Analogical Transfer in Sequence Learning

Human and Neural-Network Models of Frontostriatal Function

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INTRODUCTION

Analogical transfer in problem solving is a fundamental aspect of human intelligence that involves exploiting knowledge about the solution for one problem to solve another.[1] In order to provide a quantifiable measure of analogical transfer in sequence learning (ATSL), we developed a novel extension of the serial reaction time (SRT) paradigm. To quantify the underlying representational and computational requirements for ATSL we extended a neurobiologically based model of primate prefrontal cortex (PFC) and basal ganglia function in visuomotor sequence learning[2] to address analogical transfer. We compared the behavior of this model with preliminary data from normal subjects and patients with frontostriatal dysfunction [idiopathic dopa-sensitive Parkinson's disease (PD)] in the ATSL paradigm.

ANALOGICAL TRANSFER PARADIGM

Reaction times (RTs) for a series of motor responses to visual stimuli are significantly reduced if the stimuli appear in a repeating sequence, as opposed to in a random order, thus demonstrating a sequence learning effect.[3] Based on this observation, the ATSL task involves pointing to targets illuminated one at a time on a touch-sensitive screen. After a target is touched it is extinguished, RT is recorded and the next target is displayed. Targets appear in blocks of two types—random and sequence. In random blocks, 120 targets are presented in random order. In sequence blocks, targets are presented in a sequence of the structure $ABC\underline{B}^1\underline{C}^2\underline{D}^3C^1D^2E^3D^1E^2F^3$ . . . of length 24 that is repeated five times for a total of 120 targets. A–H denote screen locations. Superscripts indicate analogical schema "position" where positions 1 and 2 are predictable, and position 3 unpredictable. Note, for example, how $\underline{B}$ and $\underline{C}$ are predicted respectively by

the element two places behind (referred to as "n − 2"), whereas D is unpredictable (referred to as "u"). This pattern "n − 2, n − 2, u" repeats throughout the sequences, and is the underlying analogical schema.

An experimental session starts with 20 random practice targets. The recording session starts with a random block of 120 trials, followed by *three different* sequence blocks (that share the analogical schema "n − 2, n − 2, u") of 120 trials each, and a final random block of 120 trials. Partially informed subjects are told a pattern may repeat in some sequences. Fully informed subjects are additionally shown a diagram of the repeating schema before and once during the test.

A MODEL OF ANALOGICAL TRANSFER

Performance of our existing sequence learning model[2] on this task indicates sequence learning capacity alone is not sufficient for ATSL (FIG. 1A). In the updated model (FIG. 1B), instead of learning sequences of specific items, the model recognizes if elements are novel or repeated, and learns sequences of descriptions in terms of novel versus repeated elements. In this sense the sequence ABABC is the same as the sequence EFEFG. Both can be described "u, u, n − 2, n − 2, u".

HUMAN PERFORMANCE

Normal subjects display significant analogical transfer between sequence blocks in the fully informed condition (FIG. 2A), and less but still significant learning in the partially informed condition (FIG. 2B). Patients with PD appear to display impaired transfer in both conditions (FIG. 2C and D), similar to impairment in SRT of patients with PD.[4]

CONCLUSIONS

ATSL is a new, quantitative model of analogical transfer. Normal subjects and a model with capacities for (1) memory of previous elements, (2) recognition of element repetition, and (3) modulation of behavior based on this recognition can realize ATSL. Patients with PD, however, and a model that does not possess these abstract capacities are both impaired in ATSL. These capacities for abstraction are likely based in the PFC and its corresponding frontostriatal circuitry, at the head of the developmental corticofrontal hierarchy that forms progressively higher levels of representation, from muscle contraction parameters to abstract representations for goal-directed action.[5]

Our repeating sequences have length 24 (difficult even for normal subjects),[4] whereas the underlying analogical schema "n − 2, n − 2, u" has length 3. This task thus attempts to dissociate sequence learning from the induction and transfer[1] of a three-element analogical schema. The limited but existing ATSL performance of subjects with PD demonstrates an impaired capacity for analogical transfer that is at least partially independent of their impaired SRT capacity (which would be

