

# Spontaneous Causal Learning While Controlling A Dynamic System

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**Abstract:** When dealing with a dynamic causal system people may employ a variety of different strategies. One of these strategies is causal learning, that is, learning about the causal structure and parameters of the system acted upon. In two experiments we examined whether people spontaneously induce a causal model when learning to control the state of an outcome value in a dynamic causal system. After the control task, we modified the causal structure of the environment and assessed decision makers' sensitivity to this manipulation. While purely instrumental knowledge does not support inferences given the new modified structure, causal knowledge does. The results showed that most participants learned the structure of the underlying causal system. However, participants acquired surprisingly little knowledge of the system's parameters when the causal processes that governed the system were not perceptually separated (Experiment 1). Knowledge improved considerably once processes were separated and feedback was made more transparent (Experiment 2). These findings indicate that even without instruction, causal learning is a favored strategy for interacting with and controlling a dynamic causal system.

**Keywords:** Decision making, dynamic systems, causal models, learning, induction.

## INTRODUCTION

We encounter complex and dynamic causal systems in both our professional and everyday lives. Indeed we need look no further than ourselves as paradigm examples of such systems. We are constituted by biochemical and neurological mechanisms that are interconnected in a complex manner and operate to maintain a delicate equilibrium. Exercise, diet, and medications are methods by which we affect our own physiological system in order to regulate it, as well as improve it. Likewise, in cases where we interact with the external environment, we often strive to control dynamic systems. For example, workers in industrial companies need to learn to control highly complex systems such as chemical plants and production lines. On a broader scale, politicians, managers, and stockbrokers act on large social systems (e.g., nations, corporations, stock markets), where social agents causally interact in various ways. People generally engage in these systems by making goal directed interventions in order to achieve their desired outcomes. Marketing campaigns, taxation, welfare, and social norms are ways of influencing people's behavior to improve the quality of life and achieve happiness.

In this paper, we are concerned with the kind of knowledge that decision makers acquire when they intervene on and control such complex systems. In particular, we

investigate whether decision makers *spontaneously* acquire causal models, that is, representations that mirror the causal mechanisms that govern the dynamic system acted upon. In other words, we are interested in whether decision makers engage in causal induction during dynamic control tasks, even when they are not asked to do so. First, we will briefly review the literature on control tasks in relation to findings about causal learning. The existing body of evidence on control tasks will be contrasted with research on causal learning, which typically does not examine learning with dynamic systems. Then we will present two experiments that examine whether people spontaneously induce causal models when learning to control a dynamic causal system. We close by discussing the empirical findings and their relevance for the common claim that decision makers fail to acquire causal knowledge in control tasks.

## CAUSAL LEARNING WITH DYNAMIC SYSTEMS

Previous research on people's ability to control complex dynamic systems (e.g., simulated industrial power plants, management systems, ecosystems) has largely neglected the role of causal knowledge. This is because theoretical accounts of skill acquisition have focused on explaining the apparent dissociation between accurate control performance and poor reportable knowledge of the structure of the system [1, 2]. Participants' poor reported knowledge is taken as evidence that very little knowledge about the underlying causal structure is acquired. This dissociation between control performance and explicit knowledge is explained in terms of the complexity of the environment. Rather than learning the un-

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derlying structure, people are supposed to learn to control the system by strengthening their knowledge of the perceptual features of the system and the specific decisions they make (i.e., what actions they chose in a given state of the system) along with the successful outcomes produced [3, 4]. By adding more cases to their knowledge base, they proceed by matching the perceived current state of the system to stored exemplars of successful situation-decision-outcome associations. Because this process relies on generating responses from memory, no inferences are made about the underlying causal relations that govern the system's behavior. While this type of exemplar-based learning is good for controlling the system to a target, it occurs at the expense of gaining knowledge of the system's structural properties. Consequently, people show limited success in transferring control skills to other goals because their knowledge is bound to tasks that have the same perceptual and goal characteristics as the original training task [5, 6].

This view contrasts sharply with current research in causal learning and reasoning. Research on these topics shows that people are able to learn about causal structure through various cues to causality, such as temporal sequence, statistical relations, consequences of interventions, and prior knowledge [7, 8]. In addition, people are not only able to learn about the structure, but also the parameters of causal models [9-11]. Thus, it has been shown that beliefs about causal structure guide the interpretation of covariational data and strongly affect the way people structure the available learning input (for a recent overview see [12]).

Recent research has also emphasized the tight connection between causal beliefs and decision making. The *causal model theory of choice* [13, 14] assumes that people use the available information to induce a causal model of the decision problem and the choice situation. A causal model of the decision problem encompasses the decision maker's knowledge about the structure of the system and their beliefs regarding the causal influences of the available courses of actions. The empirical evidence shows that people indeed use such causal model representations when making simple one-shot decisions [13, 14]. Along similar lines, Hagmayer and Meder [15, 16] investigated whether people spontaneously induce causal models when repeatedly making decisions in order to maximize payoff variables. Their results showed that many participants induced causal models and used them to make decisions. These findings demonstrate that people do not solely learn causal models when asked to do so, but engage in causal induction even when trying to achieve a very different goal. However, in these experiments non-dynamic causal systems were used (i.e., systems that did not change when participants chose not to act). Furthermore, all variables within the system were reset to their initial value on every trial. In control tasks, by contrast, the system usually has some internal momentum and the goal of the decision maker is not to maximize, but to achieve and maintain a certain equilibrium state.

