the basis of monkey single-cell recording (Kaliukhovich & Vogels, 2011), modulate repetition suppression is actually somewhat parenthetical to predictive coding. This is because the "predictions" manipulated in the Summerfield et al. paradigm are likely to be conscious/strategic (and so may be less prevalent in monkeys). Yet the "predictions" in predictive coding theory are automatic, intrinsic properties of the brain networks that do not necessarily depend on conscious expectation. Thus while the effects of higher-order expectancy are clearly interesting and important (and probably generated by prefrontal regions that act on the ventral stream), the lack of such expectancy effects in other paradigms (Kaliukhovich & Vogels, 2011; Larsson & Smith, 2012) should not be used to reject predictive-coding models.

Another approach used to support predictive coding models of repetition suppression is to examine changes in connectivity between brain regions. Our own work, for example, has used Dynamic Causal Modelling (DCM) of fMRI data to show that repetition of bodies (Ewbank et al., 2011) or faces (Ewbank, Henson, Rowe, Stoyanova, & Calder, in press), at least across different images, modulates backward connections from "higher" regions in fusiform cortex to "lower" regions in extrastriate occipital cortex. Gotts et al. wondered why this modulation by repetition reflected a more positive coupling parameter in the DCM, when according to predictive coding, one might expect a more negative coupling associated with the suppression of prediction error in lower regions by higher regions. Again, however, the precise interpretation is more subtle because we do not know which types of excitatory/inhibitory neurons contribute to the BOLD signal. Moreover, due to high interdependency between parameters in such recurrent DCMs, inference is often more appropriate at the level of model selection rather than model parameters (Rowe, Hughes, Barker, & Owen, 2010). Thus, although we discussed our results in terms of predictive coding, the main conclusion of the Ewbank et al. papers (which were based on model selection) is that repetition suppression is not purely a local phenomenon (such as sharpening or even neuronal fatigue; Grill-Spector, Henson, & Martin, 2006), but also entails interactions between brain regions. This claim is consistent with both predictive coding and synchrony theories.

A further reason why DCM for fMRI may be limited in its ability to distinguish theories like predictive coding and synchrony is that the modulatory inputs (repetition in this case) need to be sustained over several seconds in order to have an appreciable impact on the network dynamics (Henson, Wakeman, Phillips, & Rowe, 2012). This is why we used a blocked design in the Ewbank et al. studies, where the modulation was assumed to operate throughout blocks. As Gotts et al. observe, such designs are undesirable from a behavioral perspective (e.g., encouraging use of conscious expectancies like those discussed above). Randomized designs (e.g., Henson, 2012) are clearly preferable, but in order to test for changes in effective connectivity as defined by dynamic models like DCM, data with higher temporal resolution are needed (e.g., Garrido, Kilner, Stephan, & Friston, 2009). Thus we agree with Gotts et al. that an exciting future direction is to examine connectivity, perhaps via synchrony, between regions using methods like EEG/MEG.

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Repetition accelerates neural dynamics: In defense of facilitation models

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Abstract: Gotts, Chow and Martin give an excellent contemporary summary of the neural mechanisms that have been proposed to underlie the effects of stimulus repetition on brain and behavior. Here I comment on their Facilitation mechanism, and provide EEG evidence that repetition can accelerate neural processing.

Gotts et al. (2012) review four types of neural mechanism that might underlie the reduced brain response associated with repetition of a stimulus: Facilitation, Sharpening, Synchrony and Explaining Away. In particular, they make a case for mechanisms based on

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Synchrony, while questioning the cases for Facilitation and Sharpening. However, it is important to note that these mechanisms are not mutually exclusive. For example, it is possible that predictive coding is a general property of the brain (Friston, 2012; Ewbank & Henson, 2012), and that the associated *explaining away* of stimulus-driven activity is achieved by *synchronous* activity between hierarchical brain regions, such that repetition causes *sharper* (sparser) spatial patterns of activity, and a *facilitation* (acceleration) of the dynamics of that activity. So below, I caution against the premature dismissal of Facilitation.

