

Animacy semantic network supports implicit causal inferences about illness

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eLife Assessment

This study investigates the neural basis of causal inference of illness, suggesting that it relies on semantic networks specific to living things in the absence of a generalized representation of causal inference across domains. The main hypothesis is **compelling**, and is supported by **solid** methods and data analysis. Overall, the findings make a **valuable** contribution to understanding the role of domain-specific semantic networks, particularly the precuneus, in implicit causal inference about illness.

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Abstract

Inferring the causes of illness is a culturally universal example of causal thinking. We tested the hypothesis that implicit causal inferences about biological processes (e.g., illness) depend on the animacy semantic network. Participants ($n=20$) undergoing fMRI read two-sentence vignettes that elicited causal inferences across sentences, either about the emergence of illness or about the mechanical breakdown of inanimate objects, in addition to noncausal control vignettes. All vignettes were about people and were linguistically matched. The same participants performed localizer tasks: language, logical reasoning, and mentalizing. Inferring illness causes, relative to all control conditions, selectively engaged a portion of the precuneus (PC) previously implicated in the semantic representation of animates (e.g., people, animals). Neural responses to causal inferences about illness were adjacent to but distinct from responses to mental state inferences, suggesting a neural mind/body distinction. We failed to find evidence for domain-general responses to causal inference. Implicit causal inferences are supported by content-specific semantic networks that encode causal knowledge.

Introduction

A distinguishing feature of human cognition is our ability to reason about complex cause-effect relationships, particularly when causes are not directly perceptible (Tooby & DeVore, 1987 [2](#), Lagnado et al., 2007 [3](#); Rottman, Ahn, & Luhmann, 2011 [4](#); Muentener & Schulz, 2014 [5](#); Sloman & Lagnado, 2015 [6](#); Goddu & Gopnik, 2024 [7](#)). When reading something like, *Hugh sat by sneezing*

passengers on the subway. Now he has a case of COVID, we naturally infer a causal relationship between crowded spaces and the invisible transmission of infectious disease. Here we investigate the neurocognitive mechanisms that support such automatic inferences by studying causal inferences about illness.

Adults have rich, culturally-specific causal knowledge about the invisible forces that bring about illness, from pathogen transmission to divine retribution (Notaro, Gelman, & Zimmerman, 2001 [2](#); Raman & Winer, 2004 [2](#); Lynch & Medin, 2006 [2](#); Legare & Gelman, 2008 [2](#); Legare et al., 2012 [2](#); Legare & Shtulman, 2017 [2](#)). In many societies, designated ‘healers’ become experts in diagnosing and treating disease (Foster, 1976 [2](#); Ackerknecht, 1982 [2](#); Norman et al., 2009 [2](#); Lightner, Heckelsmiller, & Hagen, 2021 [2](#)). Non-expert adults routinely infer the causes of illness in themselves and others (e.g., *how did my friend get COVID?*). Even young children think about illness in systematic ways, reflecting their burgeoning commonsense understanding of the biological world (Wellman & Gelman, 1992 [2](#); Keil, 1992 [2](#); Inagaki & Hatano, 2006 [2](#)). Young children attribute illness to contaminated food, contact with a sick person, and parental inheritance (Springer & Ruckel, 1992 [2](#); Kalish, 1996 [2](#), 1997 [2](#); Keil et al., 1999 [2](#); Notaro et al., 2001 [2](#); Raman & Winer, 2004 [2](#); Raman & Gelman, 2005 [2](#); Legare & Gelman, 2008 [2](#); Legare, Wellman, & Gelman, 2009 [2](#); DeJesus, Venkatesh, & Kinzler, 2021 [2](#)).

Illness affects living things (e.g., people and animals) rather than inanimate objects (e.g., rocks, machines, houses). Thinking about living things (animates) as opposed to non-living things (inanimate objects/places) recruits partially distinct neural systems (e.g., Warrington & Shallice, 1984 [2](#); Hillis & Caramazza, 1991 [2](#); Caramazza & Shelton, 1998 [2](#); Farah & Rabinowitz, 2003 [2](#)). The precuneus (PC) is part of the ‘animacy’ semantic network and responds preferentially to living things (i.e., people and animals), whether presented as images or words (Devlin et al., 2002 [2](#); Fairhall & Caramazza, 2013a [2](#), 2013b [2](#); Fairhall et al., 2014 [2](#); Peer et al., 2015 [2](#); Wang et al., 2016 [2](#); Silson et al., 2019 [2](#); Rabini, Ubaldi, & Fairhall, 2021 [2](#); Deen & Freiwald, 2022 [2](#); Aglinskas & Fairhall, 2023 [2](#); Hauptman, Elli, et al., 2025 [2](#)). By contrast, parts of the visual system (e.g., fusiform face area) that respond preferentially to animates do so primarily for images (Kanwisher et al., 1997 [2](#); Grill-Spector et al., 2004 [2](#); Noppeney et al., 2006 [2](#); Mahon et al., 2009 [2](#); Konkle & Caramazza, 2013 [2](#); Connolly et al., 2016; see Bi et al., 2016 [2](#) for a review). We hypothesized that the PC represents causal knowledge relevant to animates and tested the prediction that it would be activated during implicit causal inferences about illness, which rely on such knowledge (preregistration: <https://osf.io/6pnqg> [2](#)).

We also compared neural responses to causal inferences about the body (i.e., illness) and inferences about the mind (i.e., mental states). Both types of inferences are about animate entities, and some developmental work suggests that children use the same set of causal principles to think about bodies and minds (Carey, 1985 [2](#), 1988 [2](#)). Other evidence suggests that by early childhood, young children have distinct causal knowledge about the body and the mind (Springer & Keil, 1991 [2](#); Callanan & Oakes, 1992 [2](#); Wellman & Gelman, 1992 [2](#); Inagaki & Hatano, 1993 [2](#), 2004 [2](#); Keil, 1994 [2](#); Hickling & Wellman, 2001 [2](#); Medin et al., 2010 [2](#)). For instance, preschoolers are more likely to view illness as a consequence of biological causes, such as contagion, rather than psychological causes, such as malicious intent (Springer & Ruckel, 1992 [2](#); Raman & Winer, 2004 [2](#); see also Legare & Gelman, 2008 [2](#)). The neural relationship between inferences about bodies and minds has not been fully described. The ‘mentalizing network’, including the PC, is engaged when people reason about agents’ beliefs (Saxe & Kanwisher, 2003 [2](#); Saxe et al., 2006 [2](#); Saxe & Powell, 2006 [2](#); Dodell-Feder et al., 2011 [2](#); Dufour et al., 2013 [2](#)). We localized this network in individual participants and measured its neuroanatomical relationship to the network activated by illness inferences.

An alternative hypothesis is that domain-general neural mechanisms, separate from semantic networks, support causal inferences across domains. Children and adults make causal inferences across a wide range of domains and use similar cognitive principles (e.g., ‘screening off’) when

doing so (e.g., Saxe & Carey, 2006 [\(2\)](#); Tenenbaum et al., 2007 [\(2\)](#); Carey, 2011 [\(2\)](#); Cheng & Novick, 1992 [\(2\)](#); Waldmann & Holyoak, 1992 [\(2\)](#); Pearl, 2000 [\(2\)](#); Gopnik et al., 2001 [\(2\)](#); Steyvers et al., 2003 [\(2\)](#); Gopnik et al., 2004 [\(2\)](#); Schulz & Gopnik, 2004 [\(2\)](#); Rehder & Burnett, 2005 [\(2\)](#); Lagnado et al., 2007 [\(2\)](#); Rottman & Hastie, 2014 [\(2\)](#); Davis & Rehder, 2020 [\(2\)](#)). Prior neuroscience work has hypothesized that the frontotemporal language network may support a broad range of causal inferences during comprehension (Kuperberg et al., 2006 [\(2\)](#); Mason & Just, 2011 [\(2\)](#); Prat et al., 2011 [\(2\)](#); see also Spelke, 2003 [\(2\)](#); 2022 [\(2\)](#); Pinker, 2003 [\(2\)](#)). Alternatively, causal inference could depend on frontoparietal mechanisms that also support other types of reasoning, such as logical deduction (Goldvarg & Johnson-Laird, 2001 [\(2\)](#); Barbey & Patterson, 2011 [\(2\)](#); Khemlani et al., 2014 [\(2\)](#); Operksalski & Barbey, 2017 [\(2\)](#)). Finally, it has been suggested that causal inferences are supported by a dedicated ‘causal engine’ in prefrontal cortex that supports all and only causal inferences across domains (Pramod, Chomik-Morales, et al., 2023 [\(2\)](#)). We tested these alternative hypotheses in the specific case of implicit causal inferences that unfold naturally during language comprehension (Black & Bern, 1981 [\(2\)](#); Keenan et al., 1984 [\(2\)](#); Trabasso & Sperry, 1985 [\(2\)](#); Myers et al., 1987 [\(2\)](#); Duffy et al., 1990 [\(2\)](#)).

Most prior studies investigating causal inference used explicit causality judgment tasks (Ferstl & von Cramon, 2001 [\(2\)](#); Satpute et al., 2005 [\(2\)](#); Fugelsang & Dunbar, 2005 [\(2\)](#); Kuperberg et al., 2006 [\(2\)](#); Fenker et al., 2010 [\(2\)](#); Kranjec et al., 2012 [\(2\)](#); Pramod, Chomik-Morales, et al., 2023 [\(2\)](#)). For example, Kuperberg et al. (2006) [\(2\)](#) asked participants to rate the causal relatedness of three-sentence stories and observed higher responses to causally related stories in left frontotemporal cortex. Studies of implicit causal inference find frontotemporal and frontoparietal responses (Chow et al., 2008 [\(2\)](#); Mason & Just, 2011 [\(2\)](#); Prat et al., 2011 [\(2\)](#)). Across these prior studies, no consistent neural signature of causal inference has emerged. Importantly, in many studies, causal trials were more difficult, and/or linguistic variables were not matched across causal and noncausal conditions. As a result, some of the observed effects may reflect linguistic or executive load. In addition, almost no prior studies localized language or logical reasoning networks in individual participants, making it difficult to assess the involvement of these systems (e.g., Fedorenko et al., 2010 [\(2\)](#); Monti et al., 2009 [\(2\)](#); cf. Pramod, Chomik-Morales, et al., 2023 [\(2\)](#)). Most prior work also did not distinguish between causal inferences about different semantic domains known to depend on partially distinct neural networks, e.g., biological, mechanical, or mental state inferences (cf. Mason & Just, 2011 [\(2\)](#); Pramod, Chomik-Morales, et al., 2023 [\(2\)](#)). If such inferences recruit partially distinct neural systems, their neural signatures might have been missed.

In the current experiment, participants read two-sentence vignettes (e.g., “Hugh sat by sneezing passengers on the subway. Now he has a case of COVID”). The first sentence described a potential cause and the second sentence a potential effect. Such causally connected sentences arise frequently in naturalistic discourse (Singer, 1994 [\(2\)](#); Graesser et al., 1994 [\(2\)](#)). Participants performed a covert task of detecting ‘magical’ catch trial vignettes that encouraged them to attend to the meaning of the critical vignettes while reading as naturally as possible. We chose an orthogonal foil detection task rather than an explicit causal judgment task to investigate automatic causal inferences during reading and to unconfound such processing as much as possible from explicit decision-making processes. Analogous foil detection paradigms have been used to study sentence processing and word recognition (e.g., Pallier et al., 2011 [\(2\)](#); Dehaene-Lambertz et al., 2018 [\(2\)](#)).

In the current study, causal inferences about illness were compared to two control conditions: i) causal inferences about mechanical breakdown (e.g., “Jake dropped all of his things on the subway. Now he has a shattered phone.”) and ii) illness-related language that was not causally connected (e.g., “Lynn dropped all of her things on the subway. Now she has a case of COVID.”). This combination of control conditions allowed us to test jointly for sensitivity to content domain and causality. In other words, we tested the hypothesis that causal inferences about illness recruit the animacy semantic network. Critically, all vignettes, including mechanical ones, described events involving people, such that responses to causal inferences about illness in the animacy

network could not be explained by the presence of animate agents. As a further control, we included the number of people in each vignette as a covariate of no interest in our neural analysis. Noncausal vignettes were constructed by shuffling causes/effects across conditions and were therefore matched to the causal vignettes in linguistic content. A separate group of participants rated the causal relatedness of all vignettes prior to the experiment. In addition to the main causal inference experiment, we also localized language, logical reasoning, and mentalizing networks in each participant. Following prior work, we predicted that neural systems that support causal inference would exhibit increased activity during such inferences. Thus, our primary neural prediction was that animacy-responsive PC would respond more to causal inferences about illness compared to all other control conditions. We also used multivariate methods to investigate differences between conditions.

Method

Open science practices

The methods and analysis of this experiment were pre-registered prior to data collection (<https://osf.io/6pnqg>).

Participants

Twenty adults (7 women, 13 men, 25-37 years old, $M = 28.7$ years ± 3.2 SD) participated in the study. Participants either had or were pursuing graduate degrees ($M = 8.8$ years of post-secondary education). Two additional participants were excluded from the final dataset due to excessive head motion (> 2 mm) and an image artifact. One participant in the final dataset exhibited excessive head motion (> 2 mm) during 1 run of the language/logic localizer task that was excluded from analysis. All participants were screened for cognitive and neurological disabilities (self-report). Participants gave written informed consent and were compensated \$30 per hour. The study was reviewed and approved by the Johns Hopkins Medicine Institutional Review Boards.

Causal inference experiment

Stimuli

Participants read two-sentence vignettes in 4 conditions, 2 causal and 2 noncausal (**Figure 1D**). Each vignette focused on a single agent, specified by a proper name in the initial sentence and by a pronoun in the second sentence. The first sentence described something the agent did or experienced and served as the potential cause. The second sentence described the potential effect (e.g., “Kelly shared plastic toys with a sick toddler at her preschool. Now she has a case of chickenpox”). *Illness-Causal* vignettes elicited inferences about biological causes of illness, including pathogen transmission, exposure to environmental toxins, and genetic mutations (see **Supplementary Table 1** for a full list of the types of illnesses included in our stimuli).

Mechanical-Causal vignettes elicited inferences about physical causes of structural damage to personally valuable inanimate objects (e.g., houses, jewelry). Two noncausal conditions used the same sentences as in the *Illness-Causal* and *Mechanical-Causal* conditions but in a shuffled order: illness cause with mechanical effect (*Noncausal-Illness First*) or mechanical cause with illness effect (*Noncausal-Mechanical First*). Explicit causality judgments collected from a separate group of online participants ($n=26$) verified that the both causal conditions *Illness-Causal*, *Mechanical-Causal* were more causally related than both noncausal conditions, $t(25) = 36.97$, $p < .001$. In addition, *Illness-Causal* and *Mechanical-Causal* items received equally high causality ratings, $t(25) = -0.64$, $p = .53$ (see [Appendix 1](#) for details).

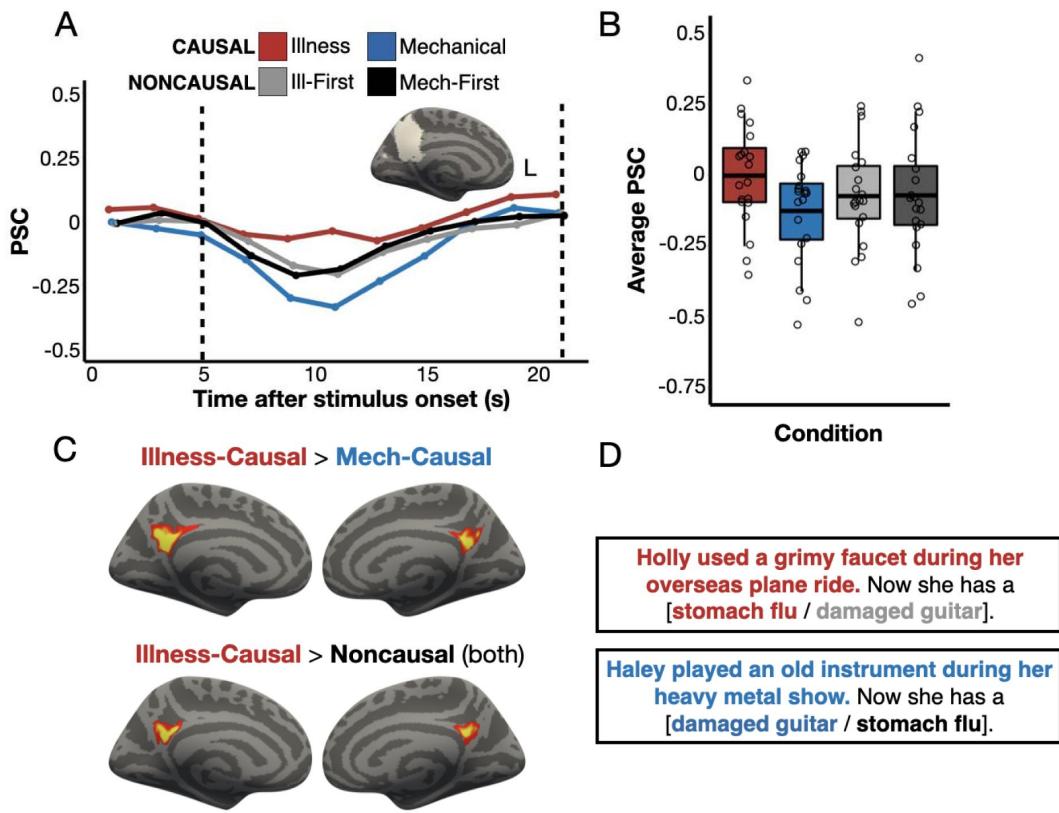


Figure 1.

Responses to illness inferences in the precuneus (PC). Panel A: Percent signal change (PSC) for each condition among the top 5% *Illness-Causal* > *Mechanical-Causal* vertices in a left PC search space (Dufour et al., 2013) in individual participants, established via a leave-one-run-out analysis. Panel B: Average PSC in the critical window (marked by dotted lines in Panel A) across participants. The horizontal line within each boxplot indicates the overall mean. Panel C: Whole-cortex results (one-tailed) for *Illness-Causal* > *Mechanical-Causal* and *Illness-Causal* > *Noncausal* (both versions of noncausal vignettes), corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$. Panel D: Example stimuli. ‘Magical’ catch trials similar in meaning and structure (e.g., “Sadie forgot to wash her face after she ran in the heat. Now she has a cucumber nose”) enabled the use of a semantic ‘magic detection’ task.

Illness-Causal and *Mechanical-Causal* vignettes were constructed in pairs, such that each member of a given pair shared parallel or near-parallel phrase structure. All conditions were also matched (pairwise t-tests, all $p > 0.3$, no statistical correction) on multiple linguistic variables known to modulate neural activity in language regions (e.g., Pallier et al., 2011 [2](#); Shain, Blank et al., 2020 [2](#)). These included number of characters, number of words, average number of characters per word, average word frequency, average bigram surprisal (Google Books Ngram Viewer, <https://books.google.com/ngrams/>), and average syntactic dependency length (Stanford Parser; de Marneffe, MacCartney, & Manning, 2006 [2](#)). Word frequency was calculated as the negative log of a word's frequency in the Google corpus between the years 2017-2019. Bigram surprisal was calculated as the negative log of the frequency of a given two-word phrase in the Google corpus divided by the frequency of the first word of the phrase (see Appendix 2 [2](#) for details). All conditions were matched for all linguistic variables across the first sentence, second sentence, and the entire vignette.

