

Supplemental Data

Posterior Parietal Cortex Encodes

Autonomously Selected Motor Plans

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Figure S1. Neural activity of single LIP (top) and PRR (bottom) cells during trials in which the monkey was instructed to make a saccade (red) and reach (green). Spike trains were aligned to the cue onset. The peri-stimulus time histograms (PSTH) were smoothed using a Gaussian kernel (s.d. = 50 ms) and its thickness represents the standard error (+/- s.e.m.) calculated with the bootstrap method.

Figure S2. Comparison of activity between saccade and reach delay-instruted trials for the entire population of LIP (left panels) and PRR (right panels) cells. *A* to *D* represent four consecutive time intervals: cue duration (0-0.6 second after cue onset), early delay (0-0.3 second after cue off), late delay (0.3-0.6 second after cue off) and post-GO (0-0.1 second after GO signal - central fixation off). Similar to effector choice trials, both LIP and PRR showed similar activity during the ambiguous cue period ($p > 0.1$ for both LIP and PRR). After the effector was specified, most PRR cells fired stronger if the monkeys were cued to reach, similar to the effector choice trials ($p < 10^{-11}$ for all three period). However, LIP activity separated in a different way: some cells fired stronger if a saccade was cued whereas others fired stronger if reach was cued ($p > 0.05$, 0.5 and 0.9 for early

delay, late delay and post-GO period, respectively). Statistical significance (p -values) was measured with a two-tailed Wilcoxon signed rank test.

Figure S3. Distribution of choice probabilities (CPs) in effector delay-instructed trials and their time course. Data from 100 LIP and 91 PRR neurons are shown in *A* and *B*, respectively. The filled bars correspond to cells whose choice probability was significantly different from 0.5 measured by a permutation test. The triangle marker indicates the mean choice probability for each population: 0.51 for LIP cells (not significantly from 0.5, $p > 0.5$): 0.28 for PRR cells (significantly less than 0.5, $p < 10^{-20}$, two-tailed t -test). There was a weak correlation between CPs in instructed and choice trials for both LIP ($r = 0.21, p = 0.04$, Spearman rank correlation) and PRR ($r = 0.17, p = 0.11$) cells. Panel C shows the time course of the mean CP (line) in effector delay-instructed trials and its 95% confidence interval (shadow) calculated by ROC analysis with a 200 ms time window sliding with 20 ms steps.

Figure S4. Population histograms averaged across all isolated LIP (top) and PRR (bottom) neurons during saccade (red) and reach (green) chosen trials. The vertical thin lines indicate cue on, cue off and central fixation off (GO signal), respectively. The horizontal thin line indicates baseline activity, which was defined by mean firing rate during the 300 ms interval beginning from 500 ms before cue onset for both saccade and reach chosen trials. For LIP, averaged activity across the population was virtually same whether a saccade or reach was cued, because the opposite differential activity was averaged out (Figure S2 and S3). In contrast, PRR exhibited stronger population activity

for trials in which the monkeys were cued a reach rather than saccade, although post-GO population activity for both LIP and PRR was also significantly higher than baseline ($p < 10^{-4}$).

