

**A Single System Account of the Relationship
between Semantic and Lexical Deficits in Semantic Dementia**

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Semantic dementia (SD) is a selective impairment to the semantic system due to progressive atrophy of the frontal and the temporal cortices. Tasks usually impaired in SD patients include object/picture naming, word-picture matching, and delayed copying. SD patients also show compromised performance on a number of lexical tasks such as word reading, spelling, and lexical decision. Both types of deficits are most prominent for irregular low-frequency items.

Performance measures on the tasks mentioned above correlate with each other (cf. Patterson et al., 2005). This finding has motivated the idea that lexical and semantic deficits arise from damage to a single integrated system. Notably, however, the correlations are partial, and there are patients who do not show a reading deficit despite severe semantic impairment (Blazely et al., 2005; Cipolotti & Warrington, 1995).

The aim of the current study was to address the partial but not perfect correlation between SD patients' performance on semantic and lexical tasks. Within a single system, the relative robustness of semantic and lexical knowledge can still depend on a number of factors. We hypothesize that the observed differences in performance might arise from differences in experience, differences in the capacity of the direct pathway mapping orthography to phonology, and/or differences in the spatial distribution of brain atrophy.

We support the hypothesis with a neural network simulation that included four visible layers - visual (V), motor (M), orthographic (O), and phonological (P), with full bidirectional connectivity between all visible layers and a hidden layer, and full recurrence within the hidden layer. There was also a fully recurrent direct pathway between O and P. The network was trained given V or O input to produce either all four corresponding outputs or only P

output. Then, it was damaged by lesioning hidden units and links between the hidden and visible layers. Testing included naming (producing P to a V input) and reading (producing P to an O input).

Three aspects of the network were manipulated singly and in combination: training regime (ratio of V to O input = 1:1 vs. 1:2 vs. 2:1); direct pathway size (10, 20 or 30 units); and lesion distribution (unbiased vs. biased toward V vs. biased toward O).

We examined the relationship between the network's accuracy on naming and its accuracy on reading irregular words. Each manipulation affected this relationship. The biggest effects were produced by extreme combinations of manipulations. The overall trend mirrored the patient data - as naming performance went down, so did reading. Importantly, however, the correlation was not perfect, and the distribution encompassed cases with severely impaired naming but little or no reading deficit.

The simulations indicate that we can account for SD patients' naming and reading data within a single system in which there is individual variation in experience, structure, and locus of damage. We suggest that it may be more useful to think of these patients as coming from a single distribution and falling at different points within that distribution rather than as individual cases somehow fundamentally different from each other.