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Report

# Visual Adaptation of the Perception of Causality

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## Summary

**We easily recover the causal properties of visual events, enabling us to understand and predict changes in the physical world. We see a tennis racket hitting a ball and sense that it caused the ball to fly over the net; we may also have an eerie but equally compelling experience of causality if the streetlights turn on just as we slam our car's door. Both perceptual [1] and cognitive [2] processes have been proposed to explain these spontaneous inferences, but without decisive evidence one way or the other, the question remains wide open [3–8]. Here, we address this long-standing debate using visual adaptation—a powerful tool to uncover neural populations that specialize in the analysis of specific visual features [9–12]. After prolonged viewing of causal collision events called "launches" [1], subsequently viewed events were judged more often as noncausal. These negative aftereffects of exposure to collisions are spatially localized in retinotopic coordinates, the reference frame shared by the retina and visual cortex. They are not explained by adaptation to other stimulus features and reveal visual routines in retinotopic cortex that detect and adapt to cause and effect in simple collision stimuli.**

## Results

We often have a strong sense of causality as events unfold, where one event apparently triggers the next. The perception of causality involves two components, one that is stimulus based and one that is inference based. First, to see causal structure between two events, they need to follow each other with little delay, and in many cases, including collisions, they also require contact. This spatiotemporal coincidence is the stimulus-based component of perceptual causality. The second component is an inference, merging two events into one. Rather than seeing one object stopping and a second one starting on its own, there is a continuity of action that is transferred from the first object to the second. Using a visual adaptation paradigm, we tested whether this inference occurs on a perceptual level, which would allow experimental access to the visual detection of causality and the parsing of events at a perceptual stage.

To measure observers' perception of causality, we displayed short animations of two test events, appearing either above/below or to the left/right of a central fixation spot (Figure 1A). Each test event consisted of two gray discs that at times were stationary and at other times moved across the black background. Identical but mirror-symmetrical events appeared simultaneously on both sides of the screen to discourage reflexive eye movements. In each event, one disc was initially stationary as the other one approached on a direct path from a random direction. After 80 ms, when the two discs overlapped by some amount, the moving disc froze and the stationary one took off in the same direction at the same speed (Figure 1B; see also Movie S1 available online). Depending on the overlap between the two discs when the second disc starts moving (Figure 1C), these test events appear either causal or noncausal. In particular, when there is little overlap, one disc appears to causally launch the other's motion, whereas when there is large overlap, one disc appears to noncausally pass over (or under) the other, which in turn remains stationary [1, 13]. On every trial, observers pressed one of two buttons to indicate what they perceived, a launch or a pass. By fitting a psychometric function to each observer's data, we obtained the point of subjective equality (PSE), which captures the amount of disc overlap at which the observer is equally likely to report the event as a launch or a pass.

### Adaptation to Collision Events, Appearing Causal

In our first experiment, each observer performed two blocks of trials, and we measured two PSEs each time, one for events presented left/right of fixation and one for events above/below fixation. After the first block, in which we obtained baseline PSEs for both sets of locations, observers saw an "adapting stream" of 320 "collision" (or, launch [1]) stimuli (Figure 2A; Movie S2)—two discs bouncing back and forth, clearly appearing causal—while maintaining fixation at the screen center. For half of the observers we showed these adapting streams only at the horizontal locations, and for the other half only at the vertical locations. Adapting streams were organized in pairs, with the first stimulus in each pair having a random direction of motion and the second having the opposite direction. After this adaptation phase, we obtained a second set of PSEs, one at the adapted locations and one at the locations where no adapting streams had appeared. Adaptation (provided it occurred) was topped up by a further stream of 16 adapting stimuli preceding every test trial. The critical dependent variable was the change in PSE at either location. If prolonged exposure to phenomenologically causal stimuli shifts the PSE, we have evidence for adaptation.

