dual-process theories (*3*).

The importance of the serial-position function in testing theories of human memory processing makes it a natural choice for testing animal memory. Only recently have procedures been developed that allow researchers to test animal serial-position functions (*4*). Variables with proven effects on human serial-position functions can now be tested on animal serial-position functions to compare memory functions and cognitive processes and to examine the evolution of cognition.

We now report similar changes in the form of the serial-position function for pigeons ($n = 4$), monkeys ($n = 2$), and humans ($n = 6$) when retention interval was controlled. An immediate test revealed no primacy effect, but the effect emerged at intermediate tests to produce a U-shaped function, and the recency effect dissipated at the longest intervals. This qualitative similarity implies similar memory mechanisms.

The task for all three species was a serial-probe-recognition task. Trials were begun by pressing down a three-position T lever (monkeys and humans) or pecking on a 9 by 9.3 cm clear window (pigeons). Lists of color slides were rear-projected one at a time on the upper of two 12 by 9 cm screens separated 17 cm (center to center). Each of four memory items was displayed for 1 second (humans and monkeys) or 2 seconds (pigeons) with a 1-second interval between items. A probe item was projected on the lower screen after a delay (retention interval) from the last list item. If the probe item was a repeat of one of the list items ("same" trial), a correct response by humans or monkeys was a lever movement to the right and by pigeons a peck to a right disk (lighted red). Otherwise (on "different" trials) a left lever movement or a left disk (lighted green) peck was correct. Humans sat in a chair and held the lever box on their laps, monkeys were restrained in a primate chair, and pigeons worked in a Skinner box. Monkeys' correct responses were rewarded with a tone (500 Hz) plus a banana pellet or orange juice, pigeons with tone plus 2.8 seconds of mixed grain, and humans with tone only. Incorrect responses produced a lighted time-out period (5 seconds for humans and monkeys and 10 seconds for pigeons).

Test items for the pigeons (*Columba livia*) and monkeys (*Macaca mulatta*) were travel slides unique to that trial (limited to one trial per session) from a collection of 3000. Test items for the humans (two male and four female, 21 to 41 years old) were trial-unique kaleidoscope slides from a collection of 550. Kaleidoscope patterns prevented what would have been a performance ceiling effect with travel slides (*5*). Sessions were randomized sequences of ten "same" and ten "different" trials with the probe delay constant. Pigeons and monkeys were tested in four randomized blocks of six delays. Humans were tested in two randomized blocks of eight delays; the delays and sequence used at each delay were counterbalanced within and across human subjects. One sequence of particular items was used to test pigeons, two to test monkeys, and four to sixteen to test the humans.

The average serial-position functions are shown in Fig. 1. For each species, the 0-second delay functions show that memory for the first serial position was poor but progressively improved toward the end of the list. These serial-position functions markedly changed with probe delay; the primacy effect appeared, and by the middle two probe delays the serial-position functions had become U-shaped, showing primacy and recency effects for all three species. Further probe delay increases produced a progressive decline in memory toward the end of the list. These serial-position function changes were significant ($P < 0.03$) as tested by polynomial trend analyses (*6*).

These serial-position function changes are similar for all three species, but take place in about 10 seconds in pigeons, 30 seconds in monkeys, and 100 seconds in humans. This difference is important in the understanding of animal cognitive processes; the time scale for animals seems to be compressed relative to ours.

Human serial-position functions are typically obtained from recall (not recognition) tests. The retention interval is not precisely controlled (there is a free recall period), which probably accounts for subjects' showing only one of the effects described here: dissipation of the recency effect (*7, 8*). First-item recall is "de-

layed'' by first recalling the last list items (*8*). When ''recall delay'' has been eliminated by requiring subjects to press a button to indicate the list position of the test item, the primacy effect was suppressed on immediate test and (as here) emerged after a short delay (*9*). Thus, different human memory studies have shown indications or portions of the continuum of serial-position effects that we show here.

These dynamic serial-position function changes implicate two or more underlying memorial processes and help to discriminate among different theories of memory. No single-process (*2, 10*) explanations (for example, association network, memory decay, end-point distinctiveness) can adequately explain the range of the serial-position function changes shown here. The findings also discriminate among dual-process theories of memory. One dual-process theory says that items are temporarily stored in short-term memory (as revealed by the recency effect) and are moved from short- to long-term memory (as revealed by the primacy effect) by rehearsal (*3*). It has been argued (*9*) that subjects rehearse during the probe delay and that this rehearsal moves items from short- to long-term memory and is responsible for the eventual emergence of the primacy effect. If subjects can retrieve the items from memory in order to rehearse them, however, this same retrieval should have served as a sufficient basis for correct responding on the 0-second delay tests; we found no primacy effect in our 0-second delay condition. Another dual-process theory factors out short-term memory by subtracting a delayed recall function from an immediate recall one (*11*). But we found no primacy effect on the immediate test; under the latter dual-process theory, negative memory values would result for the first list items. Dual-process interference theory fares better than most in relation to our results. At short delays, retroactive interference (last list items interfering with first ones) is large (*12, 13*) and could account for suppression of the primacy effect. Rapid dissipation of retroactive interference (*12, 13*) could account for the emergence of the primacy effect at intermediate probe delays. Small initial proactive interference (first list items interfering with the last ones) and its comparatively slow growth (*13, 14*) could account for a strong initial recency effect and allow it to linger after the primacy effect has emerged. Our findings represent a constraint on current and future theories of memory. Recognition of the dynamic serial-position function changes across retention intervals should lead to better conceptualizations of the underlying processes of memory.