Procedure

We used a ‘magic detection’ task to encourage participants to process the meaning of the vignettes without making explicit causality judgments. Participants saw ‘magical’ catch trials that closely resembled the experimental trials but were fantastical (e.g., “Sadie forgot to wash her face after she ran in the heat. Now she has a cucumber nose”). On each trial, participants indicated via button press whether ‘something magical’ occurred in the vignette (Yes/No). This semantic foil detection task encouraged participants to attend to the meaning of the critical vignettes while reading as naturally as possible. We required participants to press a button on every trial to ensure they were attending to the stimuli. Both sentences in a given vignette were presented simultaneously for 7 s, one above the other, followed by a 12 s inter-trial interval. Each participant saw 38 trials per condition (152 trials) plus 36 ‘magical’ catch trials (188 total trials) in one of two versions, counterbalanced across participants, such that individual participants did not see the same sentence in both causal and noncausal vignettes. The two stimulus versions had similar meanings but different surface forms (e.g., “Luna stood by coughing travelers on the train...” vs. “Hugh sat by sneezing passengers on the subway...”).

The experiment was divided into 6 10-minute runs containing 6-7 trials per condition per run presented in a pseudorandom order. Vignettes from the same experimental condition repeated no more than twice consecutively, vignettes that shared similar phrase structure never repeated within a run, vignettes that referred to the same illness never repeated consecutively, and vignettes from each condition, including catch trials, were equally distributed in time across the course of the experiment.

Mentalizing localizer experiment

To test the relationship between neural responses to inferences about the body and the mind, and to localize animacy regions, we used a localizer task to identify the mentalizing network in each participant (Saxe & Kanwisher, 2003 [2](#); Dodell-Feder et al., 2011 [2](#); <http://saxelab.mit.edu/use-our-efficient-false-belief-localizer> [2](#)). In this task, participants read 10 mentalizing stories (e.g., a protagonist has a false belief about an object’s location) and 10 physical stories (physical representations depicting outdated scenes, e.g., a photograph showing an object that has since been removed) before answering a true/false comprehension question. We used the mentalizing stories from the original localizer but created new stimuli for the physical stories condition. Our physical stories incorporated more vivid descriptions of physical interactions and did not make any references to human agents, enabling us to use the mentalizing localizer as a localizer for animacy. The new physical stories were also linguistically matched to the mentalizing stories to reduce linguistic confounds (see Shain et al., 2023 [2](#)). Specifically, we matched physical and mentalizing stories (pairwise t-tests, all $p > 0.3$, no statistical correction) for number of characters, number of words, average number of characters per word, average syntactic dependency length,

average word frequency, and average bigram surprisal, as was done for the causal inference vignettes. A comparison of both localizer versions in 3 pilot participants can be found in [Supplementary Figure 15](#).

Trials were presented in an event-related design, with each one lasting 16 s (12 s stories + 4 s comprehension question) followed by a 12 s inter-trial interval. Participants completed 2 5-minute runs of the task, with trial order counterbalanced across runs and participants. The mentalizing network was identified in individual participants by contrasting *mentalizing stories > physical stories* (Saxe & Kanwisher, 2003; Dodell-Feder et al., 2011).

Language/logic localizer experiment

To test for the presence of domain-general responses to causal inference in the language and logic networks (e.g., Kuperberg et al., 2006; Operksalski & Barbey, 2017), we used an additional localizer task. The task had three conditions: language, logic, and math. In the language condition, participants judged whether two visually presented sentences, one in active and one in passive voice, shared the same meaning. In the logic condition, participants judged whether two logical statements were consistent (e.g., *If either not Z or not Y then X vs. If not X then both Z and Y*). In the math condition, participants judged whether the variable *X* had the same value across two equations (for details see Liu et al., 2020). Trials lasted 20 s (1 s fixation + 19 s display of stimuli) and were presented in an event-related design. Participants completed 2 9-minute runs of the task, with trial order counterbalanced across runs and participants. Following prior studies, the language network was identified in individual participants by contrasting *language > math* and the logic network by contrasting *logic > language* (Monti et al., 2009; Kanjlia et al., 2016; Liu et al., 2020).

Data acquisition

Whole-brain fMRI data was acquired at the F.M. Kirby Research Center of Functional Brain Imaging on a 3T Phillips Achieva Multix X-Series scanner. T1-weighted structural images were collected in 150 axial slices with 1 mm isotropic voxels using the magnetization-prepared rapid gradient-echo (MP-RAGE) sequence. T2*-weighted functional BOLD scans were collected in 36 axial slices (2.4 2.43 mm voxels, TR = 2 s). Data were acquired in one experimental session lasting approximately 120 minutes. All stimuli were visually presented on a rear projection screen with a Cambridge Research Systems **BOLDscreen 32 UHD** LCD display (image resolution = 1920 x 1080) using custom scripts written in PsychoPy3 (<https://www.psychopy.org/>, Peirce et al., 2019). Participants viewed the screen via a front-silvered, 45° inclined mirror attached to the top of the head coil.

fMRI data preprocessing and general linear model (GLM) analysis

Preprocessing included motion correction, high-pass filtering (128 s), mapping to the cortical surface (Freesurfer), spatially smoothing on the surface (6 mm FWHM Gaussian kernel), and prewhitening to remove temporal autocorrelation. Covariates of no interest included signal from white matter, cerebral spinal fluid, and motion spikes.

For the main causal inference experiment, the GLM modeled the four main conditions (*Illness-Causal, Mechanical-Causal, Noncausal-Illness First, Noncausal-Mechanical First*) and the ‘magical’ catch trials during the 7 s display of the vignettes after convolving with a canonical hemodynamic response function and its first temporal derivative. The GLM additionally included participant response time and number of people in each vignette as covariates of no interest. For the mentalizing localizer experiment, a separate predictor was included for each condition (*mentalizing stories, physical stories*), modeling the 16 s display of each story and corresponding

comprehension question. For the language/logic localizer experiment, a separate predictor was included for each of the three conditions (*language, logic, math*), modeling the 20 s duration of each trial.

For each task, runs were modeled separately and combined within-subject using a fixed-effects model (Dale, Fischl, & Sereno, 1999 [\(2\)](#); Smith et al., 2004 [\(2\)](#)). Group-level random-effects analyses were corrected for multiple comparisons across the whole cortex at $p < .05$ family-wise error rate (FWER) using a nonparametric permutation test (cluster-forming threshold $p < .01$ uncorrected) (Winkler et al., 2014 [\(2\)](#); Eklund, Nichols, & Knutsson, 2016 [\(2\)](#); Eklund, Knutsson, & Nichols, 2019 [\(2\)](#)).

Individual-subject fROI analysis: univariate

We defined individual-subject functional ROIs (fROIs) in the PC and temporoparietal junction (TPJ) as well as in the language (frontal and temporal search spaces) and logic networks. In an exploratory analysis, we defined individual-subject fROIs in an anterior parahippocampal region (i.e., anterior PPA). For all analyses, percent signal change (PSC) was extracted and averaged over the entire duration of the trial (17 s total), starting at 4 s to account for hemodynamic lag.

Illness inference fROIs were created in bilateral PC and TPJ group search spaces (Dufour et al., 2013 [\(2\)](#)) using an iterated leave-one-run-out procedure, which allowed us to perform sensitive individual-subjects analysis while avoiding statistical non-independence (Vul & Kanwisher, 2011 [\(2\)](#)). In each participant, we identified the most illness inference-responsive vertices in bilateral PC and TPJ search spaces separately in 5 of the 6 runs (top 5% of vertices, *Illness-Causal* > *Mechanical-Causal*). We then extracted PSC for each condition compared to rest in the held-out run (*Illness-Causal*, *Mechanical-Causal*, *Noncausal-Illness First*, *Noncausal-Mechanical First*), averaging the results across all iterations. We used the same approach to create mechanical inference fROIs in bilateral anterior PPA search spaces from a previous study on place word representations (Hauptman, Elli, et al., 2025 [\(2\)](#)). All aspects of this analysis were the same as those described above, except that the most mechanical inference-responsive vertices (top 5%, *Mechanical-Causal* > *Illness-Causal*) were selected.

Mentalizing fROIs were created by selecting the most mentalizing-responsive vertices (top 5%) in bilateral PC and TPJ search spaces (Dufour et al., 2013 [\(2\)](#)) using the *mentalizing stories* > *physical stories* contrast from the mentalizing localizer. Language fROIs were identified by selecting the most language-responsive vertices (top 5%) in left frontal and temporal language areas (search spaces: Fedorenko et al., 2010 [\(2\)](#)) using the *language* > *math* contrast from the language/logic localizer. A logic-responsive fROI was identified by selecting the most logic-responsive vertices (top 5%) in a left frontoparietal network (search space: Liu et al., 2020 [\(2\)](#)) using the *logic* > *language* contrast. In each fROI, we extracted PSC for all conditions in the causal inference experiment.

Individual-subject fROI analysis: multivariate

We performed MVPA (PyMVPA toolbox; Hanke et al., 2009 [\(2\)](#)) to test whether patterns of activity in the PC, TPJ, language network, and logic network distinguished illness inferences from mechanical inferences. In each participant, we identified the top 300 vertices most responsive to the mentalizing localizer (*mentalizing stories* > *physical stories*) in bilateral PC and TPJ search spaces (Dufour et al., 2013 [\(2\)](#)). We also identified the top 300 vertices most responsive to language (*language* > *math*) in a left language network search space (Fedorenko et al., 2010 [\(2\)](#)) and the top 300 vertices most responsive to logical reasoning (*logic* > *language*) in a left logic network search space (Liu et al., 2020 [\(2\)](#)).

In an exploratory analysis, we performed MVPA to test whether patterns of activity in the left PC and in the language and logic networks distinguished causal from noncausal vignettes. To avoid statistical non-independence, we defined additional fROIs in the left PC for the purposes of this

analysis. In each participant, we identified the top 300 vertices most responsive to the critical conditions over rest (*Illness-Causal + Mechanical-Causal + Noncausal-Illness First + Noncausal-Mechanical First > Rest*) in a left PC search space (Dufour et al., 2013 [2](#)).

For each vertex in each participant's fROIs, we obtained one observation per condition per run (z-scored beta parameter estimate of the GLM). A linear support vector machine (SVM) was then trained on data all but one of the runs and tested on the left-out run in a cross-validation procedure. Classification accuracy was averaged across all permutations of the training/test splits. We compared classifier performance within each fROI to chance (50%; one-tailed test). Significance was evaluated against an empirically generated null distribution using a combined permutation and bootstrap approach (Schreiber & Krekelberg, 2013 [2](#); Stelzer et al., 2013 [2](#)). In this approach, t-statistics obtained for the observed data are compared against an empirically generated null distribution. We report the t-values obtained for the observed data and the nonparametric p-values, where p corresponds to the proportion of the shuffled analyses that generated a comparable or higher t-value.

The null distribution was generated using a balanced block permutation test by shuffling condition labels within run 1000 times for each subject (Schreiber & Krekelberg, 2013 [2](#)). Then, a bootstrapping procedure was used to generate an empirical null distribution for each statistical test across participants by sampling one permuted accuracy value from each participant's null distribution 15,000 times (with replacement) and running each statistical test on these permuted samples, thus generating a null distribution of 15,000 statistical values for each test (Stelzer et al. 2013 [2](#)).

Searchlight MVPA

We used a linear support vector machine classifier to test decoding between all pairs of causal and noncausal conditions (i.e., *Illness-Causal vs. Mechanical-Causal*, *Illness-Causal vs. Noncausal-Mechanical First*, *Illness-Causal vs. Noncausal-Illness First*, *Mechanical-Causal vs. Noncausal-Mechanical First*, and *Mechanical-Causal vs. Noncausal-Illness First*) across the whole cortex using a 10 mm radius spherical searchlight (according to geodesic distance, to better respect cortical anatomy over Euclidean distance; Glasser et al., 2013 [2](#)). This yielded for each participant 5 classification maps indicating the classifier's accuracy in a neighborhood surrounding every vertex. Individual subject searchlight accuracy maps were then averaged within analysis, and the resulting group-wise maps were thresholded using the PyMVPA implementation of the 2-step cluster-thresholding procedure described in Stelzer et al. (2013 [2](#)) (Hanke et al., 2009 [2](#)). This procedure permutes block labels within participant to generate a null distribution within subject (100 times) and then samples from these (10,000) to generate a group-wise null distribution (as in the fROI analysis). The whole-brain searchlight maps are then thresholded using a combination of vertex-wise threshold ($p < .001$ uncorrected) and cluster size threshold (FWER $p < .05$, corrected for multiple comparisons across the entire cortical surface).

Results

Behavioral results

Accuracy on the magic detection task was at ceiling ($M = 97.9\% \pm 2.2$ SD) and there were no significant differences across the 4 main experimental conditions (*Illness-Causal*, *Mechanical-Causal*, *Noncausal-Illness First*, *Noncausal-Mechanical First*), $F_{(3,57)} = 2.39$, $p = .08$. A one-way repeated measures ANOVA evaluating response time revealed a main effect of condition, $F_{(3,57)} = 32.63$, $p < .001$, whereby participants were faster on *Illness-Causal* trials ($M = 4.73 \pm 0.81$ SD) compared to *Noncausal-Illness First* ($M = 5.33$ s ± 0.85 SD) and *Noncausal-Mechanical First* ($M = 5.27$ s ± 0.89 SD) trials. There were no differences in response time between the *Mechanical-Causal*

condition ($M = 5.15$ s ± 0.88 SD) and any other conditions. Performance on the localizer tasks was similar to previously reported studies that used these paradigms (see Appendix 3 for full behavioral results).

Inferring illness causes recruits animacy-responsive PC

We found distinctly localized neural responses to causal inferences about illness relative to both mechanical causal inferences and noncausal vignettes. A bilateral precuneus (PC) region previously implicated in thinking about animate entities (i.e., people and animals) responded preferentially to causal inferences about illness over both mechanical causal inferences and causally unrelated sentences in whole-cortex analysis ($p < .05$, corrected for multiple comparisons; **Figure 1C**) and in individual-subject overlap maps (**Supplementary Figures 6** and **7**). PC responses during illness inferences overlapped with previously reported responses to people-related concepts (Fairhall & Caramazza, 2013b; **Supplementary Figure 2**).

Relative to illness inferences and noncausal vignettes, inferring the causes of mechanical breakdown in inanimate entities activated bilateral anterior parahippocampal regions (i.e., anterior PPA), suggesting a double dissociation between illness and mechanical inferences (**Figure 4C**) (Epstein & Kanwisher, 1998; Weiner et al., 2018). This anterior PPA region is engaged during memory/verbal tasks about physical spaces (Baldassano et al., 2013; Fairhall et al., 2014; Silson et al., 2019; Steel et al., 2021; Häusler et al., 2022; Hauptman, Elli, et al., 2025).

In individual-subject fROI analysis (leave-one-run-out), we similarly found that inferring illness causes activated the PC more than inferring causes of mechanical breakdown (repeated measures ANOVA, condition (*Illness-Causal, Mechanical-Causal*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 19.18$, $p < .001$, main effect of hemisphere, $F_{(1,19)} = 0.3$, $p = .59$, condition x hemisphere interaction, $F_{(1,19)} = 27.48$, $p < .001$; **Figure 1A**). This effect was larger in the left than in the right PC (paired samples t-tests; left PC: $t_{(19)} = 5.36$, $p < .001$, right PC: $t_{(19)} = 2.27$, $p = .04$). Illness inferences also activated the PC more than illness-related language that was not causally connected (repeated measures ANOVA, condition (*Illness-Causal, Noncausal-Illness First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 4.66$, $p = .04$, main effect of hemisphere, $F_{(1,19)} = 2.51$, $p = .13$, condition x hemisphere interaction, $F_{(1,19)} = 8.07$, $p = .01$; repeated measures ANOVA, condition (*Illness-Causal, Noncausal-Mechanical First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 4.38$, $p = .05$; main effect of hemisphere, $F_{(1,19)} = 1.17$, $p = .29$; condition x hemisphere interaction, $F_{(1,19)} = 17.89$, $p < .001$; **Figure 1A**). Both effects were significant only in the left PC (paired samples t-tests; *Illness-Causal* vs. *Noncausal-Illness First*, left PC: $t_{(19)} = 2.77$, $p = .01$, right PC: $t_{(19)} = 1.28$, $p = .22$; *Illness-Causal* vs. *Noncausal-Mechanical First*, left PC: $t_{(19)} = 3.21$, $p = .005$, right PC: $t_{(19)} = 0.5$, $p = .62$).

We also observed increased activity for illness inferences compared to mechanical inferences in the temporoparietal junction (TPJ) (leave-one-run-out individual-subject fROI analysis; repeated measures ANOVA, condition (*Illness-Causal, Mechanical-Causal*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 5.33$, $p = .03$, main effect of hemisphere, $F_{(1,19)} = 1.02$, $p = .33$, condition x hemisphere interaction, $F_{(1,19)} = 4.24$, $p = .05$; **Supplementary Figure 13**). This effect was significant only in the left TPJ (paired samples t-tests; left TPJ: $t_{(19)} = 2.64$, $p = .02$, right TPJ: $t_{(19)} = 1.13$, $p = .27$). Unlike the PC, the TPJ did not show a preference for illness inferences compared to illness-related language that was not causally connected (repeated measures ANOVA, condition (*Illness-Causal, Noncausal-Illness First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 0.006$, $p = .94$, main effect of hemisphere, $F_{(1,19)} = 2.19$, $p = .16$, condition x hemisphere interaction, $F_{(1,19)} = 1.27$, $p = .27$; repeated measures ANOVA, condition (*Illness-Causal, Noncausal-Mechanical First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 0.73$, $p = .41$; main effect of hemisphere, $F_{(1,19)} = 1.24$, $p = .28$; condition x hemisphere interaction, $F_{(1,19)} = 3.34$, $p = .08$; **Supplementary Figure 13**).

In contrast to the animacy-responsive PC, the anterior PPA showed the opposite pattern, responding more to mechanical inferences than illness inferences (leave-one-run-out individual-subject fROI analysis; repeated measures ANOVA, condition (*Mechanical-Causal, Illness-Causal*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 17.93, p < .001$, main effect of hemisphere, $F_{(1,19)} = 1.33, p = .26$, condition x hemisphere interaction, $F_{(1,19)} = 7.8, p = .01$; **Figure 4A**). This effect was significant only in the left anterior PPA (paired samples t-tests; left anterior PPA: $t_{(19)} = 4, p < .001$, right anterior PPA: $t_{(19)} = 1.88, p = .08$). The anterior PPA also showed a preference for mechanical inferences compared to mechanical-related language that was not causally connected (repeated measures ANOVA, condition (*Mechanical-Causal, Noncausal-Illness First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 14.81, p = .001$, main effect of hemisphere, $F_{(1,19)} = 1.81, p = .2$, condition x hemisphere interaction, $F_{(1,19)} = 7.35, p = .01$; repeated measures ANOVA, condition (*Mechanical-Causal, Noncausal-Mechanical First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 11.31, p = .003$; main effect of hemisphere, $F_{(1,19)} = 3.34, p = .08$; condition x hemisphere interaction, $F_{(1,19)} = 4, p = .06$; **Figure 4A**). Similar to the PC, both effects were larger in the left than in the right hemisphere (post-hoc paired samples t-tests; *Illness-Causal* vs. *Noncausal-Illness First*, left anterior PPA: $t_{(19)} = 3.85, p = .001$, right anterior PPA: $t_{(19)} = 2.22, p = .04$; *Illness-Causal* vs. *Noncausal-Mechanical First*, left anterior PPA: $t_{(19)} = 3.59, p = .002$, right anterior PPA: $t_{(19)} = 1.19, p = .25$).

In summary, we found distinctly localized responses to illness and mechanical causal inferences. Inferring illness causes preferentially recruited the animacy semantic network, particularly the PC.

