

Comment on “In Monkeys Making Value-Based Decisions, LIP Neurons Encode Cue Salience and Not Action Value”

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Leathers and Olson (Reports, 5 October 2012, p. 132) draw the strong conclusion that neurons in the monkey lateral intraparietal (LIP) cortical area encode only cue salience, and not action value, during value-based decision-making. Although their findings regarding cue salience are interesting, their broader conclusions are problematic because (i) their primary conclusion is based on responses observed during a brief interval at the beginning of behavioral trials but is extended to all subsequent temporal epochs and (ii) the authors failed to replicate basic hallmarks of LIP physiology observed in those subsequent temporal epochs by many laboratories.

Leathers and Olson (1) draw the strong and broad conclusion that neurons in the lateral intraparietal area (LIP) of the monkey cortex encode cue salience, and not action value, during value-based decision-making. We compliment the authors on a nicely conceived study. Their data clearly suggest that the LIP neurons they studied encode cue salience during a transient 250-msec interval immediately after cue onset. This finding, which extends previous observations (2), is important and worthy of future study.

We are troubled, however, by two aspects of the paper: (i) the authors' sweeping dismissal of action-value encoding during subsequent temporal epochs of behavior and (ii) the authors' failure to replicate several delay-period effects, including action value, that have been extensively documented in numerous studies of LIP from many laboratories.

Our laboratories and others have repeatedly observed action-value signals during delay periods subsequent to initial transient responses, an epoch only fleetingly addressed by Leathers and Olson. In some studies, the reward value of alternative actions was indicated by features of visual stimuli presented on individual trials (3–10), whereas in others the animals estimated the reward value of alternative actions from choice and reward histories over multiple trials (11–18). Leathers and Olson dismiss these reports because of their own observation of cue salience

signals during the initial transient responses in their task. Visual inspection of delay-period activity in their data (figures 2 and 3, 500- to 1500-msec interval, blue versus red curves) reveals no action-value signals where they should be present according to the many reports cited above.

It is possible that these previous reports are mistaken, having confused salience and value as suggested by Leathers and Olson. We are unconvinced, however, because basic aspects of the LIP data presented by the authors differ strikingly from the extensive LIP physiology literature. Most puzzling is the near-complete absence of robust delay-period activity, selective for the direction of the operant saccade, which has been a hallmark of LIP activity from the first published reports (19–21) onward (4, 6–8, 11–15, 17, 18, 22–27). In all of Leathers and Olson's data figures, average delay-period activity is suppressed below baseline firing rates, even when the animal is preparing a saccade toward the neuron's response field. In addition, the differential activity between preferred and nonpreferred saccades during the delay period is minimal (figure 1, D and E, 500- to 1500-msec interval, blue and red curves), amounting to only 1 to 2 spikes per second on average, which is 5 to 20 times below the difference typically reported in the literature. These data fail to replicate many previous reports. Quantitative studies (19, 23) have shown that a large fraction of LIP neurons (~30% and ~50%, respectively) exhibits robust delay-period activity, selective for the direction of the operant saccade, even during a simple remembered saccade task, which rules out one possible explanation for the difference between the authors' data and some previous reports (no target present in the response field versus target present, respectively). Future studies must address other potential explanations, including sampling biases (different subpopulations of LIP neurons) or differences in the visual cues used to indicate salience or value (e.g., motion versus form).

In the reports cited above, action-value signals in LIP typically comprise modulations of the differential activity levels associated with one or the other planned saccade. Given the paucity of differential, saccade-related delay-period activity in the authors' data, it is unsurprising that they failed to observe action-value signals: no “action,” no “action value.”

Despite our current skepticism, we are open to being convinced. We would be more intrigued, for example, had the authors been able to replicate the well-documented LIP action-value effects during the delay period of their task and then shown how cue salience at the beginning of the trial does, or does not, affect putative action-value signals during subsequent epochs. Given the current data, however, we see no basis for the authors' sweeping conclusion that their results are not compatible with “the idea that LIP neurons represent action value.”

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