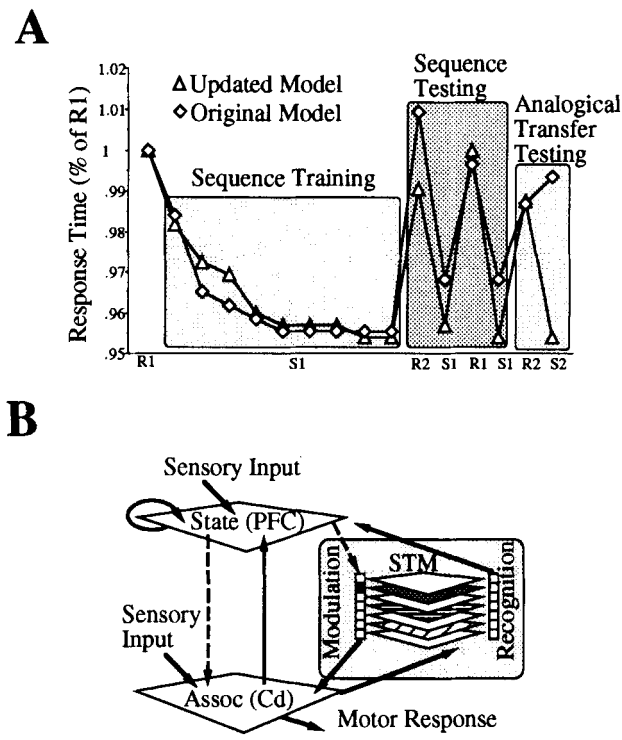

FIGURE 1. (**A**) Performance for the original and updated models of analogical transfer in sequence learning (ATSL). R1, R2 represent random blocks; S1, S2, two sequence blocks with different surface structure but shared analogical structure. R1 establishes the baseline response time. Sequence training: nine blocks of training with S1 yield standard SRT reduction in response time (RT). Sequence testing: learning demonstrated with sequence (reduced RT) and random (increased RT) blocks. Analogical transfer testing: with a new sequence (S2) that has the same analogical structure as S1. The updated model displays generalization (ATSL) to S2, whereas the original model does not. (**B**) Extension to sequence model to accommodate ATSL. Original model[2] encoded sequence state in prefrontal cortex (PFC) and learned associations between states and corresponding sequence elements. Extension is in shaded region. Layers represent 5 × 5 arrays of simulated neurons. Solid arrows, fixed connections; dotted arrows, modifiable connections. Seven short-term memory (STM) modules invariantly encode the n − 1, n − 2 . . . n − 7th previous responses. A recognition function compares the current response with these STMs. State now gets input from recognition and thus encodes both the abstract nature and the surface structure of the sequence. Modulatory neurons modulate the stored STM representations into the output. State controls this modulation. Each time a recognition occurs (that is, when sequence element is repeated), the current internal state becomes associated with the neuron in modulation that directs the recognized STM element to the output (Assoc). After training on ABABC, the model will recognize (and be able to repeat, and respond with reduced SRT) all sequences of the form 12123, or "unpredictable, unpredictable, n − 2, n − 2, unpredictable."

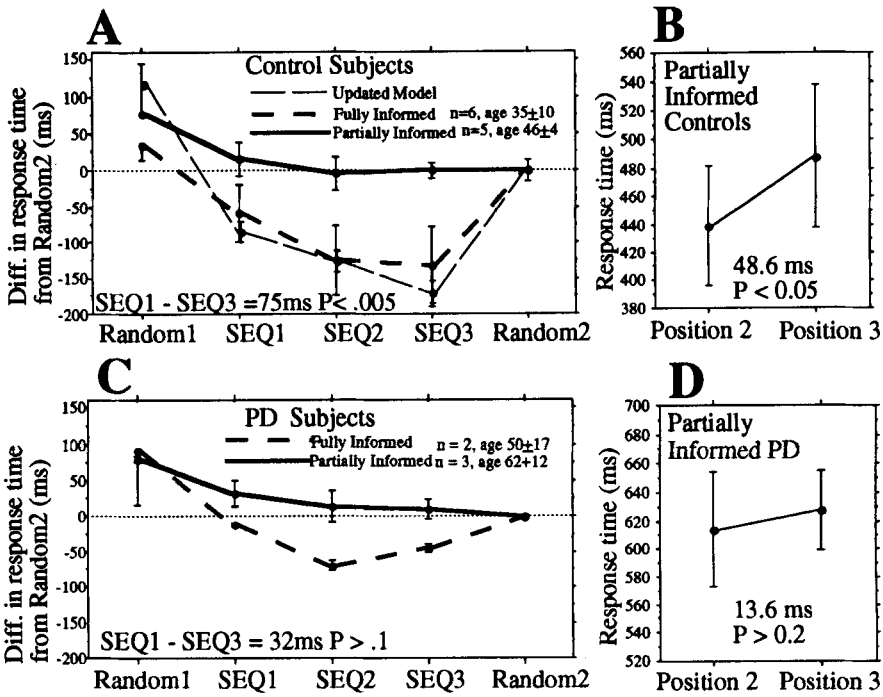

FIGURE 2. Human results in analogical transfer in sequence learning (ATSL) with a random block (Random 1), followed by three different sequence blocks (SEQ1–3), and a final random block (Random 2). (**A**) Fully informed control subjects and the updated model display significant transfer (as measured in the difference between blocks SEQ3 and SEQ1). That the three SEQ blocks are all different indicates the improvement came from learning the abstract structure common to all three. The fact that this learning does not transfer to the Random 2 block indicates that real learning has taken place, rather than simple motor facilitation. (**B**) Partially informed controls display significant learning of the analogical schema, as their reaction times in SEQ3 for position 3 elements (unpredictable) are significantly higher than those for position 2 elements (predictable). (**C**) Fully informed subjects with PD appear to display impaired transfer with respect to controls. (**D**) Partially informed subjects with PD appear to display impaired learning of the analogical schema. A three-factor ANOVA revealed the following significant effects: (1) Instruction (fully informed, partially informed), $F(1,36) = 61$, $p < .0001$; (2) Subject type (control, PD), $F(1,36) = 14$, $p < .001$; (3) Block (SEQ1, SEQ2, SEQ3), $F(2,36) = 5.5$, $p < .01$. (1 × 2) Instruction × Subject-type interaction, $F(1,36) = 5.5$, $p < .05$. Although these preliminary results indicate subjects with PD are impaired in ATSL with respect to controls, further verification with additional patients and controls is required.

of little value for sequences of length 24).[4] This implies that distinct frontostriatal systems for simple and analogical transfer sequence learning capacities may be dissociably impaired in Parkinson's disease.

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