Illness inferences and mental state inferences elicit spatially dissociable responses

Illness inferences and mental state inferences elicited spatially dissociable responses. In whole-cortex analysis, illness inferences recruited the PC bilaterally, with larger responses observed in the left hemisphere (**Figure 1**, see also fROI analysis showing left-lateralization above). By contrast, and in accordance with prior work (e.g., Saxe & Kanwisher, 2003), mental state inferences recruited a broader network, including not only bilateral PC, but also bilateral TPJ, superior temporal sulcus (STS), and medial and superior prefrontal cortex (**Supplementary Figure 1**).

Within the left PC, responses to illness inferences were located ventrally to mental state inference responses (**Figure 2**, **Supplementary Figure 3**). The z-coordinates of individual-subject activation peaks for illness inferences and mental state inferences were significantly different (repeated measures ANOVA, $F_{(1,19)} = 13.52, p = .002$). In addition, the size of the illness inference effect (*Illness-Causal* > *Mechanical-Causal*) was larger in illness-responsive vertices (leave-one-run-out individual-subject fROI analysis) than in mentalizing-responsive vertices in the left PC (individual-subject fROI analysis; repeated measures ANOVA, $F_{(1,19)} = 24.72, p < .001$, **Supplementary Figure 13**). These results suggest that illness inferences and mental state inferences are carried out by neighboring but partially distinct subsets of the PC.

No univariate evidence for domain-general responses to implicit causal inference

Prior neuroscience studies hypothesizing the existence of a domain-general ‘causal engine’ have predicted that the language network and/or domain-general executive systems (e.g., the logic network) should show elevated activity during causal inference across domains. In the current study, neither the language nor the logic network exhibited elevated neural responses during causal inferences relative to linguistically matched sentence pairs that were not causally connected. Language regions in frontotemporal cortex responded more to noncausal than causal

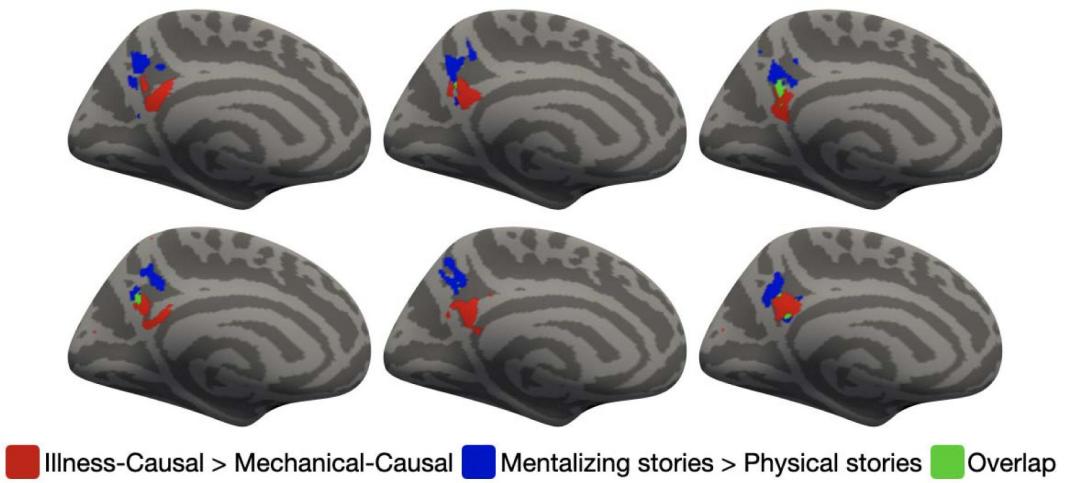


Figure 2.

Spatial dissociation between univariate responses to illness inferences and mental state inferences in the precuneus (PC). The left medial surface of 6 individual participants were selected for visualization purposes. The locations of the top 10% most responsive vertices to *Illness-Causal* > *Mechanical-Causal* in a PC search space (Dufour et al., 2013 [↗](#)) are shown in red. The locations of the top 10% most responsive vertices to *mentalizing stories* > *physical stories* (mentalizing localizer) in the same PC search space are shown in blue. Overlapping vertices are shown in green.

vignettes (frontal search space: repeated measures ANOVA, $F_{(1,19)} = 23.91, p < .001$; temporal search space: repeated measures ANOVA, $F_{(1,19)} = 4.31, p = .05$; **Figure 3**, **Supplementary Figure 8**). The logic network likewise responded marginally more to noncausal vignettes, likely reflecting greater difficulty associated with integrating unrelated sentences (repeated measures ANOVA, $F_{(1,19)} = 3.88, p = .07$; **Figure 3**).

In whole-cortex univariate analysis, no shared regions responded more to causal than noncausal vignettes across domains. Two whole-cortex univariate contrasts comparing causal and noncausal conditions (*Illness-Causal* > *Noncausal-Mechanical First*, *Mechanical-Causal* > *Noncausal-Mechanical First*) revealed increased activity for the noncausal condition in bilateral prefrontal cortex. The same prefrontal areas that responded more to noncausal than causal stimuli also responded more when participants were slower to complete the task, suggesting that these responses reflect a non-specific difficulty effect (**Supplementary Figure 4**).

In summary, none of the predicted networks nor any regions across the whole cortex exhibited the predicted domain-general causal inference pattern, i.e., larger responses to all causal than all noncausal vignettes. These results suggest that implicit causal inferences, which draw upon a person's existing knowledge of relevant causes and effects, do not depend on domain-general neural mechanisms. These results leave open the possibility that domain-general systems support the explicit search for causal connections (see Discussion section).

Multivariate analysis

In searchlight MVPA performed across the whole cortex, illness inferences and mechanical inferences produced spatially distinguishable neural patterns in the left PC extending dorsally into the superior parietal lobule, as well as in left anterior PPA and lateral occipitotemporal cortex. A whole-cortex searchlight analysis that tested whether each causal condition could be decoded from each noncausal condition found no shared regions that exhibited significant decoding across all causal vs. noncausal comparisons (**Supplementary Figure 9**).

In individual-subject fROI decoding analyses, illness inferences and mechanical inferences produced spatially distinguishable neural patterns in the left PC, right PC, and left TPJ, as well as in language and logic networks (see **Supplementary Figure 10**, **Supplementary Table 2** for full results). Note that these decoding results must be interpreted in light of the significant univariate differences observed across conditions that are reported above. Linear classifiers are highly sensitive to univariate differences (Coutanche, 2013; Kragel et al., 2012; Hebart & Baker, 2018; Woolgar et al., 2014; Davis et al., 2014; Pakravan et al., 2022). Successful decoding may be driven by univariate differences in the predicted direction (e.g., causal > noncausal) or in the opposite direction (e.g., noncausal > causal). In particular, given that both the language and the logic networks exhibited higher univariate responses to noncausal compared to causal vignettes, decoding results observed in these networks may be driven by univariate differences.

Discussion

Causal knowledge is embedded in higher-order semantic networks

We find that a semantic network previously implicated in thinking about animates, particularly the precuneus (PC), is preferentially engaged when people infer causes of illness compared to when they infer causes of mechanical breakdown or read causally unconnected sentences containing illness-related language. By contrast, mechanical inferences activate an anterior parahippocampal region previously implicated in thinking about and remembering places (Baldassano et al., 2013; Fairhall et al., 2014; Silson et al., 2019; Steel et al., 2021; Häusler et al., 2022; Hauptman, Elli, et al., 2025). This finding points to a neural double dissociation between biological and mechanical causal knowledge.

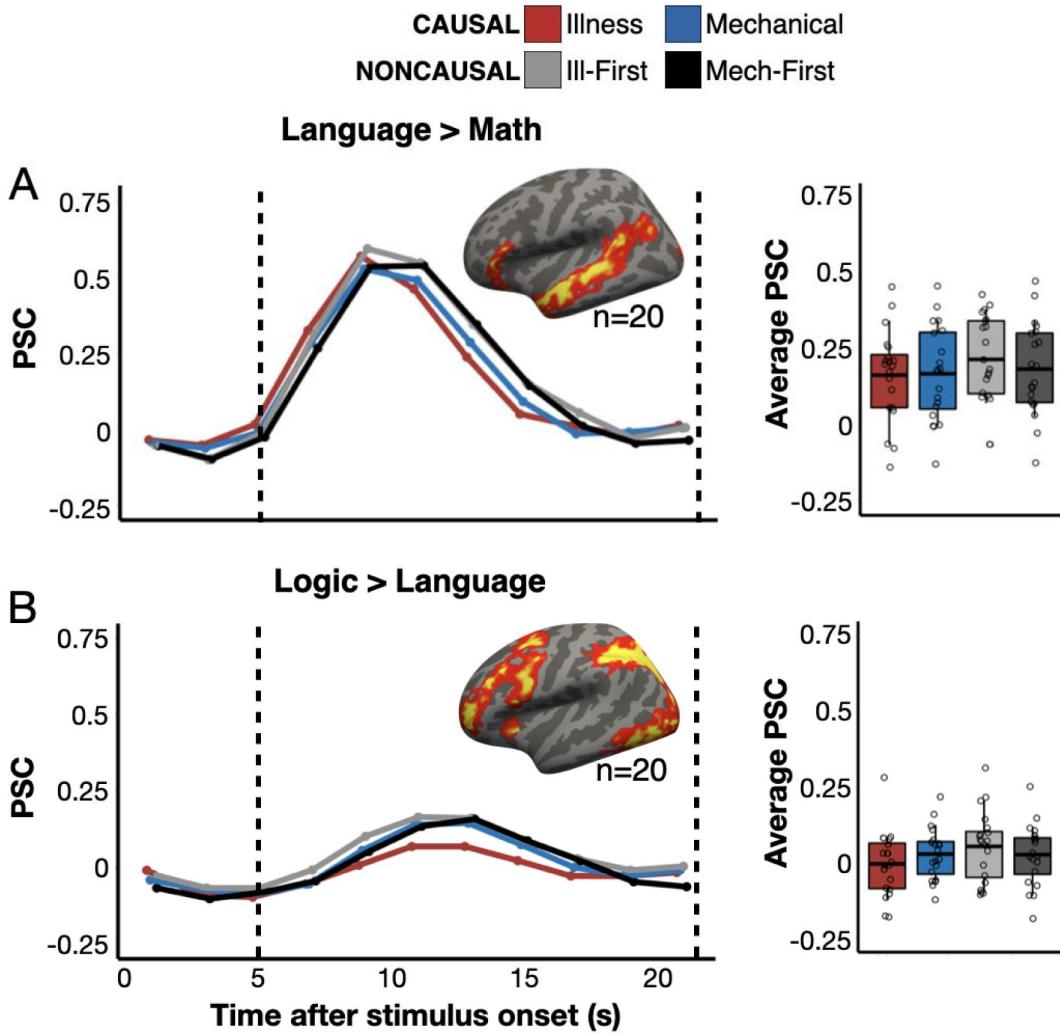


Figure 3.

Individual-subject analysis of language- and logic-responsive vertices. Panel A: percent signal change (PSC) for each condition among the top 5% most language-responsive vertices (*language > math*) in a temporal language network search space (Fedorenko et al., 2010 [🔗](#)). Results from a frontal language search space (Fedorenko et al., 2010 [🔗](#)) can be found in **Supplementary Figure 8** [🔗](#). Panel B: PSC among the top 5% most logic-responsive vertices (*logic > language*) in a logic network search space (Liu et al., 2020 [🔗](#)). Group maps for each contrast of interest (one-tailed) are corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$. Boxplots display average PSC in the critical window (marked by dotted lines) across participants. The horizontal line within each boxplot indicates the overall mean.

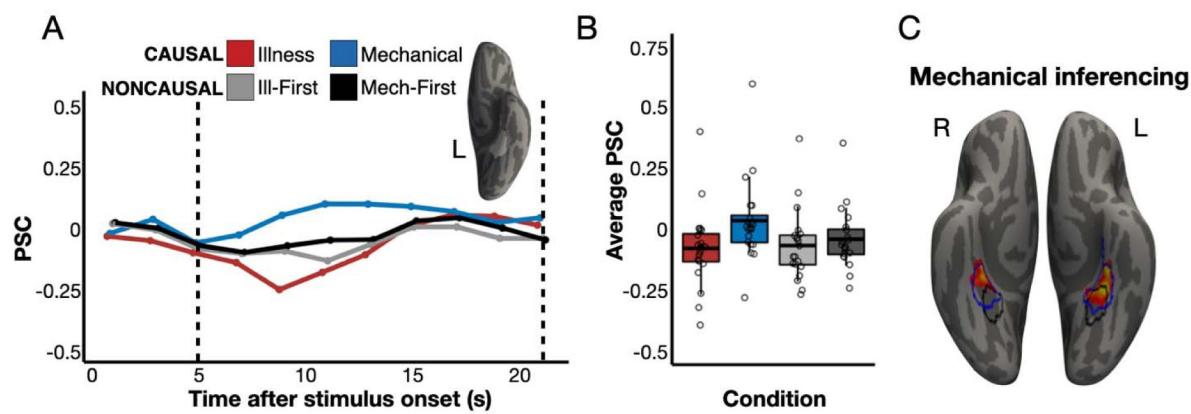


Figure 4.

Responses to mechanical inferences in anterior parahippocampal regions (anterior PPA). Panel A: Percent signal change (PSC) for each condition among the top 5% *Mechanical-Causal > Illness-Causal* vertices in a left anterior PPA search space (Hauptman, Elli, et al., 2025 [2](#)) in individual participants, established via a leave-one-run-out analysis. Panel B: Average PSC in the critical window (marked by dotted lines in Panel A) across participants. The horizontal line within each boxplot indicates the overall mean. Panel C: The intersection of two whole-cortex contrasts (one-tailed), *Mechanical-Causal > Illness-Causal* and *Mechanical-Causal > Noncausal* that are corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$. Similar to PC responses to illness inferences, anterior PPA is the only region to emerge across both mechanical inference contrasts. The average PPA location from separate study involving perceptual place stimuli (Weiner et al., 2018 [3](#)) is overlaid in black. The average PPA location from separate study involving verbal place stimuli (Hauptman, Elli, et al., 2025 [2](#)) is overlaid in blue.

Previous work has implicated the PC in the representation of animate entities, i.e., people and animals (Fairhall & Caramazza, 2013a [🔗](#), 2013b [🔗](#); Fairhall et al., 2014 [🔗](#); Peer et al., 2015 [🔗](#); Wang et al., 2016 [🔗](#); Silson et al., 2019 [🔗](#); Rabini, Ubaldi, & Fairhall, 2021 [🔗](#); Deen & Freiwald, 2022 [🔗](#); Aglinskas & Fairhall, 2023 [🔗](#); Hauptman, Elli, et al., 2025 [🔗](#)). Here we show that the PC exhibits sensitivity to causal inferences about biological processes specific to animates, such as illness.

These findings are consistent with our preregistered hypotheses and suggest that causal knowledge about animate and inanimate entities is distributed across multiple distinct semantic networks. Further, our results suggest that the animacy semantic network supports biological causal knowledge. Future work should test whether the animacy network is sensitive to causal information beyond illness, including about growth, birth, and death. We hypothesize that changes in biological causal knowledge during development as well as cultural expertise in causal reasoning about illness (e.g., medical expertise) influences activity in the animacy network (Legare et al., 2012 [🔗](#); Norman et al., 2009 [🔗](#)).

Our findings are consistent with prior evidence from naturalistic paradigms showing that the PC is sensitive to discourse-level processes across sentences (e.g., Hasson et al., 2008 [🔗](#); Lerner et al., 2011 [🔗](#); Lee & Chen, 2022 [🔗](#)). We hypothesize that PC responses observed during naturalistic narrative comprehension are driven by causal inferences about animate agents, who are often the focus of narratives. Likewise, PC involvement in episodic memory could be related to animacy-related inferential processes (DiNicola, Braga, & Buckner, 2020 [🔗](#); Ritchey & Cooper, 2020 [🔗](#)). Future work can test this hypothesis by comparing causal inferences about animate and inanimate entities in naturalistic contexts, such as films and verbal narratives (see Chen & Bornstein, 2024 [🔗](#) for a review on causal inference in narrative comprehension).

We find that neural responses during inferences about biological and mental properties of animates are linked yet separable. Inferring illness causes recruits neural circuits that are adjacent to but distinct from responses to mental state inferences in the PC (Saxe & Kanwisher, 2003 [🔗](#); Saxe et al., 2006 [🔗](#)). Even young children provide different causal explanations for biological vs. psychological processes (Springer & Keil, 1991 [🔗](#); Callanan & Oakes, 1992 [🔗](#); Wellman & Gelman, 1992 [🔗](#); Inagaki & Hatano, 1993 [🔗](#); 2004 [🔗](#); Keil, 1994 [🔗](#); Hickling & Wellman, 2001 [🔗](#); Medin et al., 2010 [🔗](#); cf. Carey, 1985 [🔗](#); see also Medin & Atran, 2004 [🔗](#)). For example, when asked why blood flows to different parts of the body, 6-year-olds endorse explanations referring to bodily function, e.g., “because it provides energy to the body,” and not to mental states, e.g., “because we want it to flow” (Inagaki & Hatano, 1993 [🔗](#)). At the same time, animate entities have a dual nature: they have both bodies and minds (Opfer & Gelman, 2011 [🔗](#); Spelke, 2022 [🔗](#)). The current findings point to the existence of distinct but related neural systems for biological and mentalistic knowledge.

Our neuroimaging findings are consistent with evidence from developmental psychology suggesting that causal knowledge is central to human concepts starting early in development (Keil, 1992 [🔗](#); Wellman & Gelman, 1992 [🔗](#); Hatano & Inagaki, 1994 [🔗](#); Springer & Keil, 1991 [🔗](#); Simons & Keil, 1995 [🔗](#); Atran, 1998 [🔗](#); Keil et al., 1999 [🔗](#); Coley, Solomon, & Shafto, 2002 [🔗](#); Medin & Atran, 2004 [🔗](#)). According to the ‘intuitive theories’ account, semantic knowledge is organized into causal frameworks that serve as ‘grammars for causal inference’ (Tenenbaum et al., 2007 [🔗](#); Wellman & Gelman, 1992 [🔗](#); Gopnik & Meltzoff, 1997 [🔗](#); Gopnik & Wellman, 2012 [🔗](#); Gerstenberg & Tenenbaum, 2017 [🔗](#); see also Boyer 1995 [🔗](#); Barrett, Cosmides, & Tooby, 2007 [🔗](#); Cosmides & Tooby, 2013 [🔗](#); Bender, Beller, & Medin, 2017 [🔗](#)). For example, preschoolers intuit that animates but not inanimate objects get sick and need nourishment to grow and live (e.g., Rosengren et al., 1991 [🔗](#); Kalish, 1996 [🔗](#); Gutheil, Vera, & Keil, 1998 [🔗](#); Raman & Gelman, 2005 [🔗](#); see Inagaki & Hatano, 2004 [🔗](#); Opfer & Gelman, 2011 [🔗](#) for reviews). The present results suggest that such knowledge is encoded in higher-order semantic brain networks. By contrast, we failed to find sensitivity to causal inference in portions of the ventral stream previously associated with the perception of animate agents (see Appendix 4 [🔗](#), Supplementary Figure 8 [🔗](#) for details).

Sensitivity to causal information may be a distinguishing characteristic of higher-order, amodal semantic networks, as opposed to perceptual regions that are activated during semantic tasks (e.g., Martin & Chao, 2001 [\(2\)](#); Thompson-Schill, 2003 [\(2\)](#); Barsalou et al., 2003 [\(2\)](#); Binder & Desai, 2011 [\(2\)](#); Bi, 2021 [\(2\)](#)).

