

Cumulative Semantic Interference

Howard et al. (2006) showed that the speed at which target pictures are named increases as a function of prior retrieval of other exemplars of the same semantic category, and is unaffected by lag since presentation of the last item in the category - the so-called *cumulative semantic interference* (SI) effect. In order to explain this effect, Howard et al. (2006) proposed three essential mechanisms. The first is *shared activation of semantic features* among categorically related objects. The second, *priming* of semantic-to-lexical connections or lexical representations, can be viewed as an example of a conceptual implicit memory mechanism (e.g., Mulligan, 2012). The third mechanism is *lexical selection by competition* (LSC). Howard et al. implemented these mechanisms in a computational model that was able to simulate the cumulative SI effect.

Oppenheim et al. (2010) subsequently linked the cumulative SI effect to a phenomenon known as retrieval-induced-forgetting (RIF), in which cued recall of an item from episodic or explicit memory is impaired by an earlier retrieval of a related item (Levy & Anderson, 2008). Thus, incremental learning weakens semantic-to-lexical connections as target items are named, making those representations less accessible on later trials. The model was able to simulate the cumulative SI effect with selection accomplished by a threshold (i.e., 'horse race') rather than LSC mechanism by including a top-down mechanism that compared activation among lexical candidates and boosted activity until one output (the target) was the most active. Naming latencies were linearly related to the operation of this booster mechanism, attributed to the left inferior frontal gyrus (IFG).

We tested hypotheses from the two rival models of cumulative SI in a perfusion fMRI experiment. Priming of semantic features and LSC were expected to engage perirhinal (PRc) and left mid-temporal cortices in accordance with previous research (Indefrey & Levelt, 2004; Wang et al., 2010). Alternatively, RIF was expected to engage the hippocampus, anterior cingulate cortex (ACC) and right IFG based on prior work (Levy & Anderson, 2008), in addition to the proposed booster mechanism in the left IFG.

Experimental design and analysis

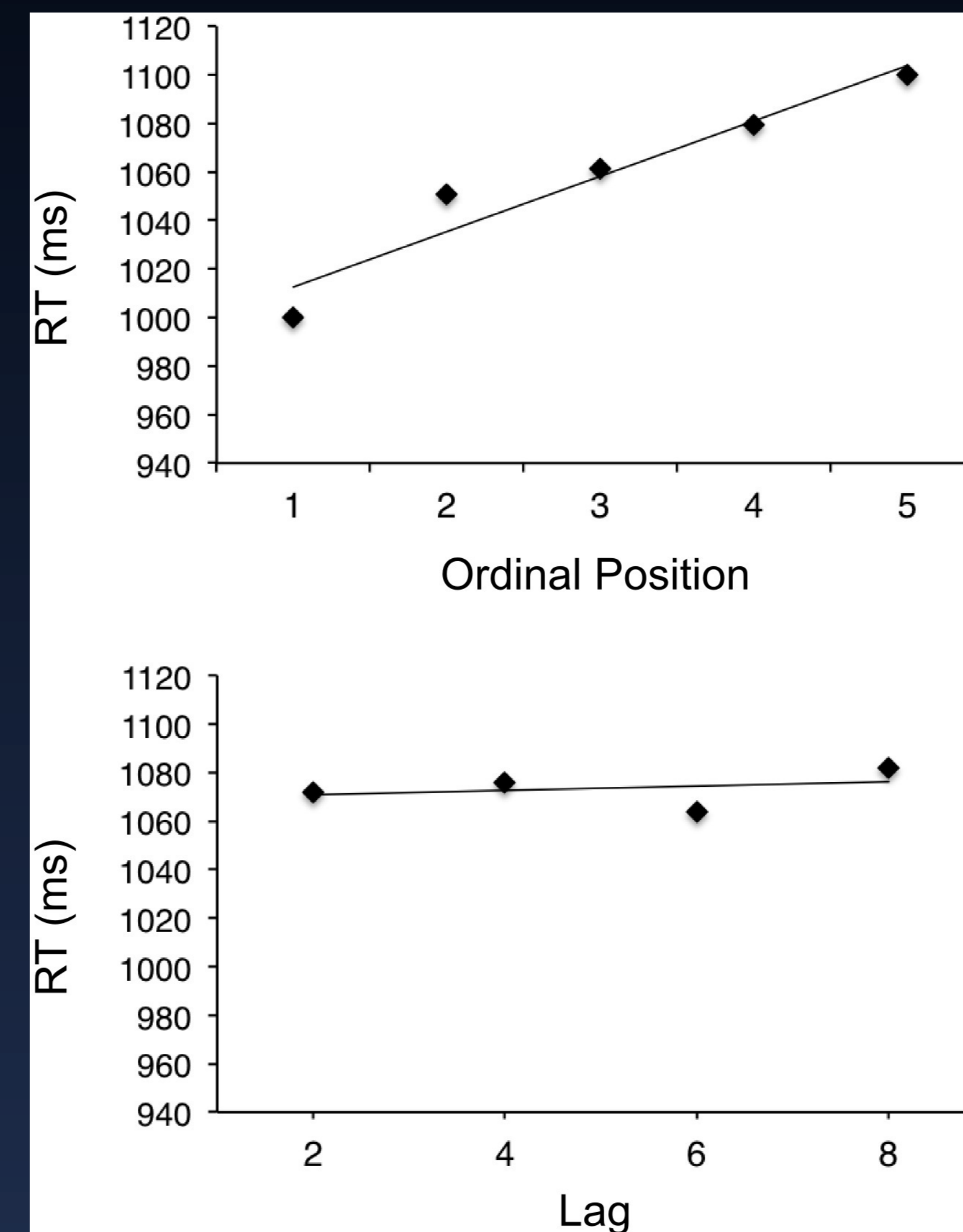
Participants. 24 healthy volunteers (10 male) with a mean age of 23 years.

