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Research report

Brain mechanisms underlying perceptual causality

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Abstract

Functional magnetic resonance imaging (fMRI) was used to examine the neural correlates of perceptual causality. Participants were imaged while viewing alternating blocks of causal events in which a ball collides with, and causes movement of another ball, versus non-causal events in which a spatial or a temporal gap precedes the movement of a second ball. There were significantly higher levels of relative activation in the right middle frontal gyrus and the right inferior parietal lobule for causal relative to non-causal events. Furthermore, when the differential effects of spatial and temporal incontiguities were subtracted from the contiguous stimuli, we observed both common (right prefrontal) and unique (right parietal and right temporal) regions of activation as a function of spatial and temporal processing of contiguity, respectively. Taken together, these data provide a means to help determine how the visual system extracts causality from dynamic visual information in the environment using spatial and temporal cues.

Theme: Neural basis of cognition
Topic: Cognition

Keywords: Causal; Thinking; Spatial; Temporal; Contiguity; fMRI

1. Introduction

An understanding of the causal relations mediating moving objects is essential for making sense of the world in which we live. Much research has demonstrated that causal links are often induced from observations of simple object interactions. For example, if an object moves toward a second object, stops when it is adjacent to the second object, and then the second object moves away, the motion of the second object is reported by the majority of observers to have been caused by the first. This has been termed the launching effect and is perhaps the best-known example of what is called perceptual causality [32,36,37]. The findings that the perception of causality appears very early in human life [31] and is culturally invariant [33] have been taken to suggest that the visual system may be specially tuned to recover causal structure from the environment.

There are a number of cues that have been demonstrated to be used by people when deciding if an action and an effect are causally related. These include covariation [5,26,34,42], temporal order [38,45], contiguity in time and space [4,32], mechanism information [1,17,18,19,22,47,48], and similarity between cause and effect [40,44]. Two cues to causality that have received extensive investigation with respect to perceptual causality are spatial and temporal contiguity. Parametric manipulations of spatial gaps and temporal delays between two stimulus movements have been shown to reduce the likelihood with which stimulus interactions are rated as causal in a parametric fashion [32,35]. That is, the larger the temporal delay or the spatial gap that precedes the second of two stimulus movements, the greater the likelihood that the relationship between the two movements will be judged non-causal. The extent to which different cues to

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causality are supported by unique or common representations is a matter of recent debate [37]. By examining the extent to which such representations are neurally dissociable, we provide insight into this fundamental question.

1.1. Examining the neuroanatomical correlates of causal perception

We [35] have recently found that the perception of causality when contrasted with stimuli that possess temporal and spatial violations of causality was supported by neural tissue lateralized in the right hemisphere of two split-brain patients. Specifically, only the right hemispheres of the two split-brain patients were able to discriminate causal from non-causal events while viewing simple collision displays. The extent to which processes supporting the perception of causality following collision displays are lateralized in the normal brain with an intact corpus callosum are relatively unknown. Recent work by Blakemore et al. [3] and Fonlupt [12] has supported the role of bilateral networks in the perception of causality. Specifically, by contrasting causal (blue ball *collided* with a red ball which subsequently moved) as opposed to non-causal events (blue ball moved across the screen and passed *under* a stationary red ball), Blakemore et al. [3] and Fonlupt [12] found significant activations in V5, medial, and superior temporal lobes bilaterally, as well as regions in the left superior temporal and intraparietal sulcus. As these regions are strongly implicated in tasks involving complex visual analyses, they argued that the visual system is specifically designed to recover the causal structure of dynamic visual events.

In the current study, we examine the extent to which causal stimuli differentially recruit neural tissue as a function of spatial and temporal processing of contiguity. Do temporal and spatial processing of contiguity recruit unique or common underlying neural tissue? One possibility is that temporal processing of contiguity selectively recruits neural tissue associated with rapid temporal processing whereas spatial processing of contiguity selectively recruits neural tissue associated with spatial perception. Support for this hypothesis would be provided if temporal and spatial processing of contiguity recruits regions in the superior temporal [52] and parietal cortices [11], respectively. An alternative hypothesis is that the processing of both sources of contiguity will recruit common neural tissue associated with the perception of motion. Here, one would expect that both spatial and temporal processing of contiguity would recruit regions in the medial temporal, parietal, and occipital (i.e., V5) cortices; areas commonly associated with the perception of motion [46].