Before adaptation, observers' PSEs were 0.63 ± 0.07 (mean ± SEM, where 1.0 indicates complete overlap) and 0.59 ± 0.08 for the (to be) adapted and unadapted location, respectively. Following adaptation, we observed a strong shift of the psychometric function to the left for test events

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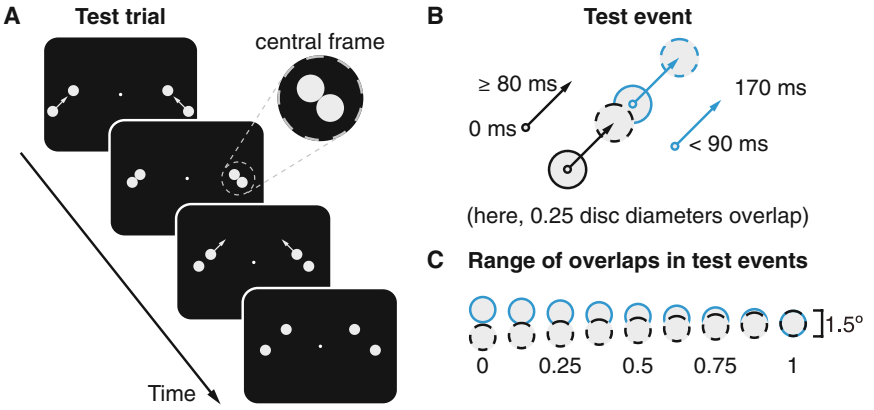


Figure 1. Procedure and Stimulus Design

(A) On every trial, two mirror-symmetrical test events appeared either to the left/right of (shown here) or above/below a central fixation spot.

(B) Test event. After 80 ms of motion, disc 1 (black) stops with some degree of overlap (here, 0.25 diameters) with disc 2 (blue). Disc 2 then moves off in the same direction and at the same speed.

(C) Test events had one of nine magnitudes of overlap in their central frame.

presented at the adapted location (PSE = 0.34 ± 0.06; Figure 2B, blue versus black curve). Events that were perceptually ambiguous before adaptation (e.g., overlap of 0.625) were now judged to be noncausal passes in the vast majority of trials; events that were regularly perceived as causal before adaptation (e.g., overlap of 0.375) had now become ambiguous. Perception of test events at the unadapted location was less affected (PSE = 0.51 ± 0.06; Figure 2B, gray versus black curve). We captured these effects by plotting the individual changes in PSE at the adapted location against those at the unadapted location (Figure 2C). Every observer's data point fell below the x axis, showing that the PSE decreased at the adapted location ($\Delta PSE_{adapted} = -0.27 \pm 0.05$, $p < 10^{-9}$, Bayes factor [BF] > $10^3$), resulting in a substantial adaptation effect (blue marker). We also observed a small adaptation effect at the unadapted location ($\Delta PSE_{unadapted} = -0.08 \pm 0.03$, $p < 0.01$, BF = 0.42). Importantly, all data points fell into the blue-shaded area, showing that the decrease in PSE was larger at the adapted than at the unadapted location ($\Delta PSE_{adapted} - \Delta PSE_{unadapted} = -0.19 \pm 0.03$, $p < 10^{-10}$, BF > $10^4$). These results therefore uncover pronounced and spatially specific negative aftereffects of prolonged exposure to perceptually causal collision stimuli.

### Adaptation to Slip Events, Appearing Noncausal

In a second step, we examined whether adaptation to other visual features of the adapting stimuli might explain the change in observers' judgments of causality. To do so, we repeated the first experiment using a "slip" adaptation stimulus (Figure 3A; Movie S3), designed to match the collision adaptation stimulus (Figure 2A) in as many physical properties as possible—the number and appearance of the discs, spatiotemporal contiguity, timing and number of motion onsets, motion direction and speed, as well as the area covered by the event—without leading to perceived causality. In a slip adaptation stimulus, rather than stopping when it touches the stationary one, the moving disc moves completely across and comes to stop immediately on the other side. With no delay, the initially stationary disc then starts off in the same direction, leaving the impression of two independent motions.