No evidence for domain-general neural responses during implicit causal inference

In the current study, participants read two sentence vignettes that either elicited causal inferences or were not causally connected. No brain regions responded more to causal inferences across domains compared to noncausal vignettes in this task. The language network responded more to noncausal than causal vignettes, possibly due to greater difficulty associated with processing the meaning of a sentence that does not follow from the prior context. Prior studies find that the language network is specialized primarily for sentence-internal processing (Fedorenko & Varley, 2016 [\(2\)](#); Jacoby & Fedorenko, 2020 [\(2\)](#); Blank & Fedorenko, 2020 [\(2\)](#)) and patients with agrammatic aphasia can make causal inferences about pictorial stimuli (Varley & Siegal, 2000 [\(2\)](#); Varley, 2014 [\(2\)](#)). Together, these results suggest that the language system itself is unlikely to support implicit causal inference. Rather, during language comprehension, the language system interacts with semantic networks to enable such inferences (Simony et al., 2016 [\(2\)](#); Yeshurun et al., 2018; Chang et al., 2022 [\(2\)](#)). Notably, in the current study, responses to causal inference in semantic networks were stronger in the left hemisphere. The left lateralization of such responses may enable efficient interfacing with the language system during comprehension.

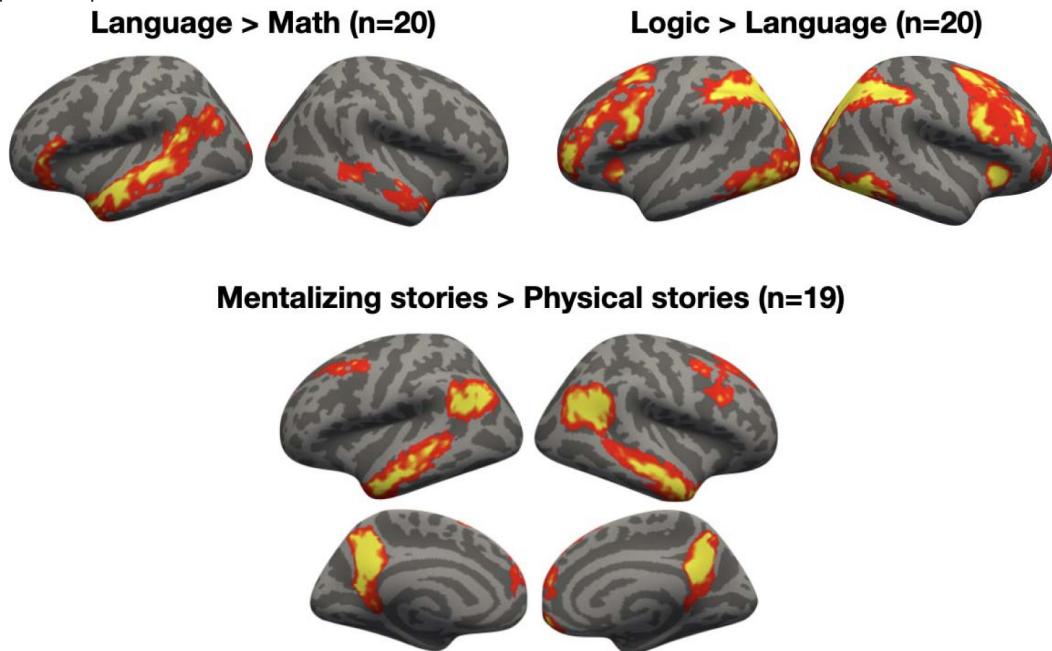
We also failed to find evidence for the claim that the frontoparietal logical reasoning network, a domain-general executive system, supports implicit causal inferences. By contrast, the frontoparietal network responded more to noncausal than causal vignettes. Finally, we failed to observe elevated responses to causal inference across domains anywhere in the brain in whole-cortex analysis. A large swath of prefrontal cortex responded more to one noncausal condition (*Noncausal-Mechanical First*) compared to both causal conditions. The same prefrontal regions also exhibited increased activity when participants were slower to respond to the task. Thus, this ‘reverse causality effect’ likely reflects processing demands rather than causal inference per se. An alternative interpretation of elevated prefrontal activity observed for one of the noncausal conditions is that it reflects the effortful search for a causal connection between sentences when such a connection is difficult to find. This interpretation would suggest that domain-general executive mechanisms become engaged when causal inferences are effortful and explicit. By contrast, semantic systems are engaged when we implicitly infer a known causal relationship.

Causal inferences are a highly varied class, and domain-general systems likely play an important role in many causal inferences not tested in the current study. The vignettes used in the current study stipulate illness causes, allowing participants to reason from causes to effects. By contrast, illness reasoning performed by medical experts proceeds from effects to causes and can involve searching for potential causes within highly complicated and interconnected causal systems (Schmidt, Norman, & Boshuizen, 1990 [\(2\)](#); Norman et al., 2009 [\(2\)](#); Meder & Mayrhofer, 2017 [\(2\)](#)). The discovery of novel causal relationships (e.g., ‘blicket detectors’; Gopnik et al., 2001 [\(2\)](#)) and the identification of complex causes, even in the case of illness, may depend in part on domain-general neural mechanisms. The present results suggest, however, that causal knowledge is embedded within higher-order semantic systems, and that biological causal knowledge is embedded with a semantic system relevant to animacy.

Supplementary Figures

Supplementary Figure 1.

Functional localization of language, logical reasoning, and mentalizing networks (see Monti et al., 2009 [2](#); Fedorenko et al., 2010 [3](#); Dodell-Feder et al., 2011 [4](#); Liu et al., 2020 [5](#)). Group maps for each contrast of interest (one-tailed) are corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$.



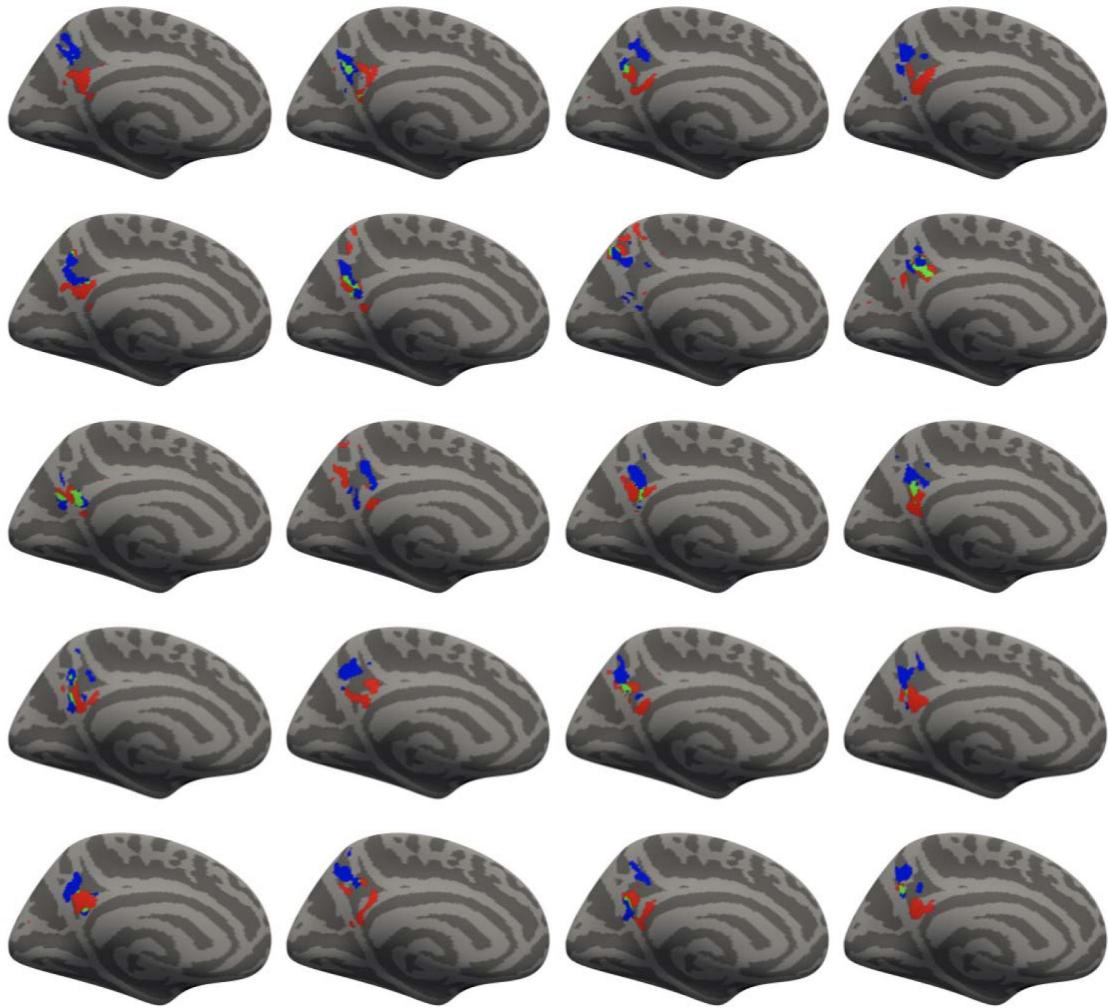
Supplementary Figure 2.

Overlap between left precuneus (PC) responses to illness inferences in the current study and people-related stimuli in a separate study (Fairhall & Caramazza, 2013b [6](#)). The average location from a separate study comparing people and place concepts (Fairhall & Caramazza, 2013b [6](#)) is overlaid in blue on the response to illness inferences observed in the current study. The group map (one-tailed) is corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$.

Illness-Causal > Mechanical-Causal

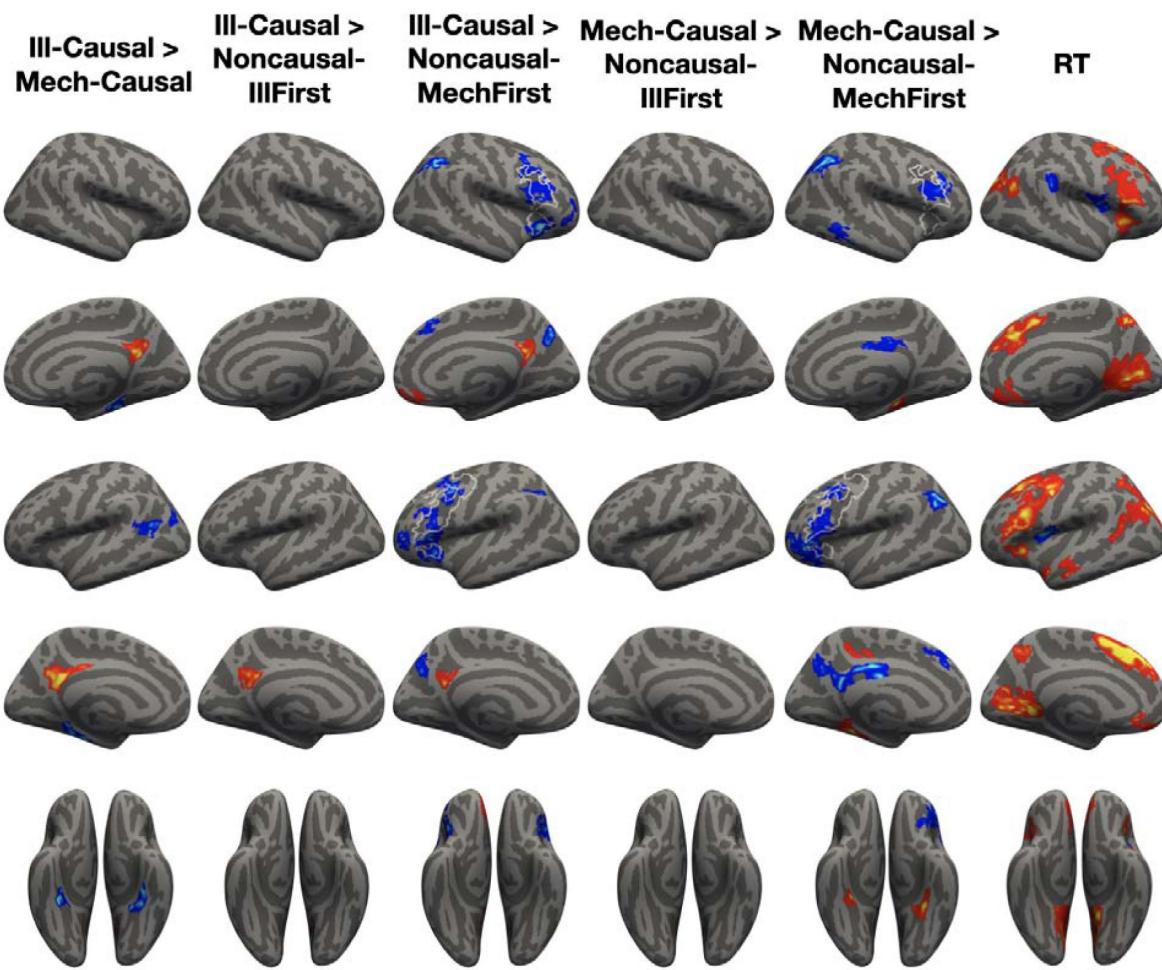


■ Illness-Causal > Mechanical-Causal ■ Mentalizing stories > Physical stories ■ Overlap



Supplementary Figure 3.

Spatial dissociation between responses to illness inferences and mental state inferences in left precuneus (PC). The left medial surface of all participants ($n=20$) is shown. The locations of the top 10% most responsive vertices to *Illness-Causal > Mechanical-Causal* in a PC search space (Dufour et al., 2013 [eLife](#)) are shown in red. The locations of the top 10% most responsive vertices to *mentalizing stories > physical stories* (mentalizing localizer) in the same PC search space are shown in blue. Overlapping vertices are shown in green.

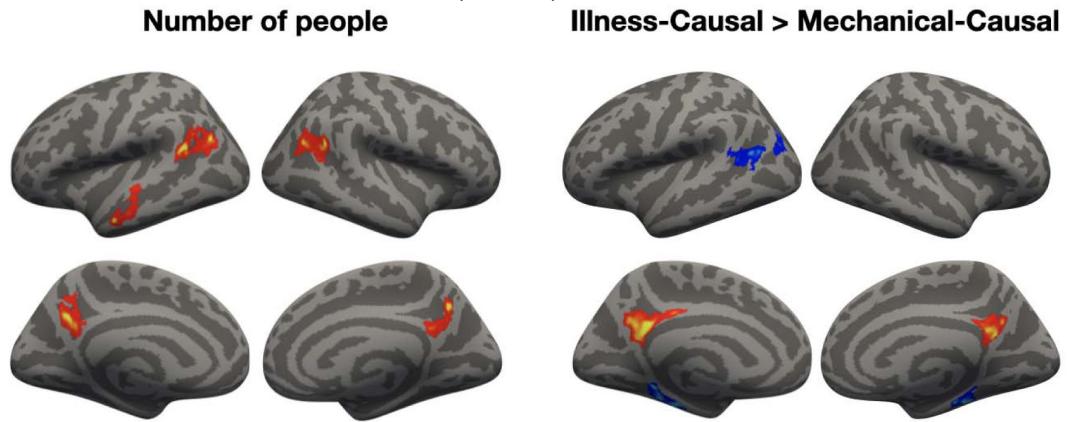


Supplementary Figure 4.

Full whole-cortex univariate results. Regions whose activity scales with response time (RT) are displayed under "RT." Frontal RT regions are outlined in white on the lateral surface for other contrasts where frontal effects are observed. Group maps (two-tailed) are corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$.

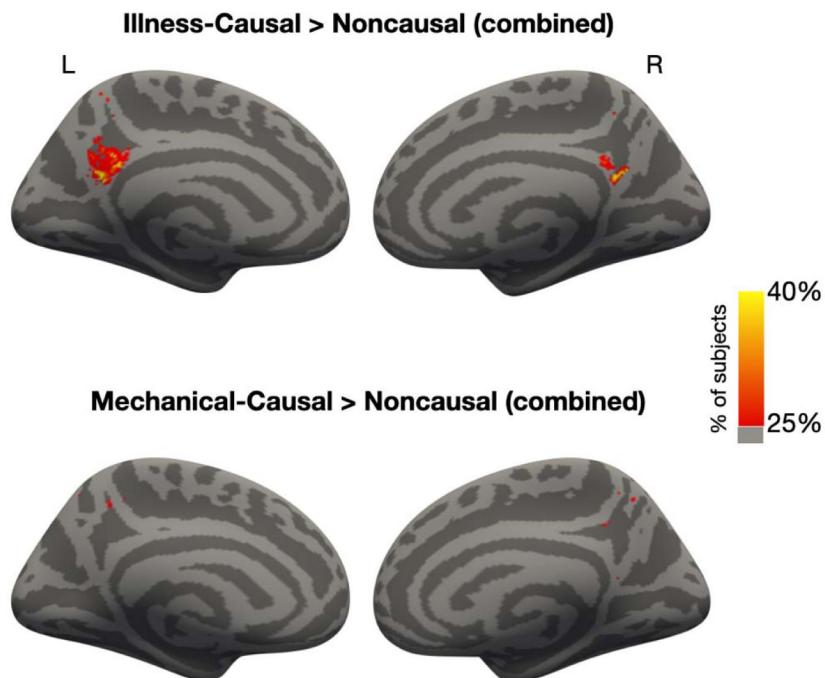
Supplementary Figure 5.

Comparison of whole-cortex results for number of people (left) and illness inferences (right) from the same GLM. Group maps (two-tailed) are corrected for multiple comparisons ($p < .05$ FWER, cluster-forming threshold $p < .01$ uncorrected). Vertices are color coded on a scale from $p=0.01$ to $p=0.00001$.



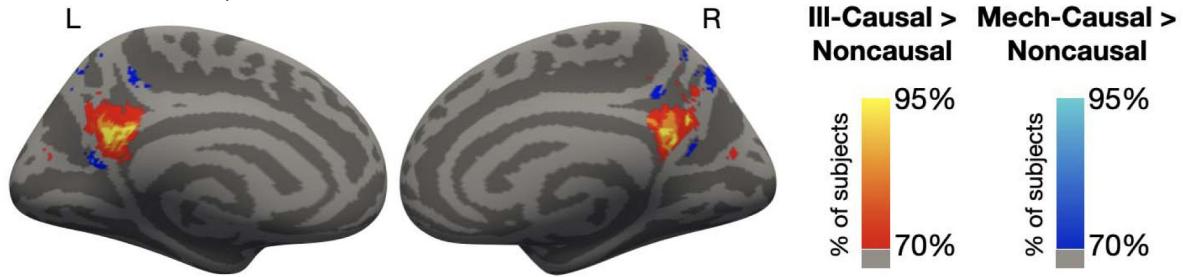
Supplementary Figure 6.

Group overlap in univariate contrasts comparing causal (*Illness-Causal, Mechanical-Causal*) and noncausal conditions (*Noncausal-Illness First + Noncausal-Mechanical First*) in the PC. Each vertex in a PC search space (Dufour et al., 2013 [\[2\]](#)) was color-coded according to the proportion of participants who showed significant activation ($p < .05$ uncorrected) at that location.