The U-shaped function has been an important concept of memory processing since the study of memory began (*15*). It seems, however, to be a transitional state, where two processes with different time courses overlap. Furthermore, the time scale is different for the three species. The delay time that reveals a U-shaped function with one species may be too long or too short for another species. The time scale differences are quantitative differences. Similar dynamic changes, however, implicate qualitative similarity. The basic memory processes and the interaction of processes producing the serial-position function seem to be similar for pigeons, monkeys, and humans.

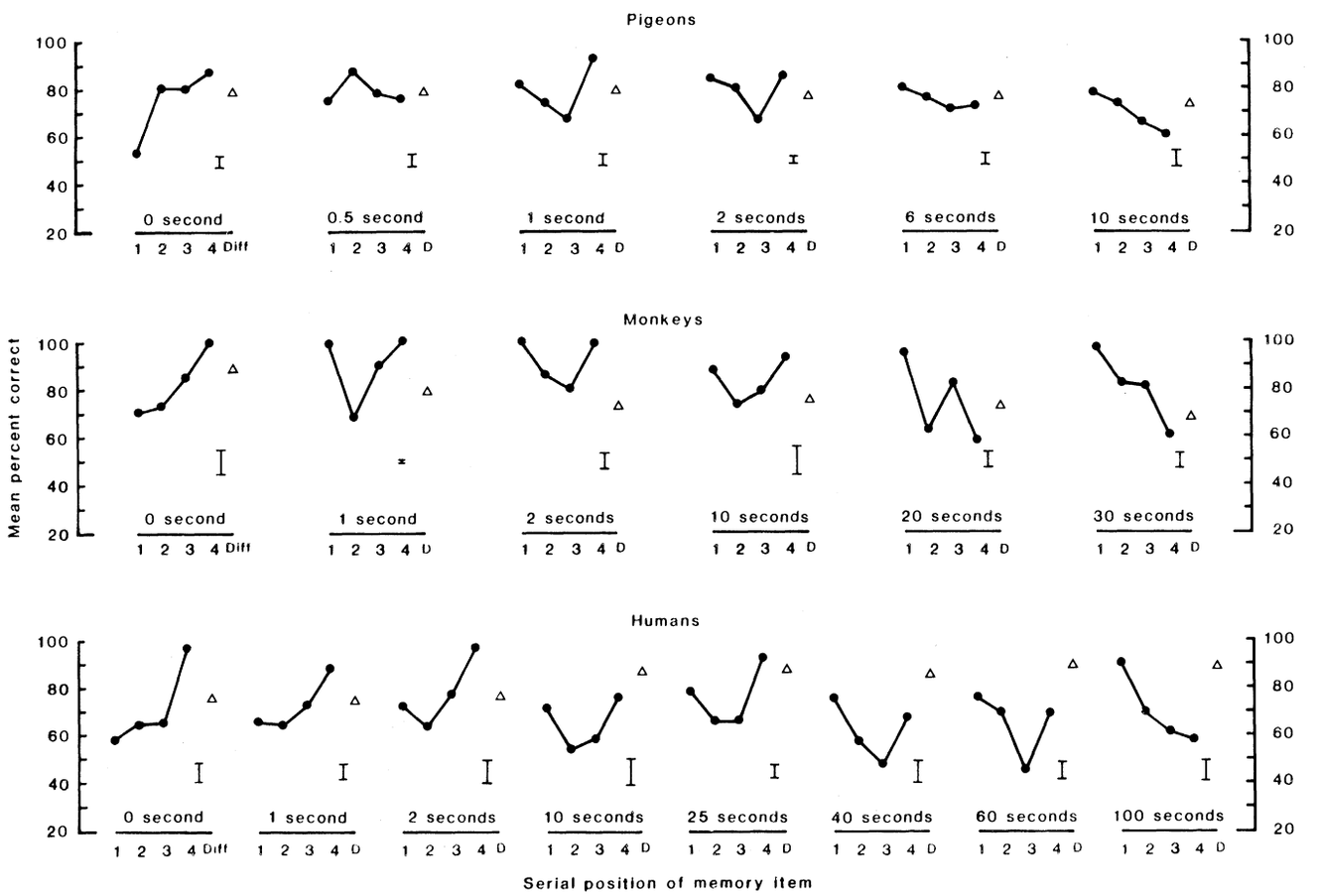

Fig. 1. Mean memory performance for four-item serial lists at different probe delays (retention intervals), the interval between the last list item (labeled 4) and the probe test item. The bar shown for each serial-position function is the average standard error of the mean for the four serial positions (''same'' trials). Open triangles show performance on ''different'' trials where the probe item matched none of the four list items.