Figure S1

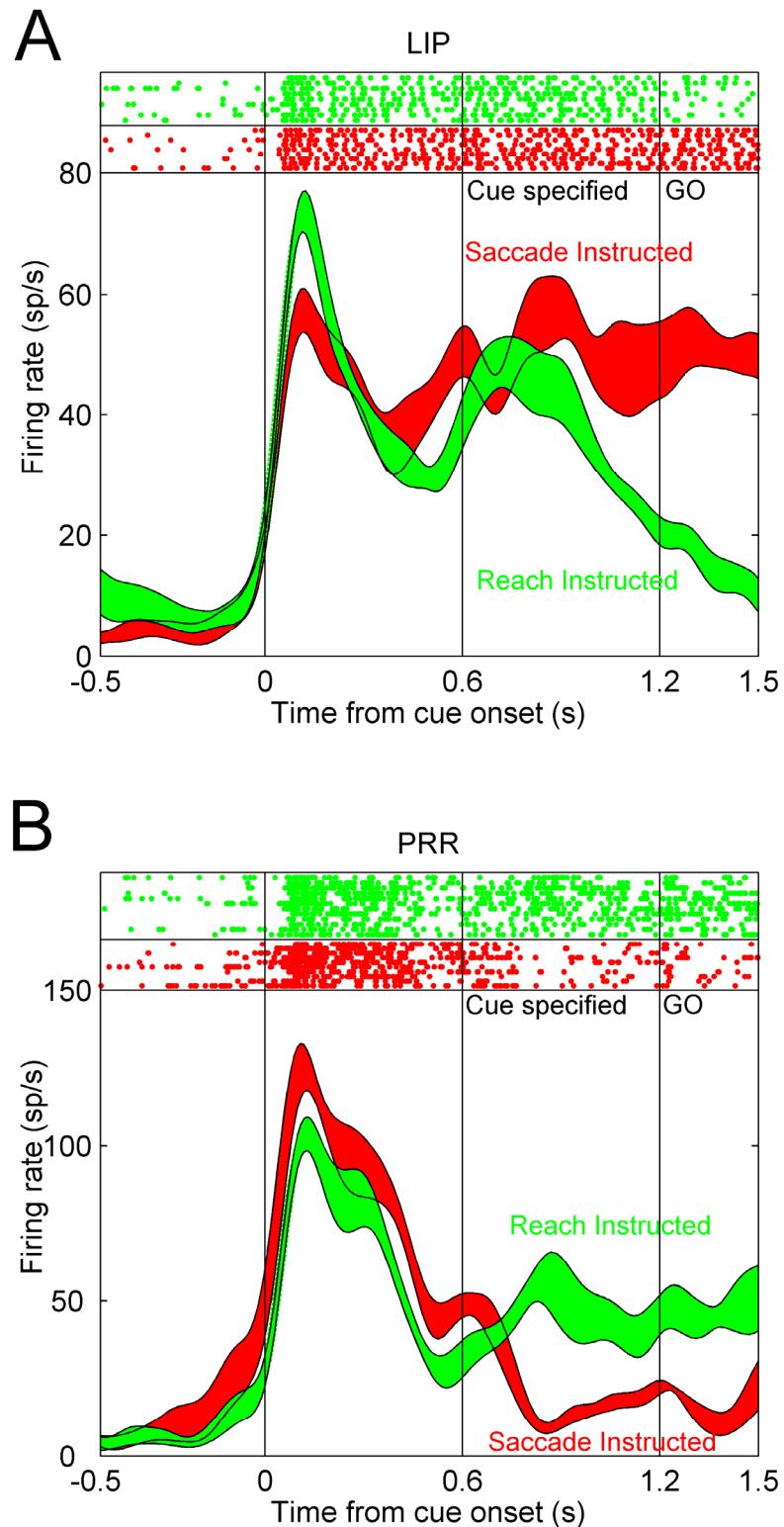


Figure S2

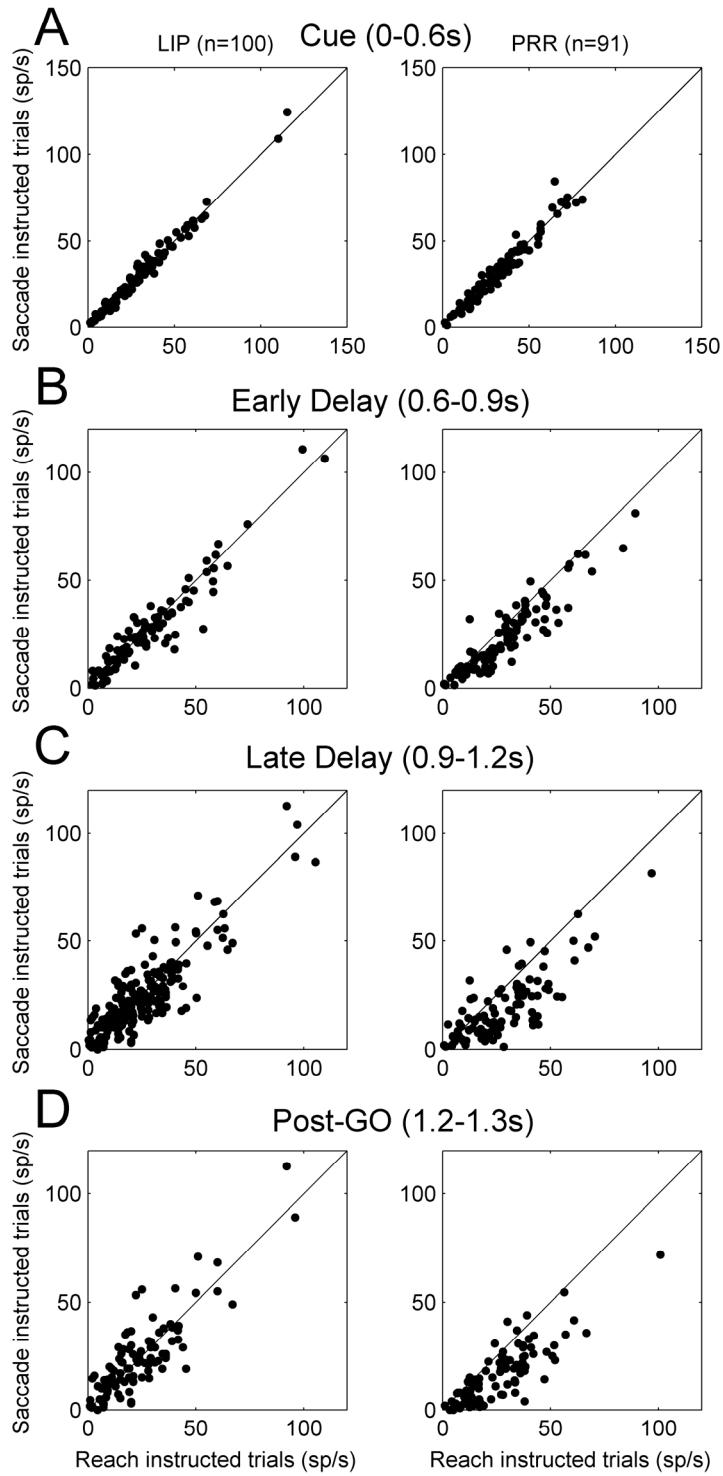


