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Evaluating the Trace Deletion Hypothesis and processing deficit accounts: a computational modeling approach

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Introduction

Caplan et al. (2007) have proposed that the sentence processing deficit in agrammatic aphasia is a consequence of intermittent deficiency (ID) in the capacity to carry out syntactic, semantic, and task-related computations. We operationalize ID as a specific impairment in a cognitive architecture for sentence processing. We also implement a complementary hypothesis of slowed processing (SP), which ascribes the deficit to a pathological slowdown (Hanne et al., 2011). We contrast these processing deficit accounts with a representational deficit account, the Trace Deletion Hypothesis (TDH) (Grodzinsky, 2000), which claims that patients suffer from impairment in their syntactic representation.

Method

We implemented computational models of the two classes of accounts in a cue-based retrieval architecture of sentence processing (Lewis & Vasishth, 2005). We modeled the sentence-picture matching task reported in Hanne et al. (2011). In this study, patients (n=7) and aged-matched controls (n=7) listened to German canonical (*Der Sohn fängt den Vater* 'The son is catching the father') and analogous reversible non-canonical (*Den Sohn fängt der Vater* 'The father is catching the son') sentences while they were shown two pictures (a target and a distractor) on the screen. After sentence presentation, participants selected the picture matching the sentence. The data consists of eye movements during sentence presentation, response times and accuracy. We first modeled controls' offline responses (accuracy and response time) and then induced impairments in the model depending on the hypotheses: ID as increased utility noise in the system (utility noise defines the degree of nondeterminism in selecting processing rules), SP as slowed procedural memory, and the TDH as an absence of trace information in the parse tree. To characterize the between subject variance in aphasia, we estimated different values for the two parameters (utility noise and procedural memory) for each patient. We evaluated the models using the root-mean-square deviation measure.

Results and discussion

The models with processing deficits (ID and SP) outperformed the models with a representational deficit (TDH). In fact, assuming only one of the two processing deficits was inadequate to capture all the crucial patterns in the data. The additive model captured: (1) reduced accuracy in the picture matching response, (2) chance-level performance on non-canonical sentences, (3) high response times, and (4) divergent eye movements for correct and incorrect responses. Moreover, the model

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captured the high variability between patients' responses–different patients showed different degrees of intermittent deficiency and slowed processing, and they formed a more spread out distribution of parameter values than controls (see the figure). In sum, the modeling results support the hypotheses of processing impairments as the source of agrammatic aphasia, and also demonstrate the differences at the level of individual patients in the severity of ID and SP.
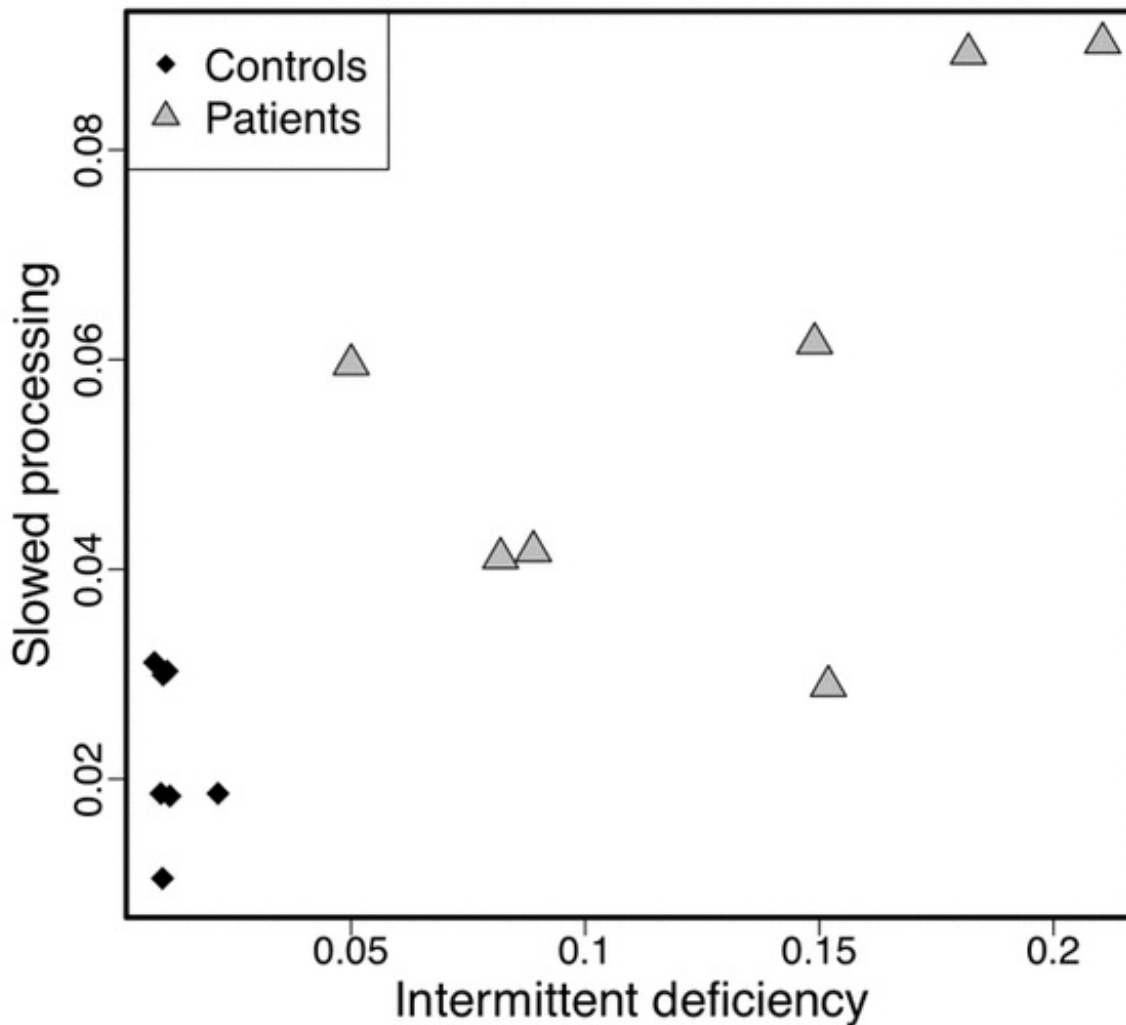
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