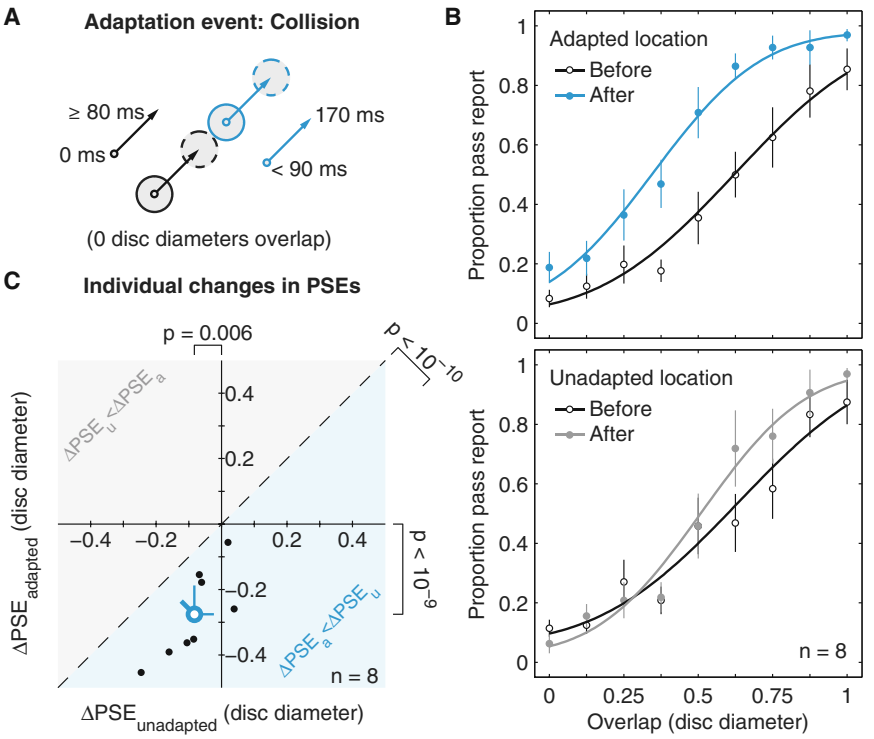


Figure 2. Adaptation to Causal Collisions

(A) Collision adaptation stimulus. After 80 ms of motion, disc 1 (black) stops next to disc 2 (blue), which then moves off.

(B) Average proportion of pass reports with cumulative Gaussian psychometric functions modeling the relationship between disc overlap and perceptual reports. Error bars are SEM.

(C) Individual (black dots) and average (blue marker) changes in point of subjective equality (PSE) for the adapted and unadapted test locations. Error bars are 95% confidence intervals (thick bar: comparison against line of equality); p values are bootstrapped tests against zero. In the blue-shaded area, the decrease in ΔPSEs is larger at the adapted than at the unadapted location.

See also Figure S1.