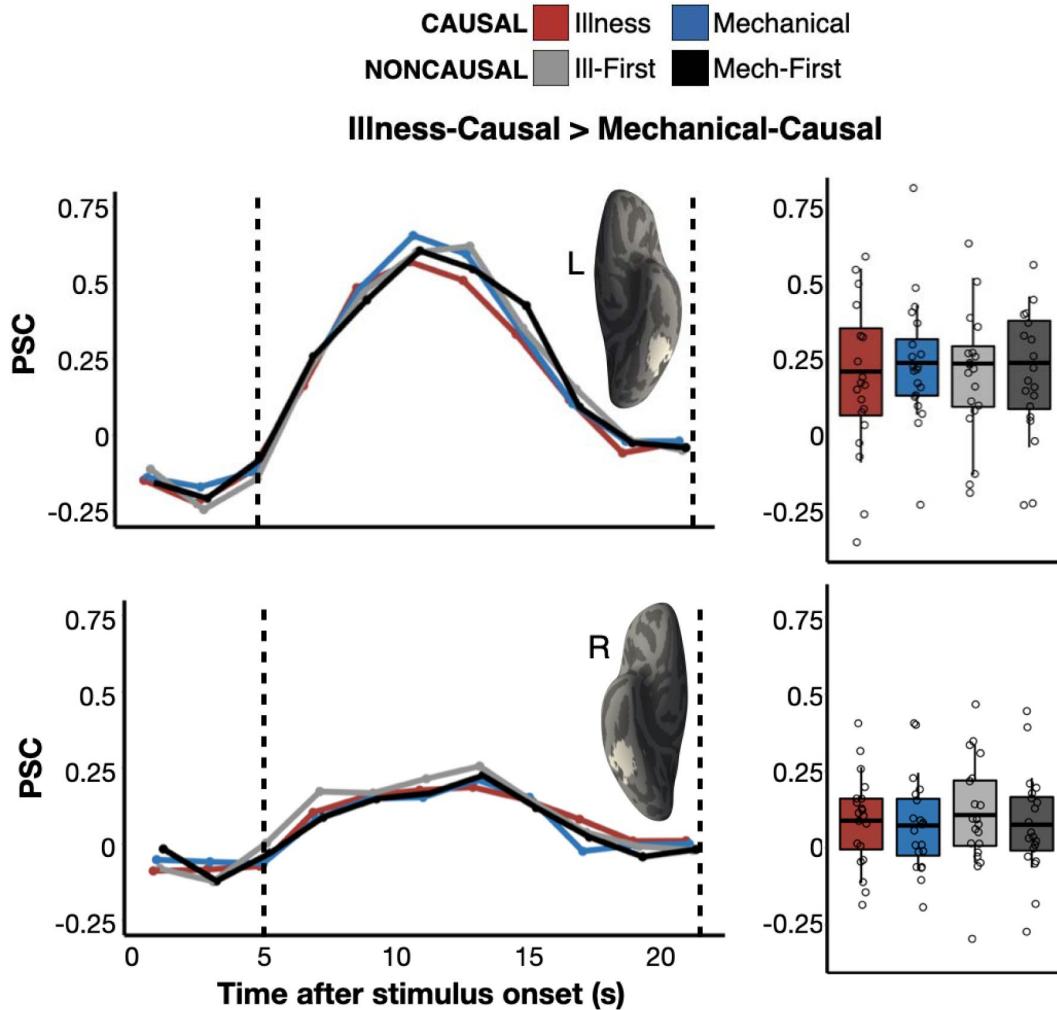
Supplementary Figure 7.

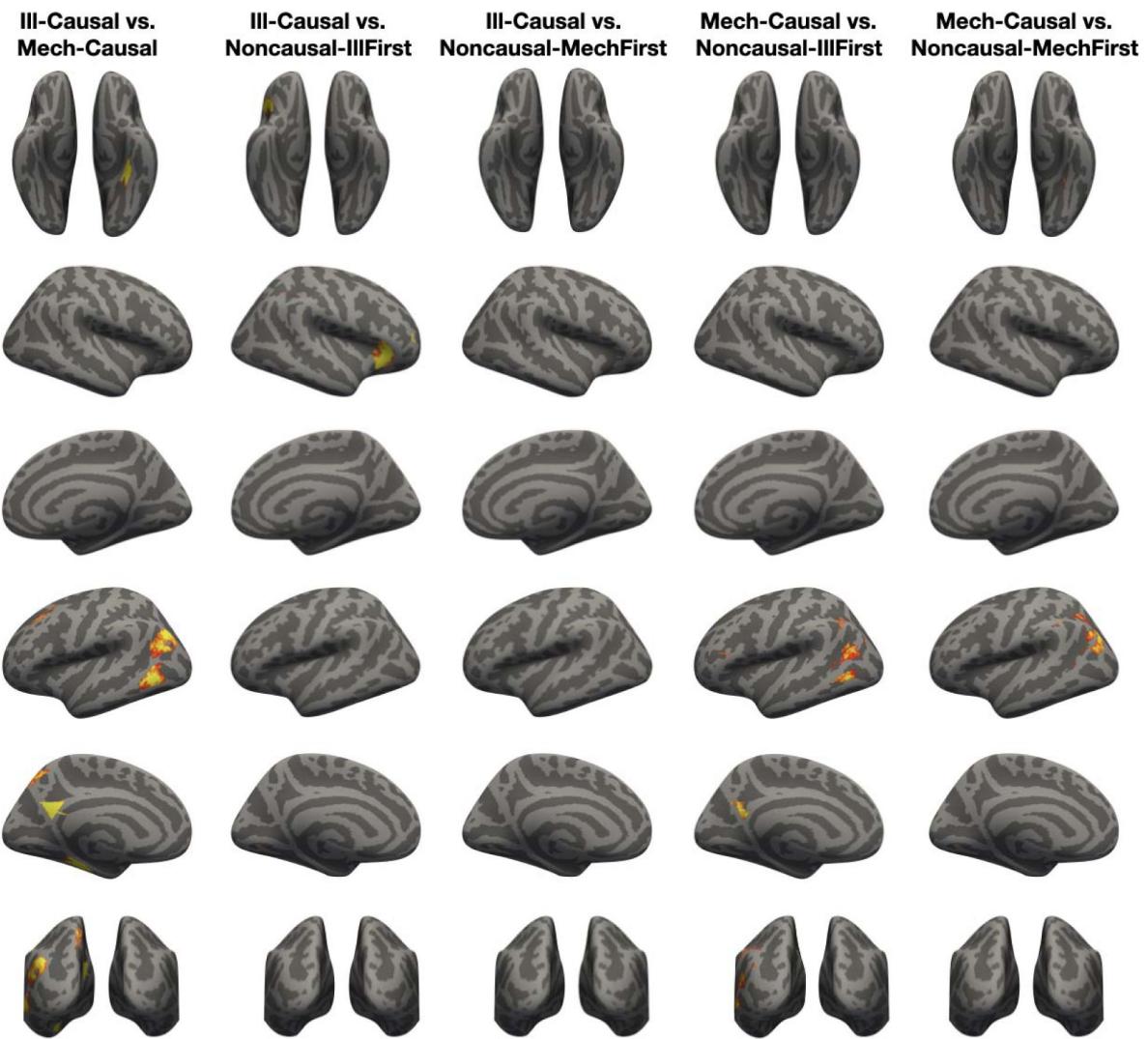
Group overlap in univariate contrasts comparing causal (*Illness-Causal*, *Mechanical-Causal*) and noncausal conditions (*Noncausal-Illness First* + *Noncausal-Mechanical First*) in the PC, winner-take-all approach. Each vertex in a PC search space (Dufour et al., 2013 [2](#)) was color-coded according to the proportion of participants who showed a preference for *Illness-Causal* > *Noncausal* compared to *Mechanical-Causal* > *Noncausal* (red) and vice versa (blue) at that location.



Supplementary Figure 8.

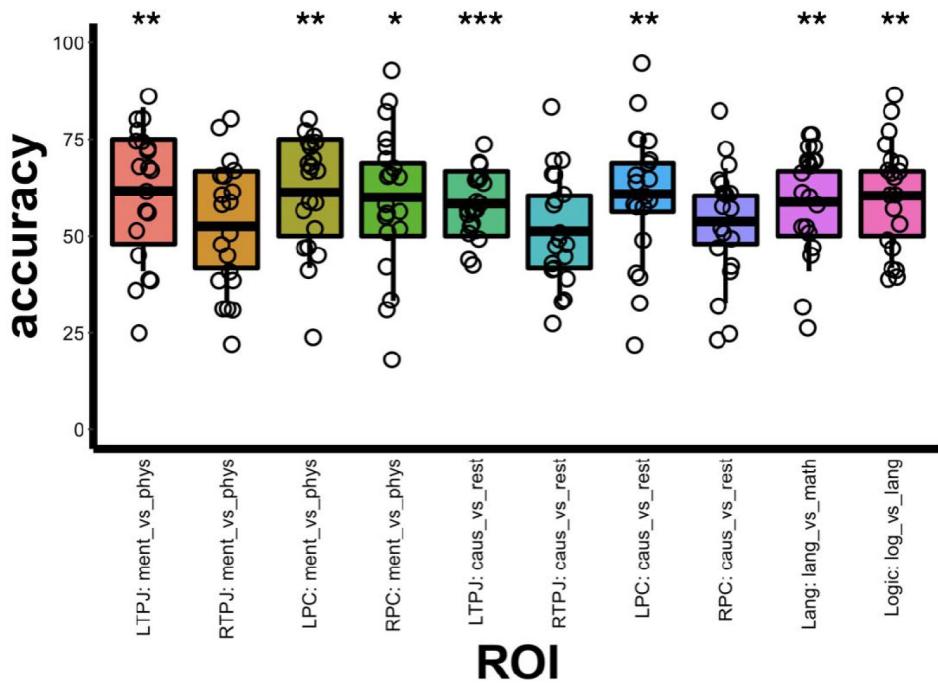
Responses to illness inferences in the fusiform face area (FFA). Percent signal change (PSC) for each condition among the top 5% *Illness-Causal* > *Mechanical-Causal* vertices in left and right FFA search spaces (Julian et al., 2012 [2](#)) in individual participants, established via a leave-one-run-out analysis. Average PSC in the critical window (marked by dotted lines in Panel A) across participants is displayed via boxplot. The horizontal line within each boxplot indicates the overall mean.





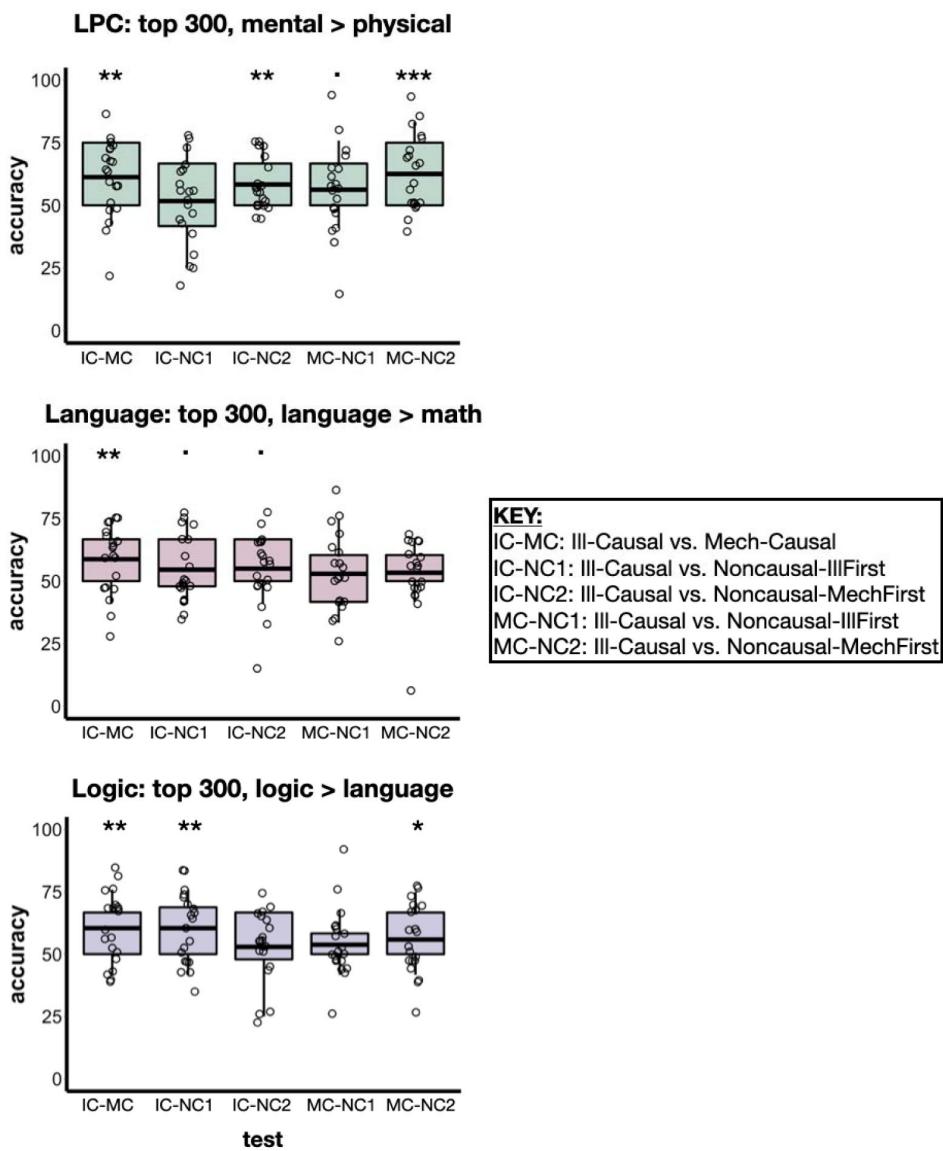
Supplementary Figure 9.

Searchlight MVPA group maps. Whole-brain searchlight maps were thresholded using a vertex-wise threshold ($p < .001$ uncorrected) and a cluster size threshold (FWER $p < .05$, corrected for multiple comparisons across the entire cortical surface). Vertices are color coded on a scale from 55-65% decoding accuracy.



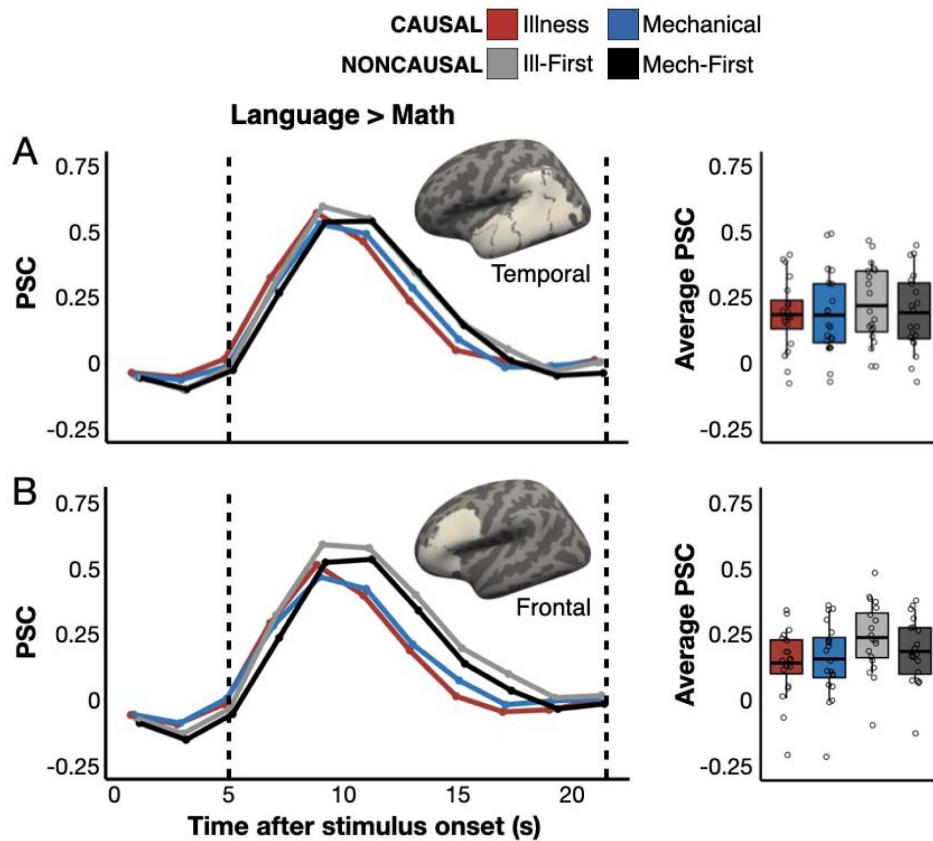
Supplementary Figure 10.

Subject dispersion data for individual-subject MVPA performed in functional ROIs. We tested whether patterns of activity elicited during illness inferences vs. mechanical inferences could be decoded in each fROI: left and right PC, left and right TPJ, language network, logic network. In accordance with our preregistration, 2 types of fROIs were constructed using PC and TPJ search spaces: 1) top 300 most active vertices for mentalizing stories compared to physical stories in the mentalizing/animacy localizer (*Mentalizing stories > Physical stories*), and 2) top 300 most active vertices for both causal conditions compared to rest (*Illness-Causal + Mechanical-Causal > Rest*) Chance: 50%. Significance codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 '' 1. Full statistical results are included in **Supplementary Table 2**.



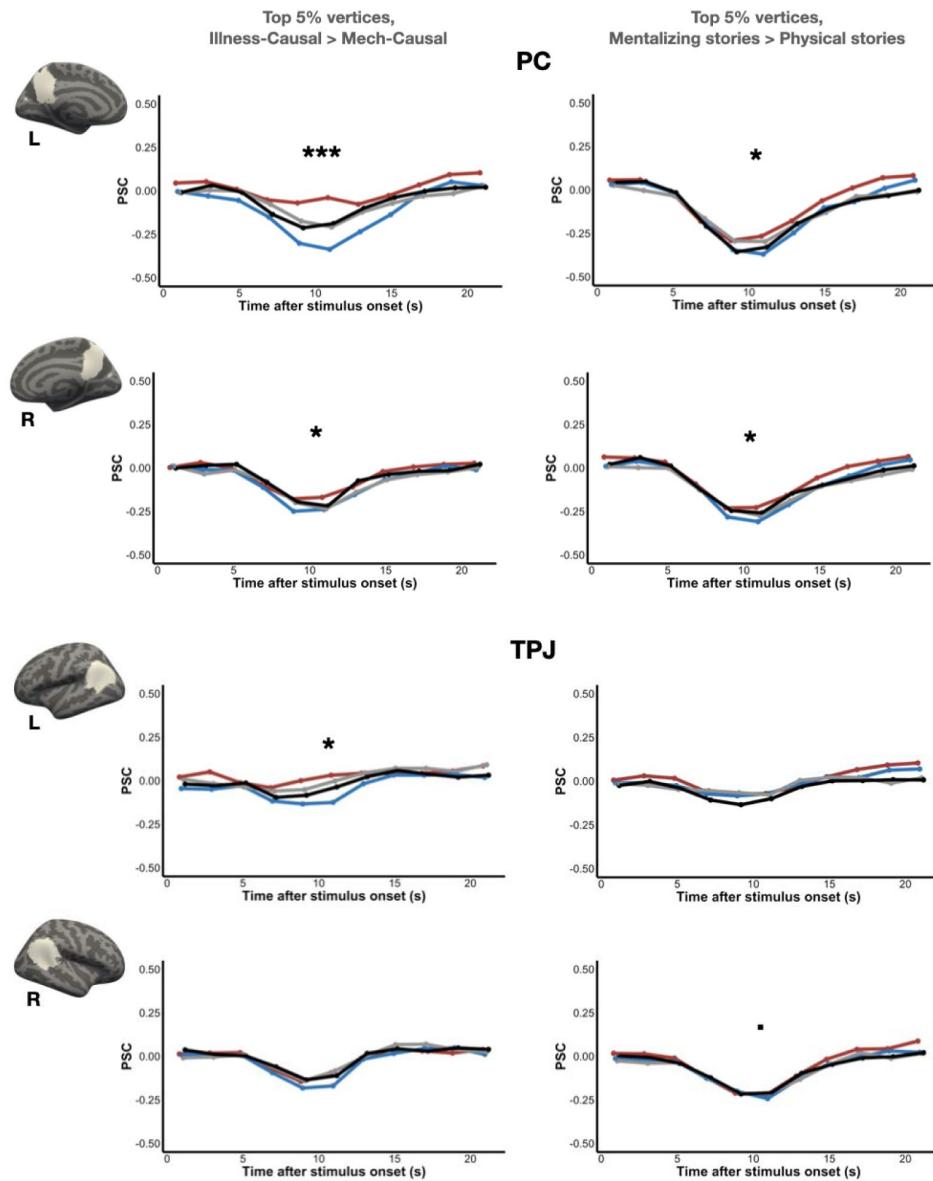
Supplementary Figure 11.

