Supplemental Modeling

To confirm the expected profile of the hemodynamic response in pLPFC for the key experimental manipulations of this study, we generated simulation models. As several studies have found that the BOLD response to short periods of neural activity (< 3 secs) shows non-linear dynamics (Birn et al., 2001; Friston et al., 2000; Huettel and McCarthy, 2001; Liu and Gao, 2000; Miezin et al., 2000; Robson et al., 1998), we employed a non-linear model in our simulations. For a given condition, we first created a boxcar function representing the hypothesized neural activity in that condition, based on the expected duration of neural activity in pLPFC (since the exact duration of pLPFC activity is unknown, we used for our simulations a duration of neural activity that varied between Task RT and Task RT – 500ms; the simulation results were robust across the range tested). We then created a gamma variate impulse response function (IRF) by interpolating between the empirically derived impulse functions in Liu & Gao (2000). Finally, we convolved the boxcar function with the IRF, a process that produced the predicted BOLD response for the condition in question.

The first simulation assessed the differential effects of increasing response selection time to Task1 on hemodynamic activity at the short and long dual-task SOAs.
At the short SOA, the Slow Task1 RT condition leads to a delay in peak latency relative to the Fast Task1 RT condition (Supplemental Fig. 1A). At the long SOA, however, there is virtually no difference in peak latency between the Fast Task1 RT and Slow Task1 RT conditions (Supplemental Fig. 1B). Thus, increasing the duration of Task1 response selection does not significantly prolong the hemodynamic response if that increase is absorbed during the ‘slack’ period between RS1 (response selection for Task 1) and RS2 at the long SOA (see Fig. 1A).

The second simulation examined the effect of increasing response selection duration on the peak and onset latency of the hemodynamic response in the Single-Task condition. The results indicate that Slow RTs lead to a delay in peak latency, but not onset latency, of the response (Supplemental Fig. 1C).

The final simulation compared the hemodynamic responses expected under single-task and dual-task conditions. If one assumes a strict serial processing model, there is a peak latency difference between single-task and dual-task conditions (Supplemental Fig. 1D). By contrast a strict parallel processing model in which both tasks can be executed at once shows only a difference in amplitude – but not in peak latency.

Importantly, these simulation results held regardless of whether neural activity was modeled as a boxcar or ramp function. In addition, a linear model, in which the boxcar function of hypothesized neural activity is convolved with a canonical hemodynamic response function (as implemented in SPM2, http://www.fil.ion.ucl.ac.uk/spm), produced the same key predictions about the direction and general magnitude of the peak latency differences as the non-linear model.
Supplemental Figure

Figure S1. Nonlinear Model Results

The boxcar function - representing response selection stages to Task1 (RS1) and Task2 (RS2) - that was used to generate each curve is shown in the lower left corner of each panel. Arrows indicate the peak latency for each time course. A and B) Expected effect of slow and fast Task1 RTs on pLPFC activity at the short and long SOA in the Dual-Task experiment. A) Short SOA. B) Long SOA. C) Expected effect of slow and fast Task RTs on pLPFC activity in the Single-Task experiment. D) Comparison of the expected hemodynamic responses under Single-Task and Dual-Task (short SOA) conditions. A
serial model is contrasted with a parallel model in the Dual-Task condition. Each curve presented is the average of the separate curves that were generated for each RT tertile. Note that only peak latency, and not peak amplitude, is diagnostic of a change in duration of neural activity.

**Supplemental References**