The current study also affords us the opportunity to examine the degree to which contiguous versus non-contiguous stimuli selectively recruit lateralized or bilateral neural tissue when the causal and non-causal displays are closely equated for visual complexity. The non-causal stimuli used in the current experiment always involve the motion of two moving objects varying only in terms of previously identified violations of causality [32]. Beyond a test of lateralized processing, the use of whole brain functional imaging affords us the opportunity to examine more localized recruitment of specialized neural tissue. Does the perception of causality simply involve the detection of contiguous movement, or do causal stimuli recruit neural tissue involved with higher-order cognition, such as attention and executive processing? Recent work by Fugelsang and Dunbar [20] found that regions in the inferior and superior frontal cortices were selectively recruited when participants reasoned about stimuli that possessed a plausible as opposed to an implausible causal mechanism. The extent to which simple perceptual stimuli, like those utilized in the current study, recruit neural tissue associated with attention and executive processing may provide support for a more domain general attentional basis of causality. Evidence for the latter hypothesis would be found if causal stimuli recruited additional neural tissue in the prefrontal cortex [9,20,21,41].

2. Materials and methods

2.1. Participants

Sixteen participants (7 females and 9 males, mean age = 26.8 years) participated in the study and were paid $10. All participants were right-handed, reported no significant abnormal neurological history, and had normal or corrected-to-normal visual acuity. Informed written consent for all participants was obtained prior to the experiment in accordance with the guidelines established by the Committee for the Protection of Human Subjects at Dartmouth College.

2.2. Design and apparatus

A standard block design was used with alternating blocks of 30 s of task versus 30 s of fixation only rest. Visual stimuli were generated using a G4 PowerBook computer running PsyScope 2.5.1 software [6]. Stimuli were projected to participants with an Epson (model ELP-7000) LCD projector onto a screen positioned at the head end of the bore. Participants viewed the screen through a mirror. Cushions were used to minimize head movement.

2.3. Materials and procedure

During the scanning session, participants viewed three types of movies: (1) CAUSAL, (2) TEMPORAL Gap, and (3) SPATIAL Gap. Each stimulus was made up of a 12 cm by 6 cm rectangular display subtending approximately 7.2° of visual angle. Each event lasted 2 s and was updated at 60 frames per second for a total of 120 frames per movie. The events moved either from left to right or from right to left. Each 30-s block consisted of 12 movies separated by a 500-ms intertrial interval. The CAUSAL, TEMPORAL gap, and SPATIAL gap movies were blocked such that each participant saw 12 repetitions of each type followed by 30 s of

fixation only rest. Each event type block was presented twice, once where the balls moved from right to left, and once where the balls moved from left to right for a total of 6 blocks per run. Each participant completed 4 runs with all 6 conditions randomized throughout each run. Participants were simply asked to respond to the direction of the movement using button presses on specially constructed fiber-optic button boxes during the scanning session. This was done by pressing a button in their left hand if the balls moved from right to left, and a button in their right hand if the balls moved from left to right.

