

1 **Abstract.** When judging what caused an event, people do not treat all
2 factors equally – for instance, they will say that a forest fire was caused by a
3 lit match, and not mention the oxygen in the air which helped fuel the fire.
4 We develop a computational model formalizing the idea that causal judgment
5 is designed to identify “portable” causes – causes that are likely to generalize
6 across a variety of background circumstances. Under minimal assumptions,
7 the model is surprisingly simple: a factor is regarded as a cause of an out-
8 come to the extent that it is, across counterfactual worlds, correlated with
9 that outcome. The model explains why causal judgment is influenced by the
10 normality of candidate causes, and outperforms other known computational
11 models when tested against an existing fine-grained dataset of human graded
12 causal judgments (Morris, A., Phillips, J., Gerstenberg, T., & Cushman, F.
13 (2019). Quantitative causal selection patterns in token causation. *PloS one*,
14 *14*(8)).

15 **keywords.** causation | causal selection | computational modelling | counter-
16 factuais

17 When multiple causes contribute to an event, we tend to discriminate
18 among them: for instance, we tend to say that the forest fire was caused
19 by the match lit by a careless camper, but we regard the presence of oxygen in
20 the air as a mere ‘enabling condition’ or ‘contributing factor’. This suggests
21 that we implicitly rank the different causes of an event, as if we computed the
22 ‘actual causal strength’ of each of them.

23 Here we propose a model of how the mind computes actual causal strength.
24 Researchers have proposed that cognitive mechanisms for causal judgment
25 are well-designed for the problem of identifying ‘portable’ causes, i.e. causes
26 that would reliably lead to an outcome, across a wide range of different back-
27 ground conditions (see (Lombrozo, 2010; Hitchcock, 2012)). For instance, the
28 lit match is a ‘portable’ cause of the forest fire, because across a variety of
29 plausible background circumstances, striking a match inside a forest would
30 result in a forest fire.

31 We formulate this hypothesis as a simple computational theory (Marr,
32 1982). Identifying portable causes requires that when one judges how much a
33 factor C was causally responsible for an outcome E, one does not focus exclu-
34 sively on what actually happened. One also needs to compute the effect that a
35 manipulation of C would have had on E in a range of alternative possible situa-
36 tions. This suggests a measure of causal strength which is similar to the ‘effect
37 size’ measures that scientists use in interpreting the results of an experiment.
38 On average, across possible situations, by how many standard deviation units
39 can one change the value of E by making a one standard-deviation change in
40 C? In many contexts, this is simply equivalent to computing the correlation
41 between C and E across the possible situations that we imagined.

42 We formally express this theory as a simple algorithm, and show that it
43 can explain a wide range of human causal intuitions.

44 1 Model

45 We define an algorithm which takes as input an event (e.g. someone lits a
46 match, there is oxygen in the air, and the forest catches fire), and delivers
47 an actual causal strength score for a candidate cause (e.g. how well the lit
48 match qualifies as having caused the forest fire). We assume that the agent
49 making a causal judgment possesses a representation of the causal structure
50 of the situation she is evaluating (e.g. she knows that lightning a match tends
51 to generate fire, unless there is no oxygen in the air). We use the formalism
52 of structural equation models to model such representations (see SI for an
53 informal introduction, and (Halpern, 2016), for a technical treatment), and
54 refer to a specific state of a causal system as a ‘world’. The following algorithm
55 generates a causal score $k_{C \rightarrow E}$ quantifying how well C qualifies as a cause of
56 E.

57 **a.** Simulate a large number of worlds by sampling the set of possible
58 worlds, according to the prior probabilities of the exogenous variables (i.e.,
59 sample worlds in proportion to how likely each world is). For each such world,
60 the values of the endogenous variables are then determined naturally according
61 to the structural equations. For each variable V in the causal system, compute
62 the standard deviation σ_V of the variable value across all sampled worlds (for
63 exogenous variables, this can simply be read off from the variable’s associated
64 probability distribution).

65 **b.** For each world generated that way, simulate a counterfactual ‘twin’
66 world by making an intervention on C, which sets C to a new, randomly
67 sampled value. Then the values of the endogenous variables in this twin world
68 are set naturally according to the structural equations.

69 **c.** For each pair of worlds thus generated, compute the specific causal effect

70 of C on E by taking the ratio of the change in the value of E to the change
71 in the value of C between the two worlds ($\frac{\Delta E}{\Delta C}$), and multiplying this ratio by
72 the standardizing factor $\frac{\sigma_C}{\sigma_E}$.