Materials and Procedure. The materials were identical to those employed by Howard et al. (2006). 165 colour picture stimuli were used, comprising 120 target items (5 exemplars for each of 24 semantic categories) and 45 filler items. In each of 24 experimental lists (each corresponding to a separate participant), category exemplars were separated by 2, 4, 6 or 8 intervening items (i.e., lags), and each lag order was realized equally often with each category (i.e. once). The procedure was also identical, with the exception of the inter-trial interval (ITI) that was doubled to 6 s to enable efficient estimation of the perfusion signal response.

Image Acquisition. Perfusion images were acquired using an arterial spin labelling (ASL) sequence on a Bruker Medspec 4 Tesla system. 446 alternating control and tag images were acquired (T1 = 800 ms, T2 = 1800 ms, TR/TE = 2500/11 ms, matrix = 64x64, voxel in-plane resolution = 3.5x3.5 mm, and flip angle = 90°). Volumes comprised 12 slices, 6 mm thick, and were oriented to optimise coverage of the temporal and inferior frontal lobes. A 3D T1-weighted structural image was also acquired (1 mm isotropic voxels).

Image analysis. SPM8 and the ASL toolbox (Wang et al., 2008). A priori regions of interest (ROIs) were defined using 3D probabilistic atlases. A height threshold of $p < .001$ was adopted with a cluster FWE corrected threshold of $p < .05$ estimated for the whole brain (153 contiguous voxels) and for each ROI using a Monte Carlo estimation procedure with 10,000 simulations (*alphasim*, AFNI).

Results



Behavioral data: Trials on which participants omitted responses were excluded (7.4%), as were voice key errors (0.2%), naming errors (6.67%) and trials in which naming onset reaction times (RTs) were < 250 ms and > 2000 ms (1.8%). For ordinal positions 2-5, there was a significant main effect, $F(3, 69) = 4.21, p < .01, \eta^2 = .13$, and no effect of lag or interaction between lag and ordinal position ($F1 < 1.5$ for all effects). For all 5 ordinal positions, collapsed across lag, RTs increased monotonically as a function of ordinal position $F(4, 92) = 10.78, p < .001, \eta^2 = .31$, replicating the results of Howard et al. (2006) (see RT plots at left).

Imaging data: Planned *t*-contrasts performed on the lag regressors failed to reveal any significant perfusion signal changes in the ROI or whole brain analyses. Significant increases in perfusion signal were revealed by the *t*-contrast examining the linear trend of ordinal position, collapsed across lag, in left mid-MTG, left perirhinal cortex, and left IFG (comprising operculum/insula) (Figure 2). No significant perfusion signal changes were observed in hippocampus, ACC, or right IFG ROIs, nor any for the reverse contrast.