For the CAUSAL events, two white balls 1.2 cm in diameter appeared motionless for 30 frames (500 ms), after which the first white ball rolled horizontally across the screen for 20 frames (5 cm; 330 ms) and collided with a second white ball whose leading edge was positioned in the center of the screen (at 5 cm) in the path of the first ball. Immediately after the first ball made contact with the second ball, the second ball moved horizontally in the same direction for 40 frames (5 cm; 660 ms) and then stopped at the edge of the screen and lay motionless for 30 frames (500 ms). For the TEMPORAL gap events, the two white balls appeared motionless for 20 frames (330 ms), after which the first white ball rolled horizontally across the screen for 20 frames (5 cm; 330 ms) and collided with a second white ball whose leading edge was positioned in the center of the screen in the path of the first ball. After a 10-frame delay (170 ms), the second ball moved horizontally in the same direction for 40 frames (5 cm; 660 ms) and then stopped at the edge of the screen and lay motionless for 30 frames (500 ms). For the SPATIAL gap events, the two white balls appeared motionless for 35 frames (580 ms), after which the first white ball rolled horizontally across the screen for 20 frames (4 cm; 333 ms) and stopped 1.2 cm before the leading edge of the second white ball whose leading edge was positioned in the center of the screen in the path of the first ball. Immediately after the first ball stopped, the second ball moved horizontally in the same direction for 40 frames (666 ms) and then stopped at the edge of the screen and lay motionless for 30 frames (500 ms). Note that the exact parameters for the spatial (1.2 cm) and the temporal (170 ms) gaps were chosen for two reasons. First, in pre-testing, these gaps were consistently judged non-causal by an independent sample of subjects. Specifically, empirical examination of similar spatial and temporal parameters with an independent group of participants revealed that similar CAUSAL movies elicited a causal impression on 95.8% of the trials, whereas the movies containing TEMPORAL gaps and SPATIAL gaps of magnitudes similar to those used in the current study elicited a causal impression on 4.2% and 10.4% of the trials, respectively [35]. Therefore, we can be confident that the stimulus manipulations in the current study had the properties to successfully eliminate the impression of causality. Second, the temporal gap of 170 ms is equal to the amount of time that it takes to traverse the 1.2 cm on the display that is equal to the spatial gap distance. Thus, the degrees of the spatial and temporal manipulations were equated. A graphical depiction of the CAUSAL, TEMPORAL gap, and SPATIAL gap movies used in the experiment are illustrated in Fig. 1. In order to create the strongest causal impression, the second ball moved at a velocity that was approximately 50% of the first ball [32].

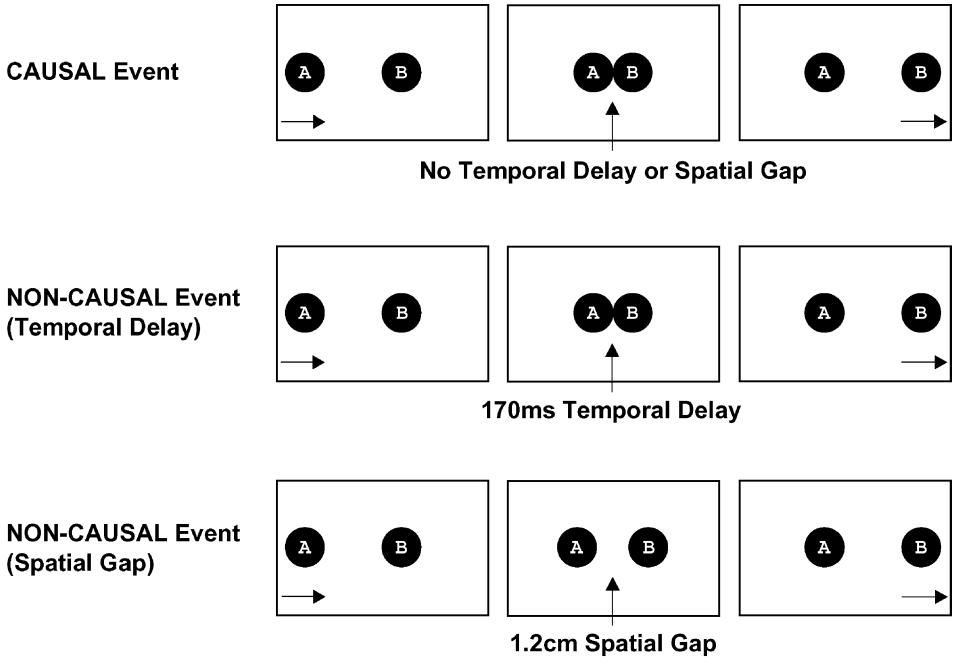

Fig. 1. Graphical illustration of CAUSAL, TEMPORAL delay, and SPATIAL gap movies used in the current experiment. The three panels depict the motion of a ball A towards a second ball B, and the subsequent motion of ball B. Note that balls used in the current study were white projected on a black display and the letters A and B are used in the figure for illustrative purposes only and did not appear on the actual stimuli.