73 **d.** The causal score of C on E is the average of all specific causal effects
74 across all pairs of worlds. Formally, we can denote it as $k_{C \rightarrow E}$ and write it as:

$$k_{C \rightarrow E} = \frac{\sum_{i=1}^n (\frac{\Delta E}{\Delta C})_i \sigma_C}{n \sigma_E}$$

75 where n is the number of simulated world pairs.

76 The first step of the algorithm generates a large number of possible worlds,
77 ensuring that we can look at the effect of C on E across a large number of
78 different background circumstances, where these circumstances are represented
79 in proportion to how likely they are to arise. The second step looks at each
80 of these worlds in turn, asking about the strength of the causal dependence of
81 E on C in each world. Our measure of causal dependence is standardized by
82 the ratio of the standard deviation of C to the standard deviation of E. This
83 standardization is akin to what scientists do when they compute statistical
84 measures of effect size such as a Pearson’s r ; it allows measures of causal
85 effects to be unit-free (so that, e.g., the causal strength of temperature does
86 not depend on whether it is measured in Fahrenheit or Celsius). Finally, the
87 last step of the algorithm takes the average of all the causal dependence scores
88 computed in this way.

89 If C and E obey the “no-confounding assumption” (Pearl, 2000), then $k_{C \rightarrow E}$
90 is simply the correlation between C and E across worlds sampled in step **a** (we
91 prove this for the case of binary variables in the SI). The “no-confounding
92 assumption” holds when C has a causal influence on E, E does not have a
93 causal influence on C, and no variable has a causal influence on both C and E.

94 Intuitively, when this assumption holds, the relationship between C and E is
95 not confounded by third variables, so we can read the causal effect of C on E
96 from the correlation between C and E even in ‘observational’ data (i.e. data
97 which was generated without performing any intervention) (Pearl, 2000).

98 **2 Comparison with human causal intuitions**

99 When judging whether a factor is causal, people are sensitive to its statistical
100 normality (i.e. its frequency, or its probability), as well as the statistical
101 normality of other factors. The present model parsimoniously explains four
102 qualitative effects of normality on human causal judgments, most of which
103 have been replicated many times across different contexts. We show below
104 that it also provides a good quantitative fit to fine-grained data from a recent
105 set of experiments (Morris, Phillips, Gerstenberg, & Cushman, 2019). For
106 reasons of space, we also describe the four qualitative effects in the context of
107 the Morris et al. (2019) set of experiments, since these experiments exhibited
108 all four effects.

109 Morris et al. asked participants to read the following vignette:

110 A person, Joe, is playing a casino game where he reaches his
111 hand into two boxes and blindly draws a ball from each box. He
112 wins a dollar if and only if he gets a green ball from the left box
113 and a blue ball from the right box. Joe closes his eyes, reaches in,
114 and chooses a green ball from the first box and a blue ball from the
115 second box. So Joe wins a dollar.

116 Participants were asked to rate, on a 1-9 scale, their agreement with the
117 statement “Joe’s first choice (where he chose a green ball from the first box)
118 caused him to win the dollar”.

119 In a first experiment, participants saw the vignette shown above, which
120 describes a conjunctive structure (Joe needs to draw a green ball from the
121 first box, AND a blue ball from the second box, in order to win). In a second
122 experiment, another set of participants read the same vignette, minimally
123 modified so as to depict a disjunctive structure (Joe needs to draw a green
124 ball from the first box, OR a blue ball from the second box, in order to win).

125 Participants were shown pictures of the two boxes. Across conditions, the
126 experimenters systematically varied the proportion of green balls in the first
127 box and blue balls in the second box. The proportion of green balls in the
128 first box varied from 0.1 to 1, in 0.1 increments; the proportion of blue balls
129 in the second box was similarly and independently manipulated. Morris et
130 al. (2018, 2019) assessed the fit of prominent existing computational models
131 of causal judgment (Icard, Kominsky, & Knobe, 2017; Halpern & Hitchcock,
132 2015; Morris et al., 2018; Cheng, 1997; Jenkins & Ward, 1965; Spellman, 1997)
133 to their dataset.