Subject dispersion data for individual-subject MVPA performed in functional ROIs. 5 tests were performed in each fROI: left PC (LPC), language network, logic network. Chance: 50%. Significance codes: 0 '****' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 '' 1. Full statistical results are included in **Supplementary Table 3**.



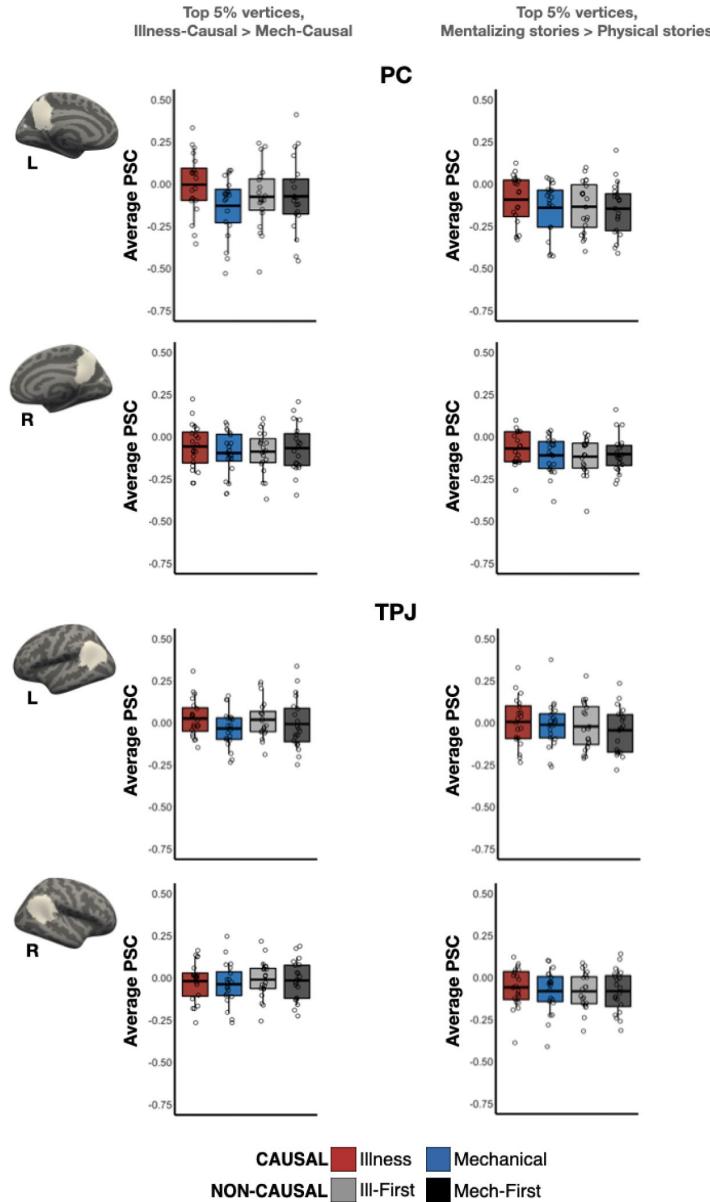
Supplementary Figure 12.

Responses to causal inference in the language network. Panel A: Percent signal change (PSC) for each condition among the top 5% most language-responsive vertices (*language > math*) in a temporal language network search space (Fedorenko et al., 2010 [🔗](#)). Panel B: The same results in a frontal language search space (Fedorenko et al., 2010 [🔗](#)). Boxplots display average PSC in the critical window (marked by dotted lines) across participants. The horizontal line within each boxplot indicates the overall mean.



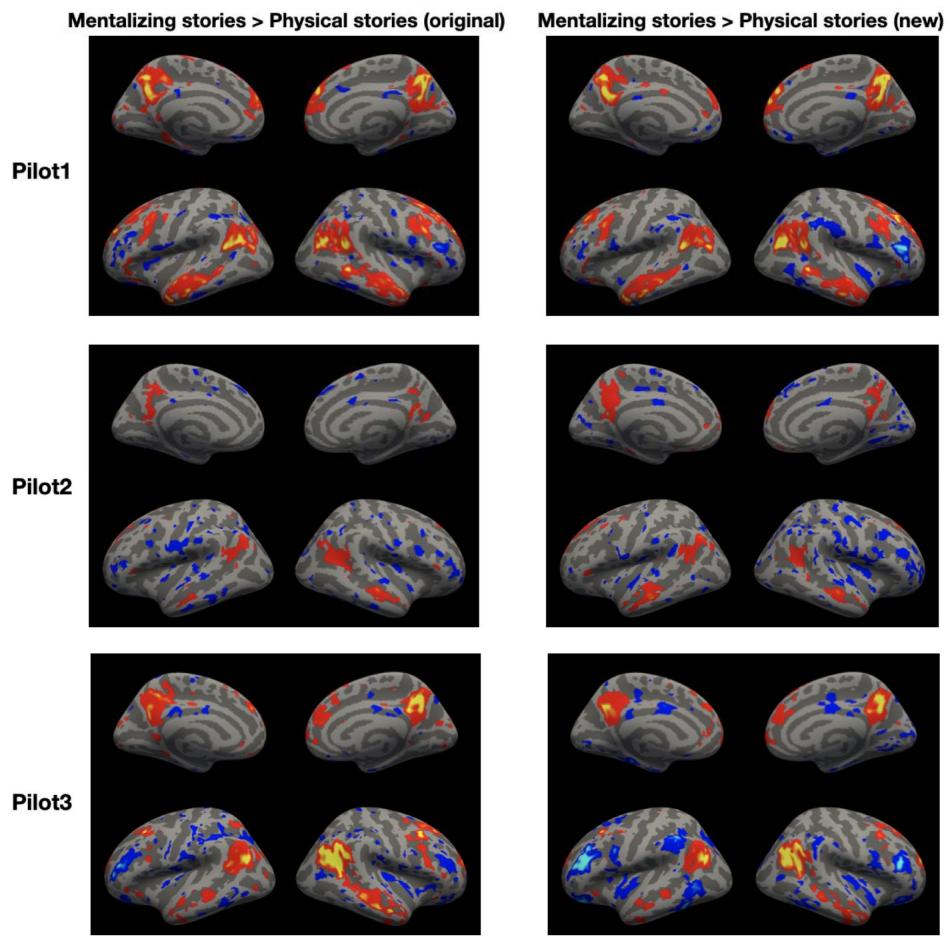
Supplementary Figure 13.

Responses to illness inferences in bilateral PC and TPJ. Percent signal change (PSC) for each condition among the top 5% *Illness-Causal* > *Mechanical-Causal* vertices in bilateral PC and TPJ search spaces (Dufour et al., 2013 [2](#)) in individual participants, established via a leave-one-run-out analysis, is shown. We hypothesized that the PC and TPJ would exhibit a preference for illness inferences and report all data for completeness (see preregistration <https://osf.io/6pnqg> [2](#)). Significance codes for *Illness-Causal* > *Mechanical-Causal* comparison (paired samples t-tests): 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 '' 1. Subject dispersion data are shown in **Supplementary Figure 14** [2](#).



Supplementary Figure 14.

Subject dispersion data for responses to illness inferences in bilateral PC and TPJ (see [Supplementary Figure 13](#)). We hypothesized that the PC and TPJ would exhibit a preference for illness inferences and report all data for completeness (see preregistration <https://osf.io/6pnqg/>). Boxplots display average PSC in the critical window (5-21 s) across participants. The horizontal line within each boxplot indicates the overall mean.



Supplementary Figure 15.

Comparison of mentalizing localizers used in previous work and in the current study, in 3 pilot participants. The mentalizing localizer in the current study used the same mentalizing stories as in previous work (Dodell-Feder et al., 2011 [28](#)) but contained new physical stories that included more vivid physical description and did not refer to animate agents. Individual-subject maps are shown at $p < .01$ uncorrected.

Supplementary Materials

Appendix 1: Online experiment protocol.

Prior to the fMRI experiment, we collected explicit causality judgments from a separate group of online participants ($n=30$). Each online participant read all vignettes from the causal inference experiment (152 vignettes) in addition to 12 filler vignettes that were designed to be either maximally causally related or unrelated (164 vignettes total), one vignette at a time. Their task was to judge the extent to which it was possible that the event described in the first sentence of each vignette caused the event described in the second sentence on a 4-point scale (1 = not possible; 4 = very possible). 4 participants were excluded on the basis of inaccurate responses on the filler trials (i.e., difference between average ratings for maximally causally related and maximally causally unrelated vignettes >2). Among the 26 remaining participants, 12 read vignettes from Version A and 14 read vignettes from Version B of the experiment. To eliminate erroneous responses, we first excluded trials with RTs 2.5 SD outside their respective condition means within participants and then excluded trials with outlier RTs (more than 1.5 IQR below Q1 or more than 1.5 IQR above Q3) across participants (approximately 5% of all trials excluded in total). We found that both causal conditions (*Illness-Causal*, *Mechanical-Causal*) were more causally connected than both noncausal conditions, $t(25) = 36.97$, $p < .001$ (causal: $M = 3.51 \pm 0.78$ SD, noncausal: $M = 1.10 \pm 0.45$ SD). In addition, *Illness-Causal* and *Mechanical-Causal* items received equally high causality ratings, $t(25) = -0.64$, $p = .53$ (*Illness-Causal*: $M = 3.49 \pm 0.77$ SD, *Mechanical-Causal*: $M = 3.53 \pm 0.79$ SD).

Appendix 2: Details on measuring linguistic variables.

All conditions were matched (pairwise t-tests, all $p > 0.3$, no statistical correction) on multiple linguistic variables known to modulate neural activity in language regions (e.g., Pallier, Devauchelle, & Dehaene, 2011 [\(2\)](#); Shain, Blank et al., 2020 [\(2\)](#)). These included number of characters, number of words, average number of characters per word, average word frequency, average bigram surprisal (Google Books Ngram Viewer, <https://books.google.com/ngrams/> [\(2\)](#)), and average syntactic dependency length (Stanford Parser; de Marneffe, MacCartney, & Manning, 2006 [\(2\)](#)). Sentences that were incorrectly parsed by the automatic syntactic parser (i.e., past participle adjectives parsed as verbs) were corrected by hand. Word frequency was calculated as the negative log of a word's occurrence rate in the Google corpus between the years 2017-2019. Bigram surprisal was calculated as the negative log of the frequency of a given two-word phrase in the Google corpus divided by the frequency of the first word of the phrase.

This calculation uses a log base of 2 in order to express surprisal in terms of "bits" that the first word provides in the context of the phrase. We used *bigram* surprisal as our surprisal measure to maximize the number of n-grams that had an entry in the corpus. Even so, 64 out of the 1515 total bigrams (4%) did not have an entry in the corpus and were therefore assigned the highest surprisal value among the rest of the bigrams (see Willems et al., 2016 [\(2\)](#)).

Illness type	Number of trials
acne	2
allergies	1
anemia	1
asthma	1
blood cancer	1
brain cancer	1
breast cancer	1
cancer (unspecified)	1
chickenpox	1
cold	2
COVID	2
epilepsy	1
flu	3
food poisoning	1
GI inflammation	2
GI virus	1
heart disease	3
high blood pressure	1
HIV-AIDS	1
liver disease	2
lung cancer	1
lung disease	2
malaria	1
pneumonia	1
skin cancer	2
throat cancer	1
Type 2 diabetes	1

Supplementary Table 1

Illness types present in the stimulus set.

Search space	Contrast	Accuracy	t	Permuted p	Bonferroni adj. p
LTPJ	ment_vs_phys	61.70%	2.96	0.0053	0.032
RTPJ	ment_vs_phys	52.50%	0.71	0.2573	1
LPC	ment_vs_phys	61.30%	3.44	0.003	0.0112
RPC	ment_vs_phys	60%	2.4	0.0176	0.108
LTPJ	caus_vs_rest	58.30%	4.16	0.0004	0.0024
RTPJ	caus_vs_rest	51.30%	0.39	0.3305	1
LPC	caus_vs_rest	60.80%	2.94	0.0055	0.0336
RPC	caus_vs_rest	53.70%	1.1	0.1516	1
Logic	logic_vs_lang	60.40%	3.46	0.0017	0.0026
Language	lang_vs_math	58.80%	2.76	0.0069	0.0124

Supplementary Table 2

Results of preregistered MVPA for *Illness-Causal* vs. *Mechanical-Causal* in individual-subject functional ROIs. Each fROI was created by selecting the top 300 vertices for each contrast (see 'Contrast') in each search space. Accuracy refers to classifier performance against chance (50%) for *Illness-Causal* vs. *Mechanical-Causal*. Permuted and Bonferroni-corrected (across fROIs) p-values are reported. *Ment_vs_phys*: *mentalizing stories* > *physical stories* (mentalizing localizer). *Caus_vs_rest*: *Illness-Causal* + *Illness-Mechanical* > *Rest*. *Logic_vs_lang*: *logic* > *language* (language/logic localizer). *Lang_vs_math*: *language* > *math* (language/logic localizer). Visualizations of these results are displayed in [Supplementary Figure 10](#).

Search space	Test	Accuracy	t	Permuted p	Bonferroni adj. p
LPC	ill-causal vs. mech-causal	61.30%	3.44	0.0018	0.007
LPC	ill-causal vs. noncausal1	51.70%	0.43	0.3345	1
LPC	ill-causal vs. noncausal2	58.30%	3.45	0.0018	0.007
LPC	mech-causal vs. noncausal1	56.20%	1.66	0.0542	0.284
LPC	mech-causal vs. noncausal2	62.50%	3.81	0.0005	0.003
Language	ill-causal vs. mech-causal	58.80%	2.76	0.0097	0.093
Language	ill-causal vs. noncausal1	54.60%	1.5	0.083	1
Language	ill-causal vs. noncausal2	55%	1.61	0.0675	0.93
Language	mech-causal vs. noncausal1	52.90%	0.85	0.1957	1
Language	mech-causal vs. noncausal2	53.30%	1.09	0.1609	1
Logic	ill-causal vs. mech-causal	60.40%	3.46	0.0029	0.0195
Logic	ill-causal vs. noncausal1	60.40%	3.27	0.0029	0.03
Logic	ill-causal vs. noncausal2	52.90%	0.88	0.1928	1
Logic	mech-causal vs. noncausal1	53.80%	1.23	0.1121	1
Logic	mech-causal vs. noncausal2	55.80%	1.97	0.0425	0.4785

Supplementary Table 3

MVPA results for all tests in select individual-subject functional ROIs. Each fROI was created by selecting the top 300 vertices for each contrast in each search space: left PC (LPC) = top *main experimental conditions > rest*, language = top *language > math* (language/logic localizer), logic = top *logic > language* (language/logic localizer). Accuracy refers to classifier performance against chance (50%) for each test. Permuted and Bonferroni-corrected (across fROIs) p-values are reported. Visualizations of these results are displayed in [Supplementary Figure 11](#).

Appendix 3: Full behavioral results.

Accuracy on the magic detection task was at ceiling ($M = 97.9\% \pm 2.2$ SD). There were no significant differences across the 4 main experimental conditions (*Illness-Causal*, *Mechanical-Causal*, *Noncausal-Illness First*, *Noncausal-Mechanical First*), but participants were more accurate on *Illness-Causal* trials compared to ‘magical’ catch trials ($F_{(4,76)} = 2.81, p = .03$; *Illness-Causal*: $M = 98.8\% \pm 2.2$ SD; ‘magical’ catch trials: $M = 96.4\% \pm 3.8$ SD).

A one-way repeated measures ANOVA evaluating response time revealed a main effect of condition, $F_{(4,76)} = 8.17, p < .001$, whereby participants were faster on *Illness-Causal* trials ($M = 4.73 \pm 0.81$ SD) compared to *Noncausal-Illness First* ($M = 5.33 \text{ s} \pm 0.85$ SD), *Noncausal-Mechanical First* ($M = 5.27 \text{ s} \pm 0.89$ SD) trials, and ‘magical’ catch trials ($M = 5.34 \text{ s} \pm 0.89$ SD). There were no differences in response time between *Mechanical-Causal* ($M = 5.15 \text{ s} \pm 0.88$ SD) and any other conditions.

Accuracy on the language/logic localizer task was significantly lower for the logic task compared to both the language and math tasks (logic: $M = 67.5\% \pm 14.0$ SD, math: $M = 93.8\% \pm 6.4$ SD, language: $M = 98.1\% \pm 5.8$ SD; $F_{(2,38)} = 60.38, p < .0001$). Similarly, response time was slowest on the logic task, followed by math and then language (logic: $M = 8.78 \text{ s} \pm 1.88$ SD, math: $M = 6.20 \text{ s} \pm 1.37$ SD, language: $M = 5.18 \text{ s} \pm 1.53$ SD; $F_{(2,38)} = 44.28, p < .001$).

Accuracy on the mentalizing localizer task was not different across the mentalizing stories and physical stories conditions (mentalizing: $83.50\% \pm 15.7$ SD, physical: $90.50\% \pm 12.3$ SD; $F_{(1,19)} = 2.73, p = .12$). However, response time for the mentalizing stories was significantly slower (mentalizing: $3.46 \text{ s} \pm 0.55$ SD, physical: $3.11 \text{ s} \pm 0.56$ SD; $F_{(1,19)} = 16.59, p < .001$).

Appendix 4: Individual-subject univariate fROI analysis in the fusiform face area (FFA).

In an exploratory analysis, we defined individual-subject fROIs in the fusiform face area (FFA). Illness inference fROIs were created in left and right FFA search spaces from a previous study on responses to images of faces in the ventral stream (Julian et al., 2012 [2](#)) using an iterated leave-one-run-out procedure. In each participant, we identified the most illness inference-responsive vertices in left and right FFA search spaces in 5 of the 6 runs (top 5% of vertices, *Illness-Causal* > *Mechanical-Causal*). We then extracted PSC for each condition compared to rest in the held-out run (*Illness-Causal*, *Mechanical-Causal*, *Noncausal-Illness First*, *Noncausal-Mechanical First*), averaging the results across all iterations.

In contrast to the PC, the FFA did not show a preference for illness inferences compared to mechanical inferences (leave-one-run-out individual-subject fROI analysis; repeated measures ANOVA, condition (*Illness-Causal*, *Mechanical-Causal*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 0.04, p = .84$, main effect of hemisphere, $F_{(1,19)} = 9.46, p = .006$, condition x hemisphere interaction, $F_{(1,19)} = 1.34, p = .26$; **Supplementary Figure 8** [2](#)). Additionally, the FFA did not show a preference for illness inferences compared to noncausal vignettes, which contained illness-related language but were not causally connected (repeated measures ANOVA, condition (*Illness-Causal*, *Noncausal-Illness First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 0.94, p = .34$, main effect of hemisphere, $F_{(1,19)} = 4.47, p = .05$, condition x hemisphere interaction, $F_{(1,19)} = 0.06, p = .82$; repeated measures ANOVA, condition (*Illness-Causal*, *Noncausal-Mechanical First*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 0.07, p = .8$;

main effect of hemisphere, $F_{(1,19)} = 7.59, p = .01$; condition x hemisphere interaction, $F_{(1,19)} = 2.72, p = .12$; **Supplementary Figure 8**). Thus, although the FFA exhibits a preference for images of animates (e.g., Kanwisher et al., 1997), the current evidence suggests that this region is not sensitive to abstract causal knowledge about animacy-specific processes (i.e., illness).

