

Lena Kästner

Where Does Your Brain Calculate Causal Effects of Your Actions?

In mPFC. — According to a recent study by Tanaka et al. [1], three different brain regions contribute to implementing goal directed behavior, whereof only mPFC is found to be sensitive to changes in local contingency.¹

In the following, I will shortly outline the experimental procedure, experimenters' motivation, their results and what these mean. Finally, I will discuss possible shortcomings and improvements of the present study.

What was done?

Using a 3T-fMRI, Tanaka et al. measured BOLD responses of fourteen healthy right-handed subjects (7 m, 7 f) during performance on a self-paced behavioral task. Each subjects performed four five-minute sessions. A session was composed of ten randomly ordered 30 s blocks: five RESPOND blocks—during which the subjects freely made as many button presses as they liked—and five REST blocks.

Four different event schedules were used in the RESPOND blocks; each subject performed one session of each. On *VR10* (variable ratio) schedules, subjects were rewarded with 25 cents every ten responses, on *VI4* (variable interval) schedules every four seconds on average. On *VR-yoked* and *VI-yoked* schedules, rewards were based on the preceding subject's performance on *VI4* and *VR10* schedules, respectively. At the end of each session, subjects rated the causal efficacy of their actions.

Why was it done?

The procedure sketched above was employed to assess the question of which neural substrates underlie the detection of contingency and subjective causality judgments in humans.

Evaluating the causal efficacy of actions is thought of as a key mechanism for adopting to a constantly changing environment. Former studies indicated contingency sensitivity in rats and rodents being mediated by mPFC and dorsomedial striatum.

What did they find?

Behavioral data revealed (1) a significant correlation (cf. [1], fig. 2) between subjective causality ratings and objective contingency values. Contrasting average evoked BOLD signals of high- and low-contingency schedules identified (2) three brain regions changing their activity as a function of objective contingency: mPFC, mOFC and dorsomedial striatum. However, only mPFC activation showed a significant correlation with local objective

¹The term *contingency* here describes the relationship between actions and their outcomes. It is defined as the difference between the probability of an outcome when an action is performed as compared to when it is not.

contingency as calculated within 10 s time intervals of performance on the task (cf. [1], fig. 3). Finally, (3) three brain regions (mPFC, lateral OFC, dorsomedial PFC) showed linearly increasing responses with higher causality ratings (cf. [1], fig. 4).

What does it mean?

Tanaka et al. take these findings to suggest that (1) subjects' sensitivity to changed relative contingency were reflected in their causality ratings, (2) on-line computation of contingency between responding and rewards involves mPFC, and (3) mPFC activation also tracks judgments of causal efficacy of subjects' behavior.

Therefore, goal-directed behavior involves different parts of PFC and striatum. mOFC and dorsomedial striatum might mediate control of behavior (since they were highly engaged in high-contingency sessions) while mPFC might be crucial for tracking the action-outcome correlation (since it showed high sensitivity to local contingency).

The brain structures found to be involved in calculating causal consequences in humans roughly match those identified in animal studies; this might suggest control for goal-directed action being preserved across species.

What's wrong/ left?

Now that Tanaka et al.'s study has been presented, let me give a few remarks. First, we miss detailed information about the subjects—e.g., age or educational status. Perceived causality might be influenced by either of these factors.

Second, yoked schedules might have produced artifacts. In a way, using data from one subject during the experiment with another, creates circularity: every subject's rewards depends upon her predecessor's performance.

Third, little is said about the stimuli. Even if they should not play a role at all, it is important to control for this factor.

Fourth, significant activations could be due to reward processing rather than estimating consequences or behavioral control. For instance, O'Doherty et al. report reward-related activity in human OFC [2]. Further, preceding losses and gains might influence the effects of rewards as well as perceived causality. Comparison of behavioral and BOLD changes during rewarded and non-rewarded button presses could be useful.

Finally, RESPOND blocks were relatively short and separated by only 30 s (if at all). This might not sufficiently respect the characteristic delay of fMRI signal. Possibly, Tanaka et al.'s results could be confirmed using techniques with higher temporal resolution (e.g. EEG or TMS).

References

- [1] Tanaka, S.C. et al. (2007). Calculating Consequences: Brain systems that encode the causal effects of actions. *Journal of Neuroscience*, 28(26), 6750-6755.
- [2] O'Doherty, J. et al. (2001). Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature Neuroscience*, 4(1), 95-102.