From a dynamical perspective, the brain's response to an external perturbation (stimulus) is likely to entail a period of higher energy (activity) that lasts several hundred milliseconds until a new, stable state of lower energy is reached (an attractor). As in many recurrent neural network models, this state-change is likely to trigger synaptic change, so as to widen/deepen the basin of attraction. When that stimulus is repeated therefore, there will be a faster settling (stabilization) of the network dynamics, i.e., a shorter duration of above-baseline neural activity (possibly despite negligible change in the onset of that activity). A shorter duration of neural activity will reduce the magnitude of response recorded by hemodynamic methods like fMRI that integrate over seconds of activity (i.e., cause *repetition suppression*; Henson, 2003).

The tension that Gotts et al. observe between faster behavioral responses (repetition priming) and reduced neural activity does not apply to Facilitation models, because both are the consequence of accelerated neural processing. However Facilitation is not really a mechanism, but rather a description of what happens at the neural level (to produce a reduced response at the hemodynamic level). Nonetheless, it remains distinct from the other mechanisms considered, in that Facilitation could occur with, or without, any concomitant change in Sharpness, Synchrony or Explaining Away.

Gotts et al. dismiss Facilitation models because of a lack of direct electrophysiological evidence. However, such evidence may be abundant in human EEG/MEG studies; just rarely conceptualized as such. Figure 1, for example, shows that the ERP to the repeated presentation of a face can be parsimoniously described as an accelerated version of the ERP to its initial presentation. Though such extracranial ERPs could originate from multiple neural sources (as Gotts et al. warn), it is unclear how this multiple determinacy would produce such a simple

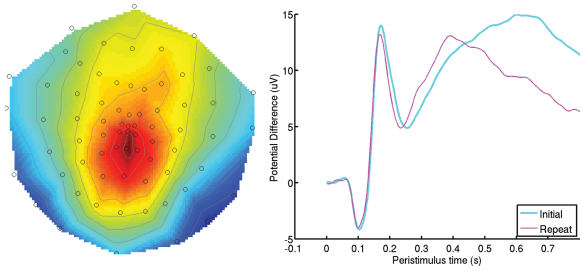

Figure 1. EEG data recorded from 70 electrodes (Henson, Wakeman, LItvak, & Friston, 2011) show that the ERP to the immediate (after ~3 seconds) yet unpredictable repetition of a face (magenta) is accelerated relative to that for its initial presentation (cyan). The topography (left; nose upward) and timecourse (right) are the first, dominant spatial and temporal components of a singular-value decomposition (SVD) of the (temporally-concatenated) trial-averaged ERPs, averaged over 18 participants. The scaling (zoom) of the time-axis for the temporal component of the initial presentation was systematically varied to minimize the RMSE between it and that for the repeat presentation. The mean acceleration factor was 92%, which was significantly less than 100% across participants, $t(17) = 3.18$, $p < .01$ (two-tailed).

temporal scaling. Since EEG/MEG data relate directly to LFPs from a population of neurons, the puzzle, as Gotts et al. observe, is why this apparent acceleration has not been observed at the level of spiking rates.

Looking forward, I fully support Gotts et al.'s proposals for future research, which can be divided into better data and better modeling. In addition to concurrent recording of local field and action potentials, to address the puzzle above, better data will come from recording from neurons in different layers of cortex, to relate to specific predictive coding models (e.g., Friston, 2008), and to establish which of these neurons contribute to M/EEG and fMRI signals. Data with high temporal resolution (such as M/EEG) is critical to test for dynamical changes over the few hundred milliseconds post-stimulus onset, for example, in terms of within- and/or across-frequency changes in power and/or phase of oscillations. In terms of better models, computational instantiations of some the above ideas are vital (e.g., the important work of Gotts, 2003), to relate both spatial (e.g., sharpness) and temporal (e.g., synchrony) dimensions of data, and to relate single-neuron data to population responses like fMRI; particularly, as noted above, if those ideas are not mutually exclusive and all turn out to reflect aspects of reality.

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