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Reduced Numbers of Somatostatin Receptors in the Cerebral Cortex in Alzheimer's Disease

Abstract. *Somatostatin receptor concentrations were measured in patients with Alzheimer's disease and controls. In the frontal cortex (Brodmann areas 6, 9, and 10) and temporal cortex (Brodmann area 21), the concentrations of somatostatin in receptors in the patients were reduced to approximately 50 percent of control values. A 40 percent reduction was seen in the hippocampus, while no significant changes were found in the cingulate cortex, postcentral gyrus, temporal pole, and superior temporal gyrus. Scatchard analysis showed a reduction in receptor number rather than a change in affinity. Somatostatin-like immunoreactivity was significantly reduced in both the frontal and temporal cortex. Somatostatin-like immunoreactivity was linearly related to somatostatin-receptor binding in the cortices of Alzheimer's patients. These findings may reflect degeneration of postsynaptic neurons or cortical afferents in the patients' cerebral cortices. Alternatively, decreased somatostatin-like immunoreactivity in Alzheimer's disease might indicate increased release of somatostatin and down regulation of postsynaptic receptors.*

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One approach to studying Alzheimer's disease is to examine its neurochemistry after death. Several neurochemical deficiencies related to the illness have thus far been identified. The best studied is a reduction in choline-acetyltransferase activity in the cerebral cortex, which has been attributed to a loss of cholinergic neurons in the basal nucleus of Meynert (*1*). Less marked cortical deficiencies of noradrenaline, serotonin, substance P, and γ-aminobutyric acid have been described (*2*). In addition, a widespread reduction in cortical somatostatin-like immunoreactivity (SLI) has been associated with Alzheimer's disease (*3*). Immunocytochemical studies have suggested that the alteration reflects degeneration of intrinsic somatostatin cortical neurons (*4*), and somatostatin appears to be a neurotransmitter or neuromodulator in the central nervous system having electrophysiologic effects on central neurons (*5*). The clinical importance and role of this peptide in the pathophysiology of Alzheimer's disease remains unknown.

Recently somatostatin receptors have been reported in the brain, the pituitary, and peripheral tissues (*6*). We have characterized somatostatin receptors in the human brain (*7*). Since the receptor is an integral component of the mechanism by which somatostatin activates or modulates neuronal activity, a study of somatostatin receptors may provide further insight into the role of somatostatin in Alzheimer's disease. In addition, an understanding of receptor changes associated with disease has implications for the development of drugs that modify receptor function. We have measured concentrations of somatostatin receptors in both control and patient tissue post-mortem and have related these findings to concentrations of SLI.

Postmortem brain tissue from 12 Alzheimer's patients (ten male, two female; mean age, 76.8 ± 2.3 years; range, 72 to 90 years) and from 13 controls (six male, seven female; mean age, 69.5 ± 3.9 years; range, 36 to 86 years) was dissected as previously described (*8*). The diagnosis in the Alzheimer's patients was confirmed by neuropathology in all cases, and other neuropathologic conditions were excluded. Neuropathologic examination showed the control tissues were normal in all cases except one that had some small lacunar infarcts in the white matter. Brain tissues from the patients and controls were handled identically. In all cases the time between death and storage of the tissue at −70°C was less than 24 hours (patients, 11.3 ± 2.0 hours; controls, 11.5 ± 1.6 hours); we have found that somatostatin receptors are stable for up to 24 hours in an animal model simulating human autopsy conditions.

Tissue extraction and radioimmunoassay for SLI were carried out as described (*9*). The assay recognizes amino acids 6 to 10 of tetradecapeptide somatostatin. Somatostatin 14 and somatostatin 28 are recognized on an equimolar basis. Somatostatin receptors in human brain membranes were measured with ^{125}I-labeled [Leu^8, DTrp^{22}, Tyr^{25}] somatostatin 28 (*10*). The peptide was iodinated by the chloramine-T method and then purified by high-performance liquid chromatography (HPLC). It was eluted isocratically with 0.25*M* tetraethyl ammonium formate buffer, *p*H 3.5, with 17 percent *n*-propanol as an organic modifier on a μBondapak C18 column (Waters Associates, Milford, Massachusetts). The major radioactive peak eluted at 18 minutes and a minor peak, corresponding to less optimal binding, at 32 minutes. The binding of ^{125}I-labeled [Leu^8, DTrp^{22}, Tyr^{25}]somatostatin 28 to somatostatin receptors was performed in a final volume of 100 μl tris-HCl buffer