134 Following (Morris et al., 2018), we generated predictions for two versions of
135 our model. The first version is the baseline version of the model. The second
136 version is a “normalized” version, generated with the softmax function:

$$\tilde{k}_{G \rightarrow D} = \frac{e^{k_{G \rightarrow D}}}{e^{k_{G \rightarrow D}} + e^{k_{B \rightarrow D}}}$$

137 Where $k_{G \rightarrow D}$ is the baseline causal strength ascribed to the draw of the
138 green ball, and $k_{B \rightarrow D}$ is the baseline causal strength ascribed to the draw of
139 the blue ball (Morris et al., 2018). We also considered a baseline and a nor-
140 malized version for all the models that are studied in Morris et al. (see Morris
141 et al., 2019, 2018 for a description of these models). For each causal structure,
142 we computed the predictions of our model by deriving analytical expressions

143 corresponding to the correlation between “Joe draws a green ball” and “Joe
144 wins a dollar” in the limit of an infinity of samples (see SI for derivation). We
145 studied the performance of each model in each causal structure by comput-
146 ing the item-level correlation between a model’s predictions and participants’
147 average causal ratings.¹

148 **2.1 Conjunctive structure**

149 Results are shown in Figure 1. Both the human data and the model exhibit
150 two well-known effects of statistical normality on causal judgment. The first
151 effect is *abnormal inflation*: as “drawing green” becomes less likely, causality
152 ratings for “drawing green” increase (Hilton & Slugoski, 1986; Kahneman &
153 Miller, 1986). The second effect is *supersession* : as “drawing blue” becomes
154 more likely, causality ratings for “drawing green” increase (Kominsky, Phillips,
155 Gerstenberg, Lagnado, & Knobe, 2015).

¹R code to reproduce analyses and figures is available in the electronic supplementary materials.

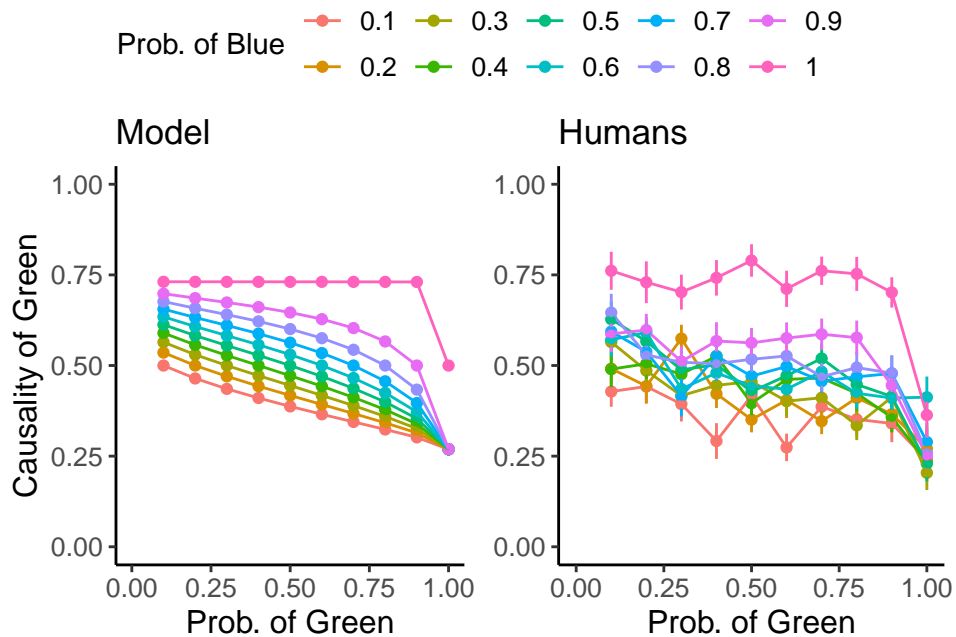


Figure 1: **Judgments made by the normalized version of the model in the conjunctive structure, along with average human judgments.** Human data are from Morris et al. (2019), and are standardized on the [0,1] interval.

156 Figure 2 shows the fit of each model to the data. The normalized version
 157 of our model had a marginally better fit than the baseline version (William's
 158 t-test, $t(97) = 1.88$, $p = .06$), and a better fit than all other models (all
 159 $ts > 6.11$, all $ps < .001$).