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Reviewer #1 (Public review):

Summary:

In this study, the authors aim to understand the neural basis of implicit causal inference, specifically how people infer causes of illness. They use fMRI to explore whether these

inferences rely on content-specific semantic networks or broader, domain-general neurocognitive mechanisms. The study explores two key hypotheses: first, that causal inferences about illness rely on semantic networks specific to living things, such as the 'animacy network,' given that illnesses affect only animate beings; and second, that there might be a common brain network supporting causal inferences across various domains, including illness, mental states, and mechanical failures. By examining these hypotheses, the authors aim to determine whether causal inferences are supported by specialized or generalized neural systems.

The authors observed that inferring illness causes selectively engaged a portion of the precuneus (PC) associated with the semantic representation of animate entities, such as people and animals. They found no cortical areas that responded to causal inferences across different domains, including illness and mechanical failures. Based on these findings, the authors concluded that implicit causal inferences are supported by content-specific semantic networks, rather than a domain-general neural system, indicating that the neural basis of causal inference is closely tied to the semantic representation of the specific content involved.

Strengths:

- The inclusion of the four conditions in the design is well thought out, allowing for the examination of the unique contribution of causal inference of illness compared to either a different type of causal inference (mechanical) or non-causal conditions. This design also has the potential to identify regions involved in a shared representation of inference across general domains.
- The presence of the three localizers for language, logic, and mentalizing, along with the selection of specific regions of interest (ROIs), such as the precuneus and anterior ventral occipitotemporal cortex (antVOTC), is a strong feature that supports a hypothesis-driven approach (although see below for a critical point related to the ROI selection).
- The univariate analysis pipeline is solid and well developed.
- The statistical analyses are a particularly strong aspect of the paper.

Weaknesses:

After carefully considering the authors' response, I believe that my primary concern has not been fully addressed. My main point remains unresolved:

The authors attempt to test for the presence of a shared network by performing only the Causal vs. Non-causal analysis. However, this approach is not sufficiently informative because it includes all conditions mixed together and does not clarify whether both the illness-causal and mechanical-causal conditions contribute to the observed results.

To address this limitation, I originally suggested an additional step: using as ROIs the different regions that emerged in the Causal vs. Non-causal decoding analysis and conducting four separate decoding analyses within these specific clusters:

- (1) Illness-Causal vs. Non-causal - Illness First
- (2) Illness-Causal vs. Non-causal - Mechanical First
- (3) Mechanical-Causal vs. Non-causal - Illness First
- (4) Mechanical-Causal vs. Non-causal - Mechanical First

This approach would allow the authors to determine whether any of these ROIs can decode both the illness-causal and mechanical-causal conditions against at least one non-causal condition. However, the authors did not conduct these analyses, citing an independence issue. I disagree with this reasoning because these analyses would serve to clarify their initial

general analysis, in which multiple conditions were mixed together. As the results currently stand, it remains unclear which specific condition is driving the effects.

My suggestion was to select the ROIs from their general analysis (Causal vs. Non-causal) and then examine in more detail which conditions were driving these results. This is not a case of double-dipping from my perspective, but rather a necessary step to unpack the general findings. Moreover, using ROIs would actually reduce the number of multiple comparisons that need to be controlled for.

If the authors believe that this approach is methodologically incorrect, then they should instead conduct all possible analyses at the whole-brain level to examine the effects of the specific conditions independently.

<https://doi.org/10.7554/eLife.101944.2.sa3>

Reviewer #2 (Public review):

Summary:

In this study, the authors test whether intuitive biological causal knowledge is embedded in domain-specific semantic networks, primarily focusing on the precuneus as part of the animacy semantic network. They do so tanks to an fMRI task, by comparing brain activity elicited by participants' exposure to written situations suggesting a plausible cause of illness with brain activity in linguistically equivalent situations suggesting a plausible cause of mechanical failure or damage and non-causal situations. These contrasts confirm the PC as the main "culprit" in whole-brain and fROIs univariate analyses. In turn, inferring causes of mechanical failure engages mostly the PPA. The authors further test whether the content-specificity has to do with inferences about animates in general, or if there are some distinctions between reasoning about people's bodies versus mental states. To answer this question, the authors localize the mentalizing network and study the relation between brain activity elicited by Illness-Causal > Mech-Causal and Mentalizing > Physical stories. They conclude that inferring about the causes of illness partially differentiates from reasoning about people's states of mind. The authors finally test the alternative yet non-mutually exclusive hypothesis that both types of implicit causal inferences (illness and mechanical) depend on shared neural machinery. Good candidates are language and logic, which justifies the use of a language/logic localizer. No evidence of commonalities across causal inferences versus non-causal situations are found.

Strengths:

- (1) This study introduces a useful paradigm and well-designed set of stimuli to test for implicit causal inferences.
- (2) Another important methodological advance is the addition of physical stories to the original mentalizing protocol.

These tools pave the way for further investigation of domain-specific causal inference.

- (3) The authors have significantly improved the manuscript, addressing previous concerns and incorporating additional analyses that strengthen their conclusions.

Key improvements:

- (1) The revised introduction makes the study's contribution more explicit and resolves initial ambiguities regarding its scope.
- (2) The rationale for focusing primarily on the precuneus is now clearer and the additional analysis in the fusiform face area provides a valuable comparison.
- (3) The revised manuscript now includes a more detailed examination of the searchlight MVPA results, showing that illness and mechanical inferences elicit spatially distinct neural

patterns in key regions, including the left PC, anterior PPA, and lateral occipitotemporal cortex.

(4) The authors' justification for using an implicit inference task, arguing that explicit tasks introduce executive function confounds, is convincing.

(5) The authors now acknowledge that while their results support a content-specific neural basis for implicit causal inference, domain-general mechanisms may still play a role in other contexts.

I have no major remaining concerns.

<https://doi.org/10.7554/eLife.101944.2.sa2>

Reviewer #3 (Public review):

Summary:

This study employed an implicit task, showing vignettes to participants while bold signal was acquired. The aim was to capture automatic causal inferences that emerge during language processing and comprehension. In particular, the authors compared causal inferences about illness with two control conditions, causal inferences about mechanical failures and non-causal phrases related to illnesses. All phrases that were employed described contexts with people, to avoid animacy/inanimate confound in the results. The authors had a specific hypothesis concerning the role of the precuneus (PC) being sensitive to causal inferences about illnesses (that was preregistered).

Findings indicate that implicit causal inferences are facilitated by semantic networks specialized for encoding causal knowledge.

Strengths:

The major strength of the study is the clever design of the stimuli (which are nicely matched for a number of features) which can tease apart the role of the type of causal inference (illness-causal or mechanical-causal) and the use of two localizers (logic/language and mentalizing) to investigate the hypothesis that the language and/or logical reasoning networks preferentially respond to causal inference regardless of the content domain being tested (illnesses or mechanical).

I think that authors' revisions of the original manuscript have strengthened the study. Overall, the paper provides an interesting contribution to the (rather new) field of study concerning the neural basis of implicit causal inference.

I see two weaknesses concerning the visualization of the data (which could be improved)

(1) Measures of dispersion are now provided for the average PSC in the critical window. It would be more appropriate to show the variance of the data also for the percentage signal changes (PSC) figures (e.g., 1A) by using shaded lines providing SE around the means or boxplots at each timepoint).

(2) The authors could consider showing in Figure 2 the data of supplementary Figure 3. It is not clear why the authors report in the main manuscript the results of a subsample of participants (and only for this figure).

<https://doi.org/10.7554/eLife.101944.2.sa1>

Author response:

The following is the authors' response to the original reviews

Reviewer #1 (Public review):**Summary:**

In this study, the authors aim to understand the neural basis of implicit causal inference, specifically how people infer causes of illness. They use fMRI to explore whether these inferences rely on content-specific semantic networks or broader, domain-general neurocognitive mechanisms. The study explores two key hypotheses: first, that causal inferences about illness rely on semantic networks specific to living things, such as the 'animacy network,' given that illnesses affect only animate beings; and second, that there might be a common brain network supporting causal inferences across various domains, including illness, mental states, and mechanical failures. By examining these hypotheses, the authors aim to determine whether causal inferences are supported by specialized or generalized neural systems.

The authors observed that inferring illness causes selectively engaged a portion of the precuneus (PC) associated with the semantic representation of animate entities, such as people and animals. They found no cortical areas that responded to causal inferences across different domains, including illness and mechanical failures. Based on these findings, the authors concluded that implicit causal inferences are supported by content-specific semantic networks, rather than a domain-general neural system, indicating that the neural basis of causal inference is closely tied to the semantic representation of the specific content involved.

Strengths:

(1) *The inclusion of the four conditions in the design is well thought out, allowing for the examination of the unique contribution of causal inference of illness compared to either a different type of causal inference (mechanical) or non-causal conditions. This design also has the potential to identify regions involved in a shared representation of inference across general domains.*

(2) *The presence of the three localizers for language, logic, and mentalizing, along with the selection of specific regions of interest (ROIs), such as the precuneus and anterior ventral occipitotemporal cortex (antVOTC), is a strong feature that supports a hypothesis-driven approach (although see below for a critical point related to the ROI selection).*

(3) *The univariate analysis pipeline is solid and well-developed.*

(4) *The statistical analyses are a particularly strong aspect of the paper.*

Weaknesses:

Based on the current analyses, it is not yet possible to rule out the hypothesis that inferring illness causes relies on neurocognitive mechanisms that support causal inferences irrespective of their content, neither in the precuneus nor in other parts of the brain.

(1) *The authors, particularly in the multivariate analyses, do not thoroughly examine the similarity between the two conditions (illness-causal and mechanical-causal), as they are more focused on highlighting the differences between them. For instance, in the searchlight MVPA analysis, an interesting decoding analysis is conducted to identify brain regions that represent illness-causal and mechanical-causal conditions differently,*

yielding results consistent with the univariate analyses. However, to test for the presence of a shared network, the authors only perform the Causal vs. Non-causal analysis. This analysis is not very informative because it includes all conditions mixed together and does not clarify whether both the illness-causal and mechanical-causal conditions contribute to these results.

(2) To address this limitation, a useful additional step would be to use as ROIs the different regions that emerged in the Causal vs. Non-causal decoding analysis and to conduct four separate decoding analyses within these specific clusters:

- (a) Illness-Causal vs. Non-causal - Illness First;*
- (b) Illness-Causal vs. Non-causal - Mechanical First;*
- (c) Mechanical-Causal vs. Non-causal - Illness First;*
- (d) Mechanical-Causal vs. Non-causal - Mechanical First.*

This approach would allow the authors to determine whether any of these ROIs can decode both the illness-causal and mechanical-causal conditions against at least one non-causal condition.

(3) Another possible analysis to investigate the existence of a shared network would be to run the searchlight analysis for the mechanical-causal condition versus the two non-causal conditions, as was done for the illness-causal versus non-causal conditions, and then examine the conjunction between the two. Specifically, the goal would be to identify ROIs that show significant decoding accuracy in both analyses.

The hypothesis that a neural mechanism supports causal inference across domains predicts higher univariate responses when causal inferences occur than when they do not. This prediction was not generated by us ad hoc but rather has been made by almost all previous cognitive neuroscience papers on this topic (Ferstl & von Cramon, 2001; Satpute et al., 2005; Fugelsang & Dunbar, 2005; Kuperberg et al., 2006; Fenker et al., 2010; Kranjec et al., 2012; Pramod, Chomik-Morales, et al., 2023; Chow et al., 2008; Mason & Just, 2011; Prat et al., 2011). Contrary to this hypothesis, we find that the precuneus (PC) is most activated for illness inferences and most deactivated for mechanical inferences relative to rest, suggesting that the PC does not support domain-general causal inference. To further probe the selectivity of the PC for illness inferences, we created group overlap maps that compare PC responses to illness inferences and mechanical inferences across participants. The PC shows a strong preference for illness inferences and is therefore unlikely to support causal inferences irrespective of their content (Supplementary Figures 6 and 7). We also note that, in whole-cortex analysis, no shared regions responded more to causal inference than noncausal vignettes across domains. Therefore, the prediction made by the ‘domain-general causal engine’ proposal as it has been articulated in the literature is not supported in our data.

Taking a multivariate approach, the hypothesis that a neural mechanism supports causal inference across domains also predicts that relevant regions can decode between all possible pairs of causal vs. noncausal conditions (e.g., Illness-Causal vs. Noncausal-Illness First, Mechanical-Causal vs. Noncausal-Illness First, etc.). The analysis described by the reviewer in (2), in which the regions that distinguish between causal vs. noncausal conditions in searchlight MVPA are used as ROIs to test various causal vs. noncausal contrasts, is non-independent. Therefore, we cannot perform this analysis. In accordance with the reviewer’s suggestions in (3), now include searchlight MVPA results for the mechanical inference condition compared to the two noncausal conditions (Supplementary Figure 9). No regions are shared across the searchlight analyses comparing all possible pairs of causal and noncausal conditions, providing further evidence that there are no shared neural responses to causal inference in our dataset.

(4) Along the same lines, for the ROI MVPA analysis, it would be useful not only to include the illness-causal vs. mechanical-causal decoding but also to examine the illness-causal vs. non-causal conditions and the mechanical-causal vs. non-causal conditions.

Additionally, it would be beneficial to report these data not just in a table (where only the mean accuracy is shown) but also using dot plots, allowing the readers to see not only the mean values but also the accuracy for each individual subject.

We have performed these analyses and now include a table of the results as well as figures displaying the dispersion across participants (Supplementary Tables 2 and 3, Supplementary Figures 10 and 11). In the left PC, the illness inference condition was decoded from one of the noncausal conditions, and the mechanical inference condition was decoded from the same noncausal condition. The language network did not decode between any causal/noncausal pairs. In the logic network, the illness inference condition was decoded from one of the noncausal conditions, and the mechanical inference condition was decoded from the other noncausal condition. Thus, no regions showed the predicted ‘domain-general’ pattern, i.e., significant decoding between all causal/noncausal pairs.

Importantly, the decoding results must be interpreted in light of significant univariate differences across conditions (e.g., greater responses to illness inferences compared to noncausal vignettes in the PC). Linear classifiers are highly sensitive to univariate differences (Coutanche, 2013; Kragel et al., 2012; Hebart & Baker, 2018; Woolgar et al., 2014; Davis et al., 2014; Pakravan et al., 2022).

(5) The selection of Regions of Interest (ROIs) is not entirely straightforward:

In the introduction, the authors mention that recent literature identifies the precuneus (PC) as a region that responds preferentially to images and words related to living things across various tasks. While this may be accurate, we can all agree that other regions within the ventral occipital-temporal cortex also exhibit such preferences, particularly areas like the fusiform face area, the occipital face area, and the extrastriate body area. I believe that at least some parts of this network (e.g., the fusiform gyrus) should be included as ROIs in this study. This inclusion would make sense, especially because a complementary portion of the ventral stream known to prefer non-living items (i.e., anterior medial VOTC) has been selected as a control ROI to process information about the mechanical-causal condition. Given the main hypothesis of the study - that causal inferences about illness might depend on content-specific semantic representations in the ‘animacy network’ - it would be worthwhile to investigate these ROIs alongside the precuneus, as they may also yield interesting results.

We thank the reviewer for their suggestion to test the FFA region. We think this provides an interesting comparison to the PC and hypothesized that, in contrast to the PC, the FFA does not encode abstract causal information about animacy-specific processes (i.e., illness). As we mention in the Introduction, although the fusiform face area (FFA) also exhibits a preference for animates, it does so primarily for images in sighted people (Kanwisher et al., 1997; Kanwisher et al., 1997; Grill-Spector et al., 2004; Noppeney et al., 2006; Konkle & Caramazza, 2013; Connolly et al., 2016; Bi et al., 2016).

We did not select the FFA as a region of interest when preregistering the current study because we did not predict it would show sensitivity to causal knowledge. In accordance with the reviewer’s suggestions, we now include the FFA as an ROI in individual-subject univariate analysis (Supplementary Figure 8, Appendix 4). Because we did not run a separate FFA localizer task when collecting the data, we used FFA search spaces from a previous study investigating responses to face images (Julian et al., 2012). We followed the same analysis procedure that was used to investigate responses to illness inferences in the PC. Neither left

nor right FFA exhibited a preference for illness inferences compared to mechanical inferences or to the noncausal conditions. This result is interesting and is now briefly discussed in the Discussion section.

(6) Visual representation of results:

In all the figures related to ROI analyses, only mean group values are reported (e.g., Figure 1A, Figure 3, Figure 4A, Supplementary Figure 6, Figure 7, Figure 8). To better capture the complexity of fMRI data and provide readers with a more comprehensive view of the results, it would be beneficial to include a dot plot for a specific time point in each graph. This could be a fixed time point (e.g., a certain number of seconds after stimulus presentation) or the time point showing the maximum difference between the conditions of interest. Adding this would allow for a clearer understanding of how the effect is distributed across the full sample, such as whether it is consistently present in every subject or if there is greater variability across individuals.

We thank the reviewer for this suggestion. We now include scattered box plots displaying the dispersion in average percent signal change across participants in Figures 1, 3, and 4, and Supplementary Figures 8, 12, and 14.

(7) Task selection:

(a) To improve the clarity of the paper, it would be helpful to explain the rationale behind the choice of the selected task, specifically addressing: (i) why an implicit inference task was chosen instead of an explicit inference task, and (ii) why the "magic detection" task was used, as it might shift participants' attention more towards coherence, surprise, or unexpected elements rather than the inference process itself.

(b) Additionally, the choice to include a large number of catch trials is unusual, especially since they are modeled as regressors of non-interest in the GLM. It would be beneficial to provide an explanation for this decision.

We chose an orthogonal foil detection task, rather than an explicit causal judgment task, to investigate automatic causal inferences during reading and to unconfound such processing as much as possible from explicit decision-making processes (see Kuperberg et al., 2006 for discussion). Analogous foil detection paradigms have been used to study sentence processing and word recognition (Pallier et al., 2011; Dehaene-Lambertz et al., 2018). We now clarify this in the Introduction. The “magical” element occurred both within and across sentences so that participants could not use coherence as a cue to complete the task. Approximately 1/5 (19%) of the trials were magical catch trials to ensure that participants remained attentive throughout the experiment.

Reviewer #2 (Public review):

Summary:

In this study, the authors hypothesize that "causal inferences about illness depend on content-specific semantic representations in the animacy network". They test this hypothesis in an fMRI task, by comparing brain activity elicited by participants' exposure to written situations suggesting a plausible cause of illness with brain activity in linguistically equivalent situations suggesting a plausible cause of mechanical failure or damage and non-causal situations. These contrasts identify PC as the main "culprit" in a whole-brain univariate analysis. Then the question arises of whether the content-specificity has to do with inferences about animates in general, or if there are some distinctions between reasoning about people's bodies versus mental states. To answer this question, the authors localize the mentalizing network and study the relation

between brain activity elicited by Illness-Causal > Mech-Causal and Mentalizing > Physical stories. They conclude that inferring about the causes of illness partially differentiates from reasoning about people's states of mind. The authors finally test the alternative yet non-mutually exclusive hypothesis that both types of causal inferences (illness and mechanical) depend on shared neural machinery. Good candidates are language and logic, which justifies the use of a language/logic localizer. No evidence of commonalities across causal inferences versus non-causal situations is found.