2.4. Image acquisition

Imaging was performed on a 1.5-T whole-body scanner (General Electric Medical Systems Signa, Milwaukee, Wisconsin) with a standard head coil. Anatomical images were acquired using a high-resolution 3-D spoiled gradient recovery sequence (SPGR; 124 sagittal slices, TE = 6 ms, TR = 25 ms, flip angle = 25°, voxel size = 1 × 1 × 1.2 mm). Functional images were collected in runs using a gradient spin-echo echo-planar sequence sensitive to blood oxygen level-dependent (BOLD) contrast (T2*) (TR = 2500 ms, T2* echo time = 35 ms, flip angle = 90°, 3.75 × 3.75 mm in-plane resolution). During each functional run, 148 sets of axial images (25 slices; 5.5-mm slice thickness, 1 mm skip between slices) were acquired allowing complete brain coverage.

2.5. Statistical image analysis

All data were analyzed using SPM99 software (Wellcome Department of Cognitive Neurology, London, UK; [14]). For each functional run, data were preprocessed to remove sources of noise and artifact. Functional data were realigned within and across runs to correct for head movement using a six-parameter, rigid body alignment technique [29,49] and coregistered with each participant's anatomical data. Functional data were then transformed into a standard anatomical space (3 mm isotopic voxels) based on the ICBM 152 brain template (Montreal Neurological Institute), which approximates Talairach and Tournoux [43] atlas space using higher-order polynomial, then non-linear basis, functions [2]. Normalized data were then spatially smoothed (10 mm full-width-at-half-maximum) using a Gaussian kernel in order to optimize signal-to-noise [39] and abide by the assumptions of Gaussian random field theory [50]. The normalized and smoothed images were then used for the subsequent statistical analysis. For each subject, a general linear model [15] incorporating task effects (modeled as a box-car function convolved with the canonical hemodynamic response function), a mean, and a linear trend were used to compute parameter estimates (β) and t-contrast images (containing weighted-parameter estimates) for each comparison at each voxel.

3. Results

Table 1 presents the relative activations associated with CAUSAL stimuli minus SPATIAL and TEMPORAL incontiguities (conjunction and independently), as well as the interaction contrasts between SPATIAL and TEMPORAL incontiguities. A random-effects analysis [16,30] consisting of one-sample t tests with a hypothesized mean of 0 was applied to the individual subject t-contrast images to create mean t-images (thresholded at $P = 0.01_{\text{uncorrected}}$) for the group of 16 participants. To further protect against the probability of type 1 error, we employed an extent voxel threshold cutoff of 5, which roughly corresponds to the smoothing kernel of 10 mm used in pre-processing [13,27,51].

When the CAUSAL condition was contrasted with the NON-CAUSAL conditions (conjunction of TEMPORAL and SPATIAL gaps), areas of greatest relative activity were observed in the right middle frontal gyrus (BA 8) and the right inferior parietal lobule (BA 39), thus revealing predominantly right hemisphere loci for extracting causal structure from dynamic launching displays. When the differential effects of SPATIAL and TEMPORAL gaps were subtracted

Table 1
Brain regions and corresponding MNI coordinates for significant BOLD signal increases at $P < 0.01$ and an extent threshold of 5 contiguous voxels