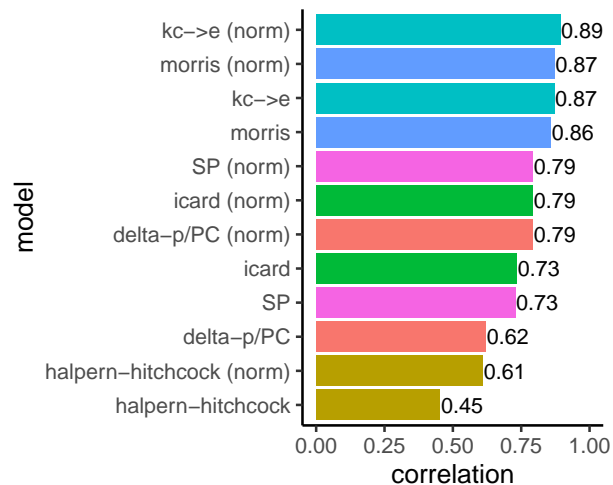


Figure 2: Fit of each model to human data, conjunctive structure.

160 2.2 Disjunctive structure

161 Results are shown in Figure 3. Both the human data and the model exhibit
 162 *abnormal deflation*: as “drawing green” becomes less likely, causality ratings
 163 for “drawing green” decrease (Icard et al., 2017; Gerstenberg & Icard, 2019;
 164 Henne, Niemi, Pinillos, De Brigard, & Knobe, 2019). They also exhibit an
 165 effect, *reverse supersession*, that had not been identified prior to the study
 166 by Morris et al.: as “drawing blue” becomes less likely, causality ratings for
 167 “drawing green” increase.

168 We note that the reverse supersession effect is relatively weak in the human
 169 data, and is mostly driven by cases where “drawing blue” is certain to occur;
 170 indeed, Kominsky et al. (2015), in a study with lower statistical power, and
 171 that did not include candidate causes that were certain to occur, were not able
 172 to find evidence for a reverse supersession effect. High-powered replications of
 173 the effect are a ripe area for future research.

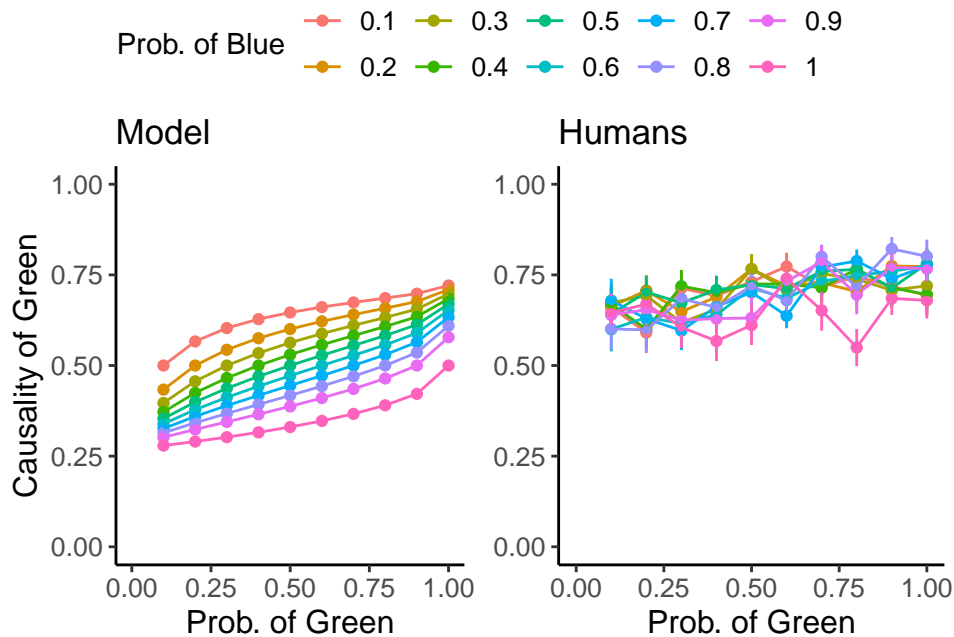


Figure 3: **Judgments made by the normalized version of the model in the disjunctive structure, along with average human judgments.** Human data are from Morris et al. (2019), and are standardized on the [0,1] interval.

174 Figure 4 shows the fit of each model to the data. The best performing
 175 models were the normalized version of our model, both versions of the Icard
 176 model and the normalized Delta-P model. None of these four models fit the
 177 data better than any other, all $ts < .73$, all $ps > .47$. The next best model was
 178 the baseline version of our model, which performed less well than the models
 179 above (all $|ts| > 3.69$, all $ps < .001$), but better than all other models (all
 180 $ts > 4.03$, all $ps < .001$).