Figure S3

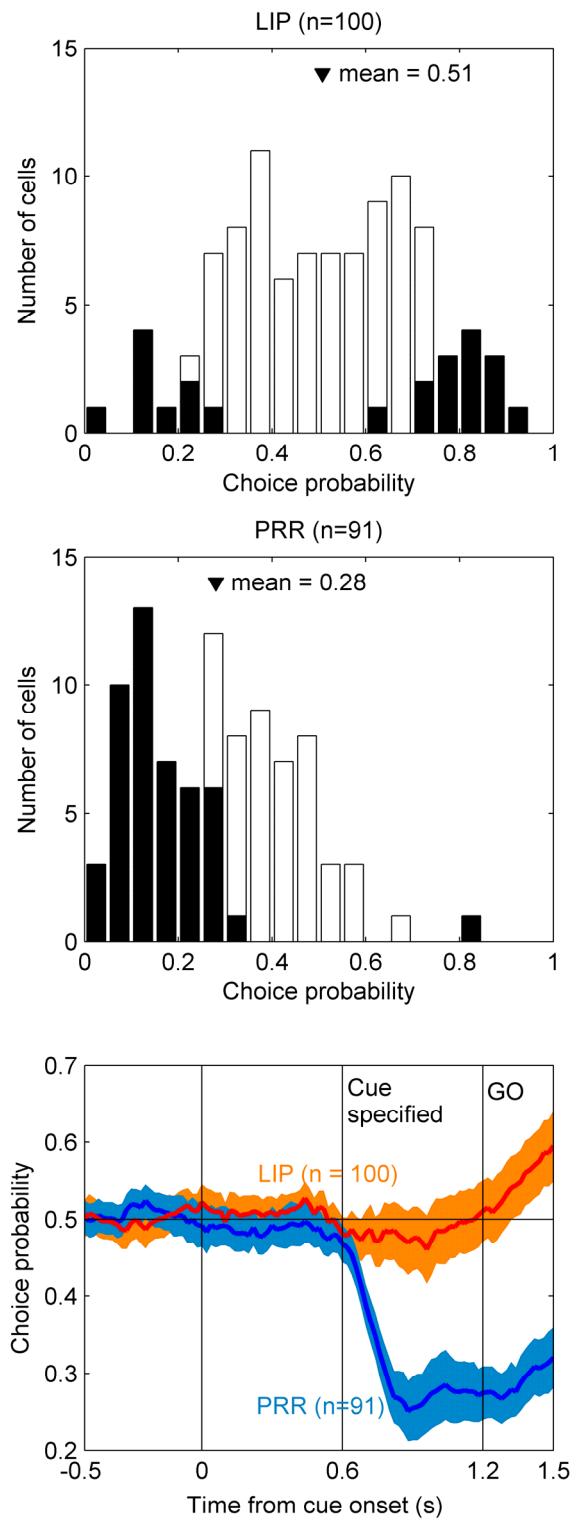


Figure S4