Brain region (Brodmann areas) by contrast	X	Y	Z	Cluster size (k)	T value	P value
CAUSAL > NONCAUSAL						
Inferior parietal lobule (BA 39)	48	−63	45	36	4.92	<0.001
Middle frontal gyrus (BA 8)	30	33	45	15	4.61	<0.001
Superior frontal gyrus (BA 6)	21	33	57	14	3.95	0.001
Post central gyrus (BA 1/2/3)	15	−33	69	5	3.87	0.001
Thalamus	−15	−9	12	13	3.43	0.002
Precuneus (BA 7)	−12	−48	60	5	3.12	0.003
Superior parietal lobule (BA 7)	−36	−75	48	5	2.71	0.008
CAUSAL > TEMPORAL						
Inferior frontal gyrus (BA 45)	54	27	3	70	4.55	<0.001
Middle frontal gyrus (BA 8)	45	18	45	25	4.08	<0.001
Inferior parietal lobule (BA 39)	51	−60	42	68	3.53	0.002
Pre central gyrus (BA 4)	45	−6	51	9	3.36	0.002
Superior frontal gyrus (BA 8)	−39	21	51	8	2.99	0.005
CAUSAL > SPATIAL						
Middle temporal gyrus (BA 21)	57	−9	−3	17	3.89	0.001
Middle frontal gyrus (BA 8)	30	33	45	9	3.24	0.003
Superior frontal gyrus (BA 6)	21	30	57	5	3.15	0.003
Cingulate gyrus (BA 23)	−6	−36	39	10	3.05	0.004
NONCAUSAL > CAUSAL						
Occipital lingual gyrus (BA 19)	18	−63	3	15	3.21	0.003
TEMPORAL > CAUSAL						
Cuneus (BA 18)	3	−78	9	10	3.19	0.003
SPATIAL > CAUSAL						
No significant activations						
SPATIAL > TEMPORAL						
Middle frontal gyrus (BA 9)	−42	27	36	9	3.57	0.001
Precentral gyrus (BA 6)	24	−12	63	9	3.45	0.001
Supramarginal gyrus (BA 40)	−57	−51	33	25	3.43	0.001
Middle frontal gyrus (BA 6)	−21	15	57	36	2.74	0.003
Inferior parietal lobule (BA 39)	−36	−57	39	31	2.66	0.004
Insula (BA 22)	−39	−24	3	7	2.61	0.005
TEMPORAL > SPATIAL						
Occipital lingual gyrus (BA 18)	0	−72	6	18	3.63	0.001
Anterior cingulate cortex (BA 32)	0	42	−9	23	3.27	0.003
Middle temporal gyrus (BA 39)	−51	−75	12	7	3.25	0.003
Superior frontal gyrus (BA 8)	21	45	45	6	3.07	0.004
Cuneus (BA 18)	3	−78	21	11	3.04	0.004
Middle frontal gyrus (BA 9)	6	51	18	7	3.01	0.004
Superior frontal gyrus (BA 9)	30	51	33	9	2.52	0.006

from the contiguous stimuli, we observed both common and unique regions of relative activation. Specifically, both the CAUSAL > TEMPORAL and CAUSAL > SPATIAL contrasts revealed activation patterns in the right superior and middle frontal cortices (BAs 6 and 8), similar to those found in the previous CAUSAL > NON-CAUSAL contrast. Areas of unique activations for the CAUSAL > TEMPORAL gap contrast included the right inferior parietal lobule (BA 39), slightly posterior to the previous CAUSAL > NON-CAUSAL analysis, whereas unique activations for the CAUSAL > SPATIAL gap contrast recruited neural tissue in the right middle temporal gyrus (BA 21). These secondary analyses provide support for the hypothesis that the impression of causality from dynamic visual displays recruits common regions associated with attentional/executive processes [9,20,21,41] in concert with more specialized neural tissue associated with rapid temporal [52] and spatial [11] processing.

Further support for this hypothesis comes from examining the interaction between SPATIAL and TEMPORAL incontiguities. When areas of activation occurring for the TEMPORAL delay condition were subtracted from the SPATIAL gap condition, areas of greatest relative activity were observed in the left middle frontal gyrus (BA 9), right precentral gyrus (BA 6), left supramarginal gyrus (BA 40), and inferior parietal lobule (BA 39). These activation patterns closely mirror those of the CAUSAL > TEMPORAL contrast revealing a common frontal and more specialized parietal neural recruitment pattern. When areas of activation occurring for the SPATIAL gap condition were subtracted from the TEMPORAL delay condition, areas of greatest relative activity were observed in the occipital lingual gyrus (BA 18), anterior cingulate cortex (BA 32), and middle temporal gyrus (BA 39). Here too, like the CAUSAL > SPATIAL contrast, subtracting spatial incontiguities from the dynamic displays selectively recruited more neural tissue in a frontal/parietal/temporal network. It is of interest to note that these interaction contrasts that involve NON-CAUSAL stimuli recruit more bilateral regions when compared to the CAUSAL contrasts (i.e., CAUSAL > TEMPORAL and CAUSAL > SPATIAL).