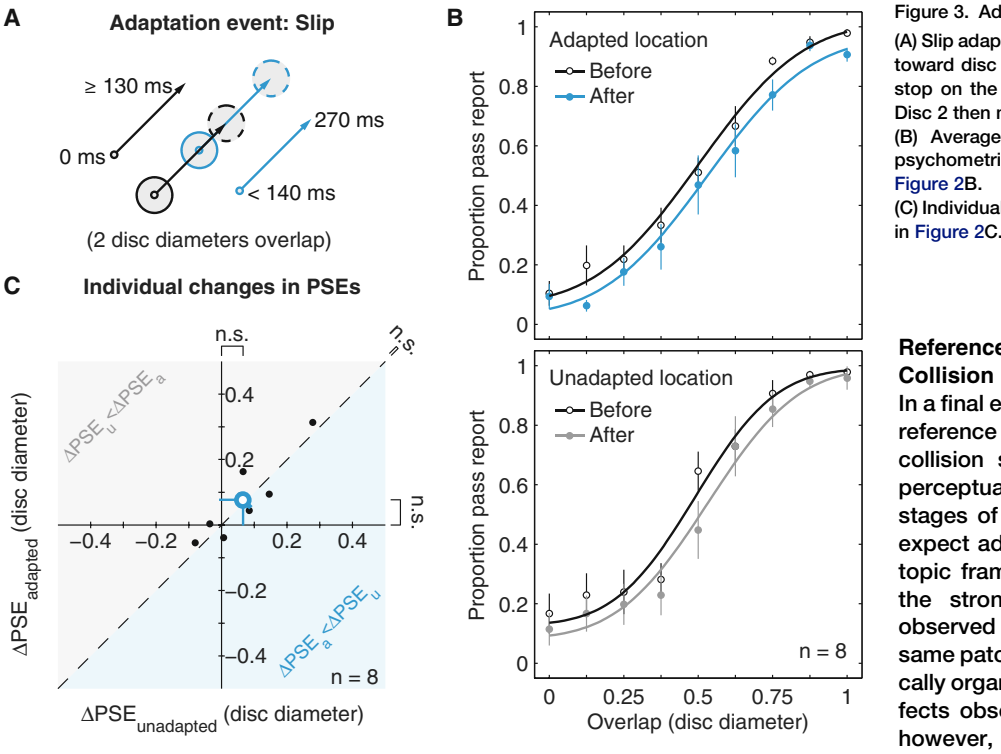


Figure 3. Adaptation to Noncausal Slips

(A) Slip adaptation stimulus. Disc 1 (black) moves toward disc 2 (blue), slips over it, and comes to stop on the other side after 130 ms of motion. Disc 2 then moves off.

(B) Average proportion of pass reports with psychometric functions. Conventions are as in Figure 2B.

(C) Individual changes in PSE. Conventions are as in Figure 2C.

Note that slip and collision adaptation stimuli both feature spatial as well as temporal coincidence; specifically, one object stops next to the other just as the other takes off. However, one appears causal and the other does not.

Contrary to the effects of adaptation to collisions, adaptation to slip stimuli had little or no effect on observers' perceptual reports. Before adaptation, we again observed the increase in the proportion of pass reports with increasing disc overlap of the test stimuli (Figure 3B, black), with PSEs of 0.45 ± 0.03 and 0.41 ± 0.05 for the (to be) adapted and unadapted location, respectively. On average, psychometric functions shifted slightly to the right both for the adapted (Figure 3B, blue; PSE: 0.53 ± 0.05; $\Delta PSE_{adapted}$ = 0.08 ± 0.04, p = 0.052, BF = 0.19) and the unadapted location (Figure 3B, gray; PSE: 0.47 ± 0.06; $\Delta PSE_{unadapted}$ = 0.07 ± 0.04, p = 0.076, BF = 0.15), with no difference between them ($\Delta PSE_{adapted} - \Delta PSE_{unadapted}$ = 0.01 ± 0.02, p = 0.52, BF < $10^{-1}$). Individual changes in PSEs are shown in Figure 3C. A direct statistical comparison of these results to those from experiment 1 again established the spatial specificity of aftereffects following adaptation to collision versus adaptation to slip events (−0.20 ± 0.03, p < $10^{-8}$, BF > $10^{3}$). We conclude that the perceptual changes after exposure to streams of collisions were not caused by adaptation to any of the visual attributes shared by collision and slip stimuli. Instead, adaptation affected visual processing of causal structure, present only in our collision stimuli.

An alternative explanation of the effect of adaptation is that it did not affect perceived causality directly but instead changed the perceived timing of the individual events, which in turn reduced perceived causality [1, 14–17]. Our collision and slip stimuli were constructed to equate the timing of the two discs' motions, but to be sure, we ran a second control experiment to test potential effects of timing directly (see Supplemental Results and Figure S1). These supplemental results showed that we had adapted detectors of causality for this type of stimuli, not detectors of timing.

## Reference Frame of Adaptation to Collision Events

In a final experiment, we determined the reference frame of adaptation to causal collision stimuli. If this adaptation of perceptual causality occurs at early stages of visual processing, we would expect adaptation to occur in a retinotopic frame of reference. In that case, the strongest aftereffects would be observed at locations falling on the same patch of the retina (and retinotopically organized brain areas). The aftereffects observed in the first experiment, however, are equally compatible with changes of perception tagged to a location in the world, possibly indicating an association of the experience with the adaptation stimulus with a location in external space. In that case, the strongest aftereffects would be observed at a fixed location in space, irrespective of the retinal location of the test. Identifying the reference frame of adaptation therefore requires changes in gaze position between adaptation and test [18].