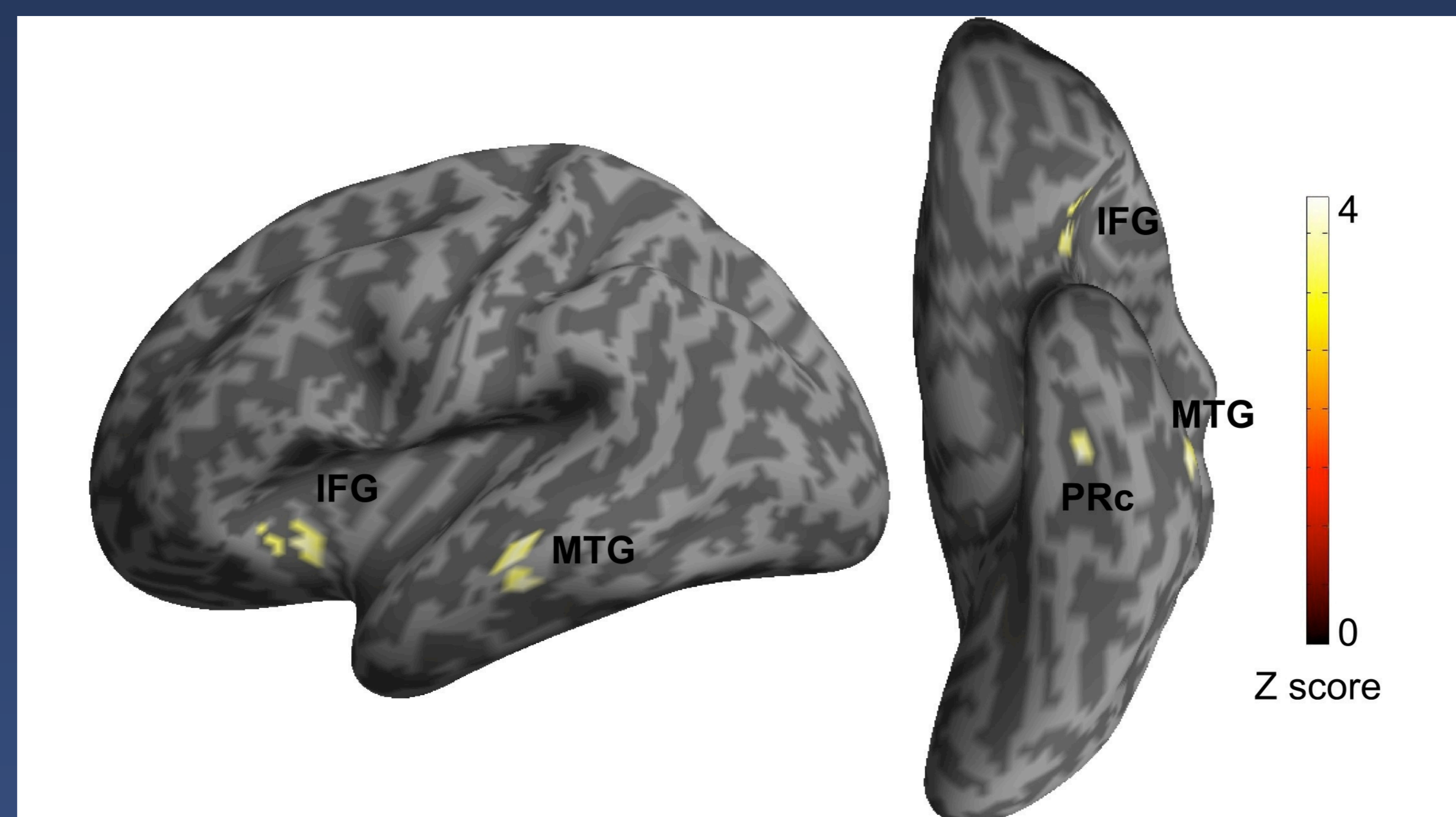


Figure 2. Left hemisphere cerebral regions showing significantly increased perfusion signal for exemplar naming as a function of ordinal position, superimposed on lateral (left) and ventral (right) inflated surface renderings of an individual brain. PRc - perirhinal cortex; MTG - middle temporal gyrus; IFG - inferior frontal gyrus.