Strengths:

- (1) *This study introduces a useful paradigm and well-designed set of stimuli to test for implicit causal inferences.*
- (2) *Another important methodological advance is the addition of physical stories to the original mentalizing protocol.*
- (3) *With these tools, or a variant of these tools, this study has the potential to pave the way for further investigation of naïve biology and causal inference.*

Weaknesses:

- (1) *This study is missing a big-picture question. It is not clear whether the authors investigate the neural correlates of causal reasoning or of naïve biology. If the former, the choice of an orthogonal task, making causal reasoning implicit, is questionable. If the latter, the choice of mechanical and physical controls can be seen as reductive and problematic.*

We have modified the Introduction to clarify that the primary goal of the current study is to test the claim that semantic networks encode causal knowledge – in this case, causal intuitive theories of biology. Most conceptions of intuitive biology, intuitive psychology, and intuitive physics describe them as causal frameworks (e.g., Wellman & Gelman, 1992; Simons & Keil, 1995; Keil et al., 1999; Tenenbaum, Griffiths, & Niyogi, 2007; Gopnik & Wellman, 2012; Gerstenberg & Tenenbaum, 2017). As noted above, we chose an implicit task to investigate automatic causal inferences during reading and to unconfound such processing as much as possible from explicit decision-making processes. We are not sure what the reviewer means when they say that mechanical and physical controls are reductive. This is the standard control condition in neural and behavioral paradigms that investigate intuitive psychology and intuitive biology (e.g., Saxe & Kanwisher, 2003; Gelman & Wellman, 1991).

- (2) *The rationale for focusing mostly on the precuneus is not clear and this choice could almost be seen as a post-hoc hypothesis.*

This study is preregistered (<https://osf.io/6pnqg>). The preregistration states that the precuneus is a hypothesized area of interest, so this is not a post-hoc hypothesis. Our hypothesis was informed by multiple prior studies implicating the precuneus in the semantic representation of animates (e.g., people, animals) (Fairhall & Caramazza, 2013a, 2013b; Fairhall et al., 2014; Peer et al., 2015; Wang et al., 2016; Silson et al., 2019; Rabini, Ubaldi, & Fairhall, 2021; Deen & Freiwald, 2022; Aglinskas & Fairhall, 2023; Hauptman, Elli, et al., 2025). We also conducted a pilot experiment with separate participants prior to pre-registering the study. We now clarify our rationale for focusing on the precuneus in the Introduction:

“Illness affects living things (e.g., people and animals) rather than inanimate objects (e.g., rocks, machines, houses). Thinking about living things (animates) as opposed to non-living things (inanimate objects/places) recruits partially distinct neural systems (e.g., Warrington & Shallice, 1984; Hillis & Caramazza, 1991; Caramazza & Shelton, 1998; Farah & Rabinowitz, 2003). The precuneus (PC) is part of the ‘animacy’ semantic network and responds

preferentially to living things (i.e., people and animals), whether presented as images or words (Devlin et al., 2002; Fairhall & Caramazza, 2013a, 2013b; Fairhall et al., 2014; Peer et al., 2015; Wang et al., 2016; Silson et al., 2019; Rabini, Ubaldi, & Fairhall, 2021; Deen & Freiwald, 2022; Aglinskas & Fairhall, 2023; Hauptman, Elli, et al., 2025). By contrast, parts of the visual system (e.g., fusiform face area) that respond preferentially to animates do so primarily for images (Kanwisher et al., 1997; Grill-Spector et al., 2004; Noppeney et al., 2006; Mahon et al., 2009; Konkle & Caramazza, 2013; Connolly et al., 2016; see Bi et al., 2016 for a review). We hypothesized that the PC represents causal knowledge relevant to animates and tested the prediction that it would be activated during implicit causal inferences about illness, which rely on such knowledge (preregistration: <https://osf.io/6pnqg/>).

(3) The choice of an orthogonal 'magic detection' task has three problematic consequences in this study:

- (a) It differs in nature from the 'mentalizing' task that consists of evaluating a character's beliefs explicitly from the corresponding story, which complicates the study of the relation between both tasks. While the authors do not compare both tasks directly, it is unclear to what extent this intrinsic difference between implicit versus explicit judgments of people's body versus mental states could influence the results.*
- (b) The extent to which the failure to find shared neural machinery between both types of inferences (illness and mechanical) can be attributed to the implicit character of the task is not clear.*
- (c) The introduction of a category of non-interest that contains only 36 trials compared to 38 trials for all four categories of interest creates a design imbalance.*

We disagree with the reviewer's argument that our use of an implicit "magic detection" task is problematic. Indeed, we think it is one of the advances of the current study over prior work.

- a) Prior work has shown that implicit mentalizing tasks (e.g., naturalistic movie watching) engages the theory of mind network, suggesting that the implicit/explicit nature of the task does not drive the activation of this network (Jacoby et al., 2016; Richardson et al., 2018). With these data in mind, it is unlikely that the implicit/explicit nature of the causal inference and theory of mind tasks in the present experiment can explain observed differences between them.
- b) Explicit causal inferences introduce a collection of executive processes that potentially confound the results and make it difficult to know whether neural signatures are related to causal inference per se. The current study focuses on the neural basis of implicit causal inference, a type of inference that is made routinely during language comprehension. We do not claim to find neural signatures of all causal inferences, we do not think any study could claim to do so because causal inferences are a highly varied class.
- c) Our findings do not exclude the possibility that content-invariant responses are elicited during explicit causality judgments. We clarify this point in the Results (e.g., "These results leave open the possibility that domain-general systems support the explicit search for causal connections") and Discussion (e.g., "The discovery of novel causal relationships (e.g., 'blicket detectors'; Gopnik et al., 2001) and the identification of complex causes, even in the case of illness, may depend in part on domain-general neural mechanisms").
- d) Because the magic trials are excluded from our analyses, it is unclear how the imbalance in the number of magic trials could influence the results and our interpretation of them. We note that the number of catch trials in standard target detection paradigms are sometimes much lower than the number of target trials in each condition (e.g., Pallier et al., 2011).

(4) Another imbalance is present in the design of this study: the number of trials per category is not the same in each run of the main task. This imbalance does not seem to be accounted for in the 1st-level GLM and renders a bit problematic the subsequent use of MVPA.

Each condition is shown either 6 or 7 times per run (maximum difference of 1 trial between conditions), and the number of trials per condition is equal across the whole experiment: each condition is shown 7 times in two of the runs and 6 times four of the runs. This minor design imbalance is typical of fMRI experiments and should not impact our interpretations of the data, particularly because we average responses from each condition within a run before submitting them to MVPA.

(5) The main claim of the authors, encapsulated by the title of the present manuscript, is not tested directly. While the authors included in their protocol independent localizers for mentalizing, language, and logic, they did not include an independent localizer for "animacy". As such, they cannot provide a within-subject evaluation of their claim, which is entirely based on the presence of a partial overlap in PC (which is also involved in a wide range of tasks) with previous results on animacy.

We respectfully disagree with this assertion. Our primary analysis uses a within-subject leave-one-run-out approach. This approach allows us to use part of the data itself to localize animacy-relevant causal responses in the PC without engaging in 'double-dipping' or statistical non-independence (Vul & Kanwisher, 2011). We also use the mentalizing network localizer as a partial localizer for animacy. This is because the control condition (physical reasoning) does not include references to people or any animate agents (Supplementary Figures 1 and 15). We now clarify this point in Methods section of the paper (see below).

From the Methods: "To test the relationship between neural responses to inferences about the body and the mind, and to localize animacy regions, we used a localizer task to identify the mentalizing network in each participant (Saxe & Kanwisher, 2003; Dodell-Feder et al., 2011; <http://saxelab.mit.edu/use-our-efficient-false-belief-localizer>)...Our physical stories incorporated more vivid descriptions of physical interactions and did not make any references to human agents, enabling us to use the mentalizing localizer as a localizer for animacy."

Reviewer #3 (Public review):

Summary:

This study employed an implicit task, showing vignettes to participants while a bold signal was acquired. The aim was to capture automatic causal inferences that emerge during language processing and comprehension. In particular, the authors compared causal inferences about illness with two control conditions, causal inferences about mechanical failures and non-causal phrases related to illnesses. All phrases that were employed described contexts with people, to avoid animacy/inanimate confound in the results. The authors had a specific hypothesis concerning the role of the precuneus (PC) in being sensitive to causal inferences about illnesses.

These findings indicate that implicit causal inferences are facilitated by semantic networks specialized for encoding causal knowledge.

Strengths:

The major strength of the study is the clever design of the stimuli (which are nicely matched for a number of features) which can tease apart the role of the type of causal

inference (illness-causal or mechanical-causal) and the use of two localizers (logic/language and mentalizing) to investigate the hypothesis that the language and/or logical reasoning networks preferentially respond to causal inference regardless of the content domain being tested (illnesses or mechanical).

Weaknesses:

I have identified the following main weaknesses:

(1) Precuneus (PC) and Temporo-Parietal junction (TPJ) show very similar patterns of results, and the manuscript is mostly focused on PC (also the abstract). To what extent does the fact that PC and TPJ show similar trends affect the inferences we can derive from the results of the paper? I wonder whether additional analyses (connectivity?) would help provide information about this network.

We thank the reviewer for this suggestion. While the PC shows the most robust univariate preference for illness inferences compared to both mechanical inferences and noncausal vignettes, the TPJ also shows a preference for illness inferences compared to mechanical inferences in individual-subject fROI analysis. However, as we mention in the Results section, the TPJ does not show a preference for illness inferences compared to noncausal vignettes, suggesting that the TPJ is selective for animacy but may not be as sensitive to causal knowledge about animacy-specific processes. When describing our results, we refer to the ‘animacy network’ (i.e., PC and TPJ) but also highlight that the PC exhibited the most robust responses to illness inferences (from the Results: “Inferring illness causes preferentially recruited the animacy semantic network, particularly the PC”; from the Discussion: “We find that a semantic network previously implicated in thinking about animates, particularly the precuneus (PC), is preferentially engaged when people infer causes of illness...”). We did not collect resting state data that would enable a connectivity analysis, as the reviewer suggests. This is an interesting direction for future work.

(2) Results are mainly supported by an univariate ROI approach, and the MVPA ROI approach is performed on a subregion of one of the ROI regions (left precuneus). Results could then have a limited impact on our understanding of brain functioning.

The original and current versions of the paper include results from multiple multivariate analyses, including whole-cortex searchlight MVPA and individual-subject fROI MVPA performed in multiple search spaces (see Supplementary Figures 10 and 11, Supplementary Tables 2 and 3).

We note that our preregistered predictions focused primarily on univariate differences. This is because the current study investigates neural responses to inferences, and univariate increases in activity is thought to reflect the processing of such inferences. We use multivariate analyses to complement our primary univariate analyses. However, given that we observe significant univariate effects and that multivariate analyses are heavily influenced by significant univariate effects (Coutanche, 2013; Kragel et al., 2012; Hebart & Baker, 2018; Woolgar et al., 2014; Davis et al., 2014; Pakravan et al., 2022), our univariate results constitute the main findings of the paper.

(3) In all figures: there are no measures of dispersion of the data across participants. The reader can only see aggregated (mean) data. E.g., percentage signal changes (PSC) do not report measures of dispersion of the data, nor do we have bold maps showing the overlap of the response across participants. Only in Figure 2, we see the data of 6 selected participants out of 20.

We thank the reviewer for this suggestion. We now include graphs depicting the dispersion of the data across participants in the following figures: Figures 1, 3, and 4, and Supplementary Figures 8, 12, and 14. We have also created 2 figures that display the overlap of univariate responses across participants (Supplementary Figures 6 and 7). These figures show that there is high overlap across participants in PC responses to illness inferences but not mechanical inferences. In addition, all participants' results from the analysis depicted in Figure 2 are included in Supplementary Figure 3.

(4) Sometimes acronyms are defined in the text after they appear for the first time.

We thank the reviewer for pointing this out. We now define all acronyms before using them.

Recommendations for the authors:

Reviewer #1 (Recommendations for the authors):

(1) I was unable to access the pre-registration on OSF because special permission is required.

We apologize for this technical error. The preregistration is now publicly available: <https://osf.io/6pnqg>.

(2) The length of the MRI session is quite long (around 2 hours). It is generally discouraged to have such extended data acquisition periods, as this can affect the stability and cleanliness of the data. Did you observe any effects of fatigue or attention decline in your data?

The session was 2 hours long including 1-2 10-minute breaks. Without breaks, the scan would be approximately 1.5 hours. This is a standard length for MRI experiments. The main experiment (causal inference task) was always conducted first and lasted approximately 1 hour. Accuracy did not decrease across the 6 runs of this experiment (repeated measures ANOVA, $F_{(5,114)} = 1.35, p = .25$).

(3) The last sentence of the results states: "Although MVPA searchlight analysis identified several areas where patterns of activity distinguished between causal and non-causal vignettes, all of these regions showed a preference for non-causal vignettes in univariate analysis (Supplementary Figure 5)." This statement is not entirely accurate. As I previously pointed out, the MVPA searchlight analysis is not very informative and is difficult to interpret. However, as previously suggested, there are additional steps that could be taken to better understand and interpret these results. It is incorrect to conclude that because the brain regions identified in the MVPA analyses show a preference for non-causal vignettes in univariate analyses, the multivariate results lack value. While univariate analyses may show a preference for a specific condition, multivariate analyses can reveal more fine-grained representations of multiple conditions. For a notable example, consider the fusiform face area (FFA) that shows a clear preference for faces at the univariate level but can significantly decode other categories at the multivariate level, even when faces are not included in the analysis.

The decoding analysis that the reviewer is suggesting for the current study would be analogous to identifying univariate differences between faces and places in the FFA and then decoding between faces and places and claiming that the FFA represents places because the decoding is significant. The decoding analyses enabled by our design are not equivalent to decoding within a condition (e.g., among face identities, among types of illness inferences), as the reviewer suggests above. It is not that such multivariate analyses "lack value" but that

they recapitulate established univariate differences. Multivariate analyses are useful for revealing more fine-grained representations when i) significant univariate differences are not observed, or ii) when it is possible to decode among categories within a condition (e.g., among face identities, among types of illness inferences). We are currently collecting data that will enable us to perform within-condition decoding analyses in future work, but the design of the current study does not allow for such a comparison.

We note that the original quotation from the manuscript has been removed because it is no longer accurate. When including participant response time as a covariate of no interest in the GLM, no regions are shared across the 4 searchlight analyses comparing causal and noncausal conditions, suggesting that there are no shared neural responses to causal inference in our dataset.

Reviewer #2 (Recommendations for the authors):

(1) Moderating the strength of some claims made to justify the main hypothesis (e.g., "people but not machines transmit diseases to each other through physical contact").

We changed this wording so that it now reads: “Illness affects living things (e.g., people and animals) rather than inanimate objects (e.g., rocks, machines, houses).” (Introduction)

(2) Expanding the paragraph introducing the sub-question about inferring people's "body states" vs "mental states". In addition, given the order in which the hypotheses are introduced, and the results are presented, I would suggest switching the order of presentation of both localizers in the methods section and adding a quick reminder of the hypotheses that justify using these localizers.

We thank the reviewer for these suggestions. In accordance their suggestions, we have expanded the paragraph Introduction that introduces the “body states” vs. “mental states” question (see below). We have also switched the order of the localizer descriptions in the Methods section and added a sentence at the start of each section describing the relevant hypotheses (see below).

From the Introduction: “We also compared neural responses to causal inferences about the body (i.e., illness) and inferences about the mind (i.e., mental states). Both types of inferences are about animate entities, and some developmental work suggests that children use the same set of causal principles to think about bodies and minds (Carey, 1985, 1988). Other evidence suggests that by early childhood, young children have distinct causal knowledge about the body and the mind (Springer & Keil, 1991; Callanan & Oakes, 1992; Wellman & Gelman, 1992; Inagaki & Hatano, 1993; 2004; Keil, 1994; Hickling & Wellman, 2001; Medin et al., 2010). For instance, preschoolers are more likely to view illness as a consequence of biological causes, such as contagion, rather than psychological causes, such as malicious intent (Springer & Ruckel, 1992; Raman & Winer, 2004; see also Legare & Gelman, 2008). The neural relationship between inferences about bodies and minds has not been fully described. The ‘mentalizing network’, including the PC, is engaged when people reason about agents’ beliefs (Saxe & Kanwisher, 2003; Saxe et al., 2006; Saxe & Powell, 2006; Dodell-Feder et al., 2011; Dufour et al., 2013). We localized this network in individual participants and measured its neuroanatomical relationship to the network activated by illness inferences.”

From the Methods, localizer descriptions: “To test the relationship between neural responses to inferences about the body and the mind, and to localize animacy regions, we used a localizer task to identify the mentalizing network in each participant... To test for the presence of domain-general responses to causal inference in the language and logic networks (e.g., Kuperberg et al., 2006; Operksalski & Barbey, 2017), we used an additional localizer task to identify both networks in each participant.”

(3) Adding a quick analysis of lateralization to support the corresponding claim of left lateralization of responses to causal inferences.

In accordance with the reviewer's suggestion, we now include hemisphere as a factor in all ANOVAs comparing univariate responses across conditions.

From the Results: "In individual-subject fROI analysis (leave-one-run-out), we similarly found that inferring illness causes activated the PC more than inferring causes of mechanical breakdown (repeated measures ANOVA, condition (*Illness-Causal, Mechanical-Causal*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 19.18, p < .001$, main effect of hemisphere, $F_{(1,19)} = 0.3, p = .59$, condition x hemisphere interaction, $F_{(1,19)} = 27.48, p < .001$; Figure 1A). This effect was larger in the left than in the right PC (paired samples t-tests; left PC: $t_{(19)} = 5.36, p < .001$, right PC: $t_{(19)} = 2.27, p = .04$)...In contrast to the animacy-responsive PC, the anterior PPA showed the opposite pattern, responding more to mechanical inferences than illness inferences (leave-one-run-out individual-subject fROI analysis; repeated measures ANOVA, condition (*Mechanical-Causal, Illness-Causal*) x hemisphere (left, right): main effect of condition, $F_{(1,19)} = 17.93, p < .001$, main effect of hemisphere, $F_{(1,19)} = 1.33, p = .26$, condition x hemisphere interaction, $F_{(1,19)} = 7.8, p = .01$; Figure 4A). This effect was significant only in the left anterior PPA (paired samples t-tests; left anterior PPA: $t_{(19)} = 4, p < .001$, right anterior PPA: $t_{(19)} = 1.88, p = .08$)."

(4) Making public and accessible the pre-registration OSF link.

We apologize for this technical error. The preregistration is now publicly available: <https://osf.io/6pnqg>.

Reviewer #3 (Recommendations for the authors):

In all figures: there are no measures of dispersion of the data across participants. The reader can only see aggregated (mean) data. E.g., percentage signal changes (PSC) do not report measures of dispersion of the data, nor do we have bold maps showing the overlap of the response across participants. Only in Figure 2, we see the data of 6 selected participants out of 20.

We thank the reviewer for this suggestion. We now include graphs depicting the dispersion of the data across participants in the following figures: Figures 1, 3, and 4, and Supplementary Figures 8, 12, and 14. We have also created 2 figures that display the overlap of univariate responses across participants (Supplementary Figures 6 and 7). In addition, all participants' results from the analysis depicted in Figure 2 are included in Supplementary Figure 3.

Minor

(1) Figure 2: Spatial dissociation between responses to illness inferences and mental state inferences in the precuneus (PC). If the analysis is the result of the MVPA, the figure should report the fact that only the left precuneus was analyzed.

Figure 2 depicts the spatial dissociation in univariate responses to illness inferences and mental state inferences. We now clarify this in the figure legend.

(2) VOTC and PSC acronyms are defined in the text after they appear for the first time. TPJ is never defined.

We thank the reviewer for pointing this out. We now define all acronyms before using them.

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