We presented stimuli on one side of fixation and monitored eye position, ensuring that stimuli occurred at the intended retinal location (Figure S2). Observers made two fixation steps before a test event appeared—first away from initial fixation, then either back to fixation or to the opposite location (Figure 4A). Two initial blocks of test trials preceded the adaptation phase (another two blocks) where an adaptation stream of 320 collision stimuli appeared before the first and another 16 appeared before every subsequent test trial. Depending on (1) the correspondence of the fixation location during adaptation and test stimulus presentation and (2) the location of the test event relative to fixation, the test fell onto the same retinal but different spatial location (retinotopic aftereffect; green in Figure 4A), the same spatial but different retinal location (spatiotopic; beige), the same spatial and retinal location (full; blue), or an eccentricity-matched unadapted location (nonspecific; gray).

Data from four participants showed an aftereffect only if test events coincided with the adaptation stream in retinal coordinates, i.e., in the retinotopic and full aftereffect conditions (Figure 4B; PSEs at nonspecific location: 0.64 ± 0.02 before versus 0.64 ± 0.02 after adaptation, BF < $10^{-1}$; full: 0.63 ± 0.03 versus 0.48 ± 0.03, BF > $10^{12}$; spatiotopic: 0.67 ± 0.05 versus 0.65 ± 0.04, BF < $10^{-1}$; retinotopic: 0.64 ± 0.04 versus 0.46 ± 0.03, BF > $10^{2}$). Accordingly, an ANOVA with test phase (before versus after adaptation), retinal test location (adapted versus unadapted), and fixation position (matched versus unmatched between adaptation and test) as within-subject factors yielded significant effects of retinal test location

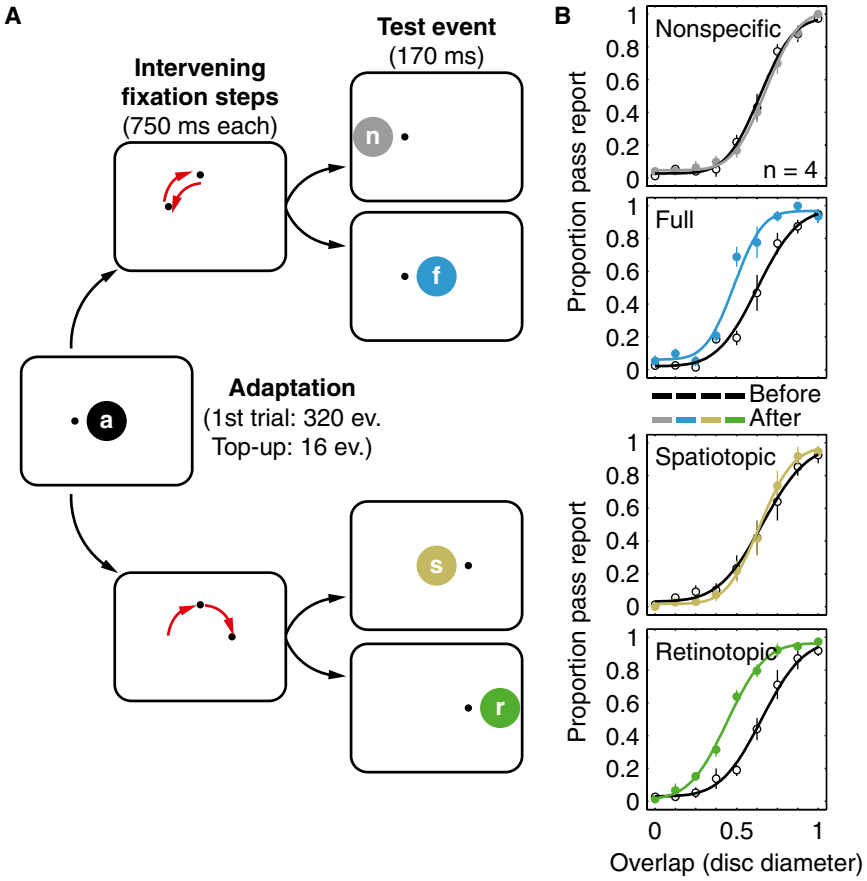


Figure 4. Reference Frame of Adaptation to Collisions

(A) Observers fixated a spot 5° to the left or to the right of the screen center. Following the adaptation stream at the screen center, observers made two fixation steps (red arrows). Subsequently, a test event occurred at 5° eccentricity, either to the left or to the right of fixation, yielding a total of four possible test locations: nonspecific (gray), full aftereffect (blue), spatiotopic (beige), and retinotopic (green).