Results cont'd

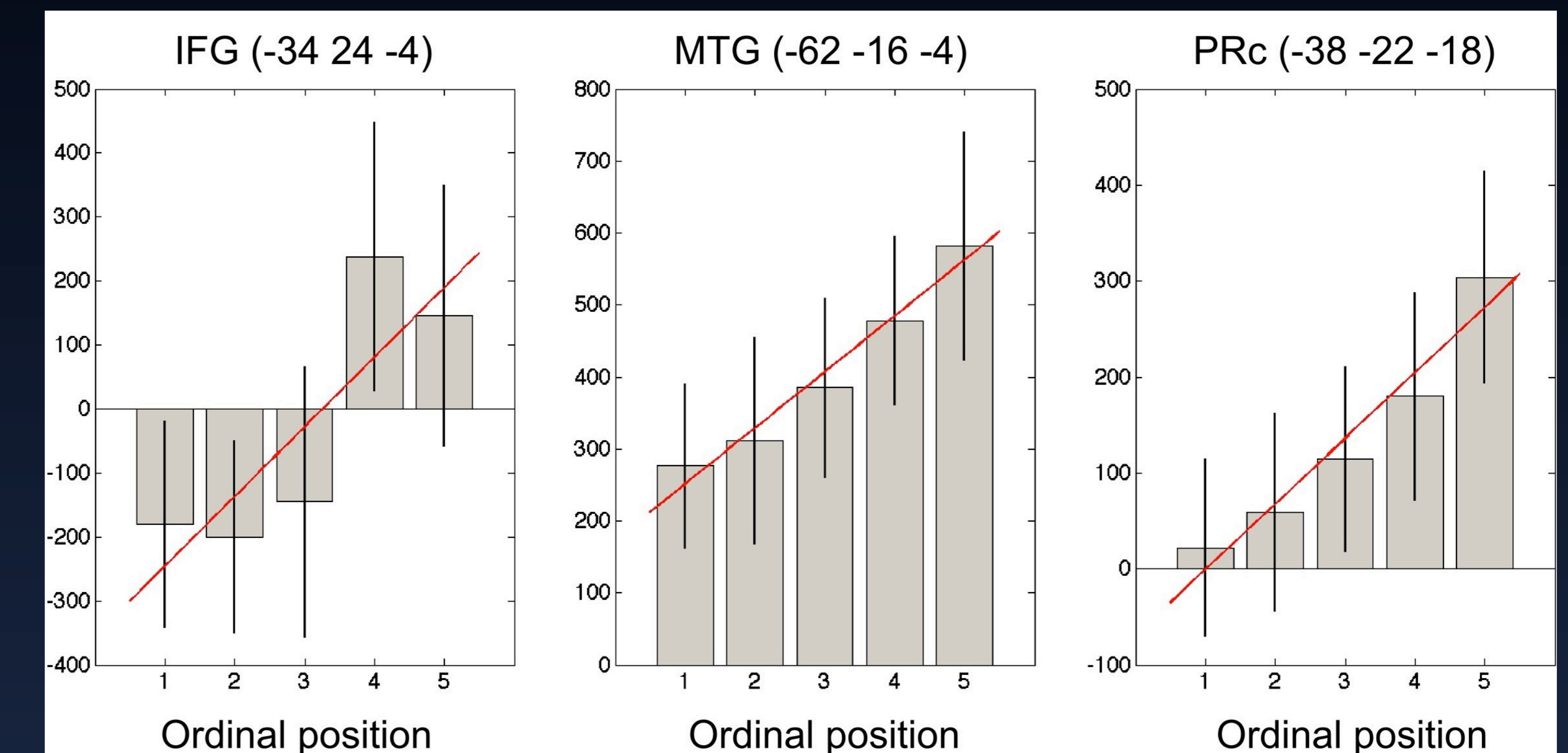


Figure 3. Plots of mean centered effect sizes at peak voxels with linear trend lines superimposed in red. Bars represent SEMs. PRc - perirhinal cortex; MTG - middle temporal gyrus; IFG - inferior frontal gyrus.

Plotting the significant perfusion signal responses (see Figure 3 above) revealed the left IFG response did not conform to a linear function.

Conclusions

The present findings demonstrate that regions typically engaged in conceptual implicit priming and lexical (lemma) level selection, such as the left PRc and mid-MTG, respectively, are engaged incrementally by the cumulative SI effect. None of the regions associated with a RIF account of cumulative SI, such as the hippocampus, right IFG and ACC showed significant perfusion signal changes (e.g., Levy & Anderson, 2008). Further, while the left IFG showed significant perfusion signal changes across ordinal presentations, these responses did not conform to a linear function.

The results are consistent with the predictions of the Howard et al. (2006) model. Future modelling of cumulative SI effects may benefit from making a distinction between the conceptual implicit versus explicit memory requirements (e.g., Mulligan, 2012) of different naming paradigms.

References

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Acknowledgements

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