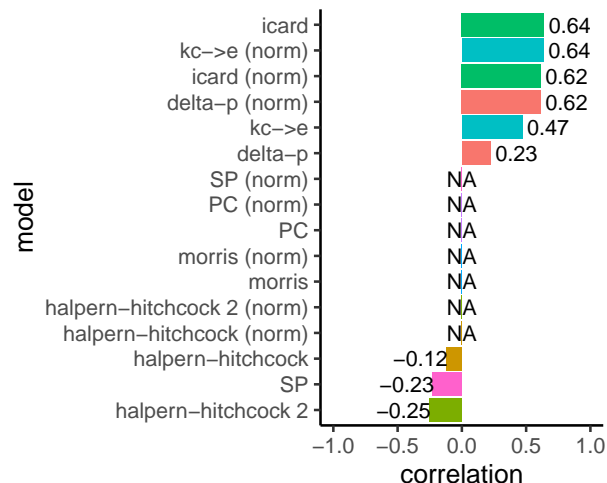


Figure 4: **Fit of each model to human data, conjunctive structure.**

181 Morris et al. (2019) also highlight interesting non-linear patterns in their
 182 data, for both experiments. Our model mostly reproduces these non-linear
 183 patterns (see SI).

184 3 Discussion

185 Our simple model provides a normative justification for the complex pattern of
 186 effects of statistical normality on causal judgment: causal cognition appears
 187 to be well-designed to identify ‘portable’ causes. Our work also provides a
 188 normative justification for the hypothesis that causal judgment relies on a
 189 process which samples counterfactuals according to their normality (Icard et
 190 al., 2017) ².

191 Another recent measure of actual causal strength, the SAMPLE mea-
 192 sure (Morris et al., 2018) can be easily derived from the present model. For

²at least as far as *statistical* normality is concerned; this could be extended to other types of normality using recent arguments by (Phillips, Morris, & Cushman, 2019)

193 any causal structure in which C and E are binary variables obeying the no-
194 confounding assumption, and C is necessary for E, the SAMPLE measure is
195 equivalent to the square of $k_{C \rightarrow E}$ (see SI).

196 Many existing measures of actual causal strength are based on the notions
197 of necessity and sufficiency (Gerstenberg, Goodman, Lagnado, & Tenenbaum,
198 2015; Icard et al., 2017; Morris et al., 2018). Necessity and sufficiency are
199 not primitives in our model, but in the special case where we assume binary
200 variables, then the $\frac{\Delta E}{\Delta C}$ term used by the algorithm reduces to a measure of
201 sufficiency (when we consider an intervention setting C from 0 to 1) or a
202 measure of necessity (for an intervention setting C from 1 to 0): C is sufficient
203 (or necessary) for E if $\frac{\Delta E}{\Delta C} = 1$.

204 Why is causal judgment well-designed to identify portable causes? The
205 present results are consistent with several possibilities. Morris et al. (2018)
206 recently argued that causal judgment serves to identify our best-bet interven-
207 tion if we want to bring about an outcome but do not know the exact state of
208 the causal system. Our model is consistent with this argument. On average,
209 we can expect that an intervention on C will result in a change of $k_{C \rightarrow E}$ stan-
210 dard deviation units in E for each one standard deviation unit change in C.
211 Therefore, if we want to set E to a certain value, we are generally better off
212 making an intervention on the variable X with the highest $k_{X \rightarrow E}$. However,
213 identifying portable causes may also be useful for a broader range of cognitive
214 activities, such as prediction or explanation. The proper evolutionary domain
215 of causal judgment remains an open question.

216 Closer to Marr’s algorithmic level of analysis (Marr, 1982), future research
217 should take a closer look at which actual causal strength measure best approx-
218 imates human judgments. Our model had the best overall fit to the Morris
219 et al. (2019) dataset, but other models (notably Icard et al., 2017) also per-

220 formed well. It will be important to extend this comparison to a wider range
221 of experimental setups (see e.g. (Sytsma, 2019) for preliminary evidence that
222 differences in study design may influence causal attributions).

223 Although very general, our model is not a full theory of causal judgment.
224 Just as other models of actual causal strength, it is relatively insensitive to
225 the specifics of what *actually* happened. Imagine that Suzy and Billy throw a
226 rock at a bottle, but Suzy’s rock gets there first. Against intuition, the present
227 model assigns positive causal strength to “Billy’s rock broke the bottle”, be-
228 cause there are possible worlds where Billy’s rock would have made a difference
229 to whether the bottle breaks. Future work should integrate the present ideas
230 with theories which can handle such cases (e.g. (Halpern & Pearl, 2005)).

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