4. General discussion

Recent studies of the neural basis of causal cognition indicate that specific brain networks are involved in extracting causal structure from the world [3,12,20,21,35]. In the present study, we examined the neurological foundations of two cues that are involved in determining causality: spatial and temporal contiguity. Using fMRI, we demonstrate that spatial and temporal cues to causality recruited both common and unique neural tissue in the brain. The frontal/parietal and frontal/temporal nature of these activation patterns suggest that the process of extracting causal structure from dynamic visual events involves the recruitment of a distributed network of brain regions that have been implicated in visual perception [8,23,25,28], and executive processing [9,41]. In addition, our data lend support to theories of causal perception that propose the extraction of causal structure as being an inherent property of the visual system [3,12,35,37] akin to processes such as visual grouping, and illusory contour completion [7].

The right lateralized nature of these activations replicates and extends our recent research examining these issues in split-brain patients [35]. Specifically, these data demonstrate that extracting causal structure from dynamic visual displays depends on brain networks localized within the right hemisphere, even in the presence of callosal transfer in the normal brain. In addition, the finding that causal stimuli, as opposed to stimuli with spatial or temporal incontiguities, invoked regions of the right prefrontal cortex suggests that such stimuli may recruit additional higher-order executive/attentional resources beyond those afforded by the visual system. These data are also consistent with, and extend recent work on, complex causal reasoning conducted by Fugelsang and Dunbar [20]. Fugelsang and Dunbar presented participants with a task requiring them to interpret data relative to plausible and implausible causal theories. The plausibility of the causal theories were manipulated by presenting participants with a brief introductory statement that depicted a causal theory that contained either a plausible mechanism of action, or an implausible mechanism of action. Evaluation of theories that contained a plausible mechanism as opposed to an implausible mechanism selectively recruited neural tissue in bilateral prefrontal regions. They proposed that this prefrontal recruitment may represent the selective allocation of attentional resources to stimuli that contain plausible causal mechanisms of action. In the present experiment, using simple stimuli that do not require complex inferential processes, we see a similar pattern of data. Specifically, when stimuli are perceived to possess causal structure (i.e., collide and immediately move), similar regions in the prefrontal cortex, albeit localized in the right hemisphere, are recruited. Here too, the preferential recruitment of regions in the prefrontal cortices for causal stimuli suggests that such stimuli may capture visual attention [10] and result in more attentional resources devoted to such stimuli [9,20,21,41]. Indeed, this allocation of attentional resources and subsequent recruitment of prefrontal cortex may be one of the hallmarks of the perception of causality.

An important avenue for future research is to examine the extent to which perceptual and inferential processes of causality are dissociable in the normal brain. Gazzaniga [24] has argued for a ‘left hemisphere interpreter’ that is proposed to interpret and generate hypotheses about complex stimuli and actions. One could easily envision a primary role of the ‘left hemisphere interpreter’ for a task involving causal inference. Roser et al. [35] have recently shown that the direct perception of causality and the ability to infer causality depend on different hemispheres of the divided brain. Our right hemispheric loci observed in the present study are

consistent with this evidence and support the hypothesis that perceiving causality from dynamic visual events is predominantly achieved through right hemisphere processing. The degree to which perceptual and inferential components of causality rely on common or dissociable neural tissue in the normal brain is still unknown and remains an important issue for future research. In addition, our findings, coupled with prior split-brain work, imply that understanding causality may not be a unitary process and that processes associated with the direct perception of, and with inference about, complex stimulus interactions may proceed independently and rely on different underlying brain networks.

Taken together, our data represent an initial step in examining how the brain makes use of various cues to uncover causal structure in the environment. The extent to which causal perception represents a unique faculty requiring specialized neural circuitry versus one that is an emergent property that draws on a network of shared cognitive resources remains an important question for future research. In addition, much can be learned by examining the ways in which spatial and temporal contiguity are integrated with alternative cues to causality, such as mechanism [1] and covariation information [5].

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