(B) Average proportion of pass reports with psychometric functions. Conventions are as in Figure 2B.

See also Figure S2.

[F(1,3) = 219.21, p < 0.001] and test phase [F(1,3) = 64.33, p = 0.004] as well as an interaction of these two factors [F(1,3) = 67.53, p = 0.004; other Fs < 1.8, p values > 0.27].

## Discussion

The retinal specificity of the observed aftereffects argues strongly that it is a consequence of a shift in a perceptual, not a cognitive, boundary between causal and noncausal events. Previous experiments have shown that repeated exposure to or training in the categorization of causal stimuli alters the frequency of causal reports, but these shifts have been interpreted as cognitive anchoring effects [19, 20]. Cognitive boundary shifts are common and may even be contingent on location in the world—what looks like steam over a pot will look like smoke over a chimney. Never, however, will cognitive boundary shifts be specific to a particular location on our retina, independent of location in the world. Nevertheless, this retinal specificity, on its own, is not sufficient evidence that causal processing occurs at the perceptual level. The inference of causality is the end result of a chain of analyses, and the adaptation of visual signals at early levels, prior to the determination of causality, could certainly produce a location-specific effect. This would be true even if the final decision stage was cognitive and nonretinotopic. Our conclusions therefore rest on our combined results of retinotopically specific adaptation and the absence of adaptation to slip events that were matched to the collision events in low-level visual signals.

Visual adaptation demonstrates the perceptual consequences of a reduction in the responsiveness of neural populations that encode primary visual features [10, 12]. Using this general paradigm, we provided support for the existence of adaptable, visual neurons (or neural populations) that underlie the perception of at least one causal interaction in dynamic scenes. Stimuli that do not appear causal (including our "slip" adaptation stimuli) leave the responses of these neurons unaffected. These neuronal populations must be located in brain areas that encode visual information in an eye-centered reference frame, because the resulting aftereffects are specific to the adapted location on the retina. Candidates for such areas are the mediotemporal area V5 and the superior temporal sulcus, both of which have eye-centered representations [21] and are part of a network involved in the perception of causal launches [22–25]. These areas also respond to other forms of meaningful motion patterns, such as biological motion [26, 27]. Using adaptation, we can now examine the visual computations underlying the perception of causal structure in the visual world. These include not only the routines recognizing familiar motion patterns [28] but also complex interactions involving cause and effect, possibly even animacy and intentionality.

We have focused on one specific causal stimulus (collisions, or launches) and have shown that it induces adaptation. It is not yet known whether these aftereffects generalize to other types of causal stimuli, but our finding takes a more important step—it isolates a visual process that merges two events into a single percept, thus parsing the continuity of action in the visual scene. This finding allows us to move phenomena that have been regarded as higher-level processes into the realm of perception, opening them to systematic study using the tools of perceptual science. In a similar sense, the discovery of amodal completion allowed us to study the integration of object structure behind an occluding surface, and the discovery of apparent motion allowed us to study the merging of two object identities at different locations and times into the motion of a single one. Both of these percepts require sophisticated inference, and it is now widely agreed that perception is the locus of these advanced decisional processes. This has led to pathbreaking studies of their neural correlates [29–34]. The present findings take an equally important step toward determining how the brain parses events and assigns causal links, which paves the way for tracking down the neural mechanisms underlying these visual processes.

### Supplemental Information

Supplemental Information includes Supplemental Results, two figures, Supplemental Experimental Procedures, and three movies and can be found with this article online at http://dx.doi.org/10.1016/j.cub.2012.12.017.

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