### Why Causal Models?

Research on control tasks shows that people can successfully learn to control complex systems without acquiring explicit knowledge about causal structure. One might therefore wonder why decision makers should bother with causal

learning at all. The answer is that causal knowledge is more flexible and adaptive than other forms of knowledge. For example, causal model representations enable decision makers to infer the consequences of novel interventions or to evaluate changes in the structure of a causal system [8, 15, 16]. Crucially, these inferences can be derived from the structure and parameters of the causal model without the necessity of additional learning input. This is a clear advantage over non-causal representations, which require further learning whenever the causal system or the available options change. Causal knowledge is also critical when there are interferences within a dynamic system, such as when an ecosystem is on the edge of collapsing or when an industrial system such as a production line breaks down.

Consider the simple dynamic causal system depicted in Fig. (1a) and assume that the decision maker's task is to maintain the value of the final outcome variable at a certain target level. In total, there are eight causal relations within this causal system (indicated by the arrows in Fig. 1a). First, the decision maker has three options to intervene on the system (termed 'do Alpha', 'do Beta' and 'do Gamma'), whose effects are not known prior to learning. These three actions affect three intermediate variables (*A*, *B*, and *C*) which differentially contribute to the value of the final outcome: variable *A* increases the level by 80 points, *B* by 80 points, and *C* by 120 points. Crucially, outcome variable *A* not only contributes to the value of the final outcome variable, but also activates the intermediate variable *B*. As a consequence, choosing 'do Alpha' raises the level of the outcome variable by 160 points. In addition, the system is dynamic because a decay function reduces the final outcome value by 50% from trial to trial, regardless of the action taken by the decision maker. Thus, even when one does not act upon the system, the value of the target variable changes over time. Formally, the value of the final outcome variable is determined by the following equation:

$$Value_{t_1} = Value_{t_0} \cdot decay + Points | do(X)_{t_0} \quad (1)$$

For example, when the current value of the target variable is 120 and option 'do Alpha' is chosen, the resulting value is  $120 \cdot .5 + 160 = 220$  points. Note that Equation 1 can be derived from the causal model depicted in Fig. (1a), but not vice versa. For example, the equation states how the value of the outcome is influenced by a particular intervention, but is silent about the intermediate causal processes (e.g., that 'do Alpha' generates variable *A*, which in turn generates variable *B*).

Assume that the decision maker's task is to maintain the final outcome variable at a target value of 160 points by repeatedly intervening on the causal system. An exemplar-based approach supposes that participants encode which value of the target variable results from a specific action taken in a particular state of the system. Thus the experienced feedback is used to incrementally build up a knowledge base. To decide which action to take next, the current state of the system is matched to previously seen cases, and successful actions are retrieved from memory and repeated. For example, a decision maker may have learned that when the state of the target variable is very low it is useful to choose 'do Alpha', since this action entails the largest increase. Conversely, when the value of the target variable is



than psychological factors may explain the dissociation [18], and many have reported findings in which there is a close correspondence between structural knowledge and control performance [18, 19]. For example, by regularly probing knowledge of structural relations during learning, people show insight into the relations that govern the behavior of the system. In addition, they use this knowledge to control the system in subsequent phases. However, while people may have knowledge of the structure of the relations, the parameters of these relations tend to be only poorly represented [6, 20]. People may know whether a relation is positive or negative, but typically they have very little knowledge about the functional form or the strength of the relation. Crucially, research in this area has yet to examine whether these relations are causal representations of the system, and if so, how they are used to inform decisions to control an outcome to a specified criterion. In particular, if the information available to participants (instructions, feedback resulting from interventions, and background knowledge) is insufficient for inferring a causal model of the underlying system, it is not surprising that participants refrained from doing so. Whenever the underlying causal system is dynamic and consists of probabilistic non-linear mechanisms connecting partially unobservable variables, it might simply be impossible to pin down the structure and parameters of the system because the causal model is underdetermined by the available data. In this case an exemplar-based strategy seems a much better approach, because it can easily adapt to any underlying causal system, as it only represents which action is successful under specific circumstances. However, this does not mean that participants are necessarily reluctant to induce causal models in control tasks. We speculate that participants are inclined to infer the causal mechanisms of a system, because causal knowledge enables them to predict the consequences that would result from a change in the causal system. Thus, decision makers who learn to control a dynamic system should induce a causal model whenever the feedback from the system allows them to do so. We pursued this hypothesis in two experiments.

## EXPERIMENT 1

The goal of Experiment 1 was to investigate whether participants engage in spontaneous causal learning when learning to control a dynamic system. To examine the knowledge participants acquired during the control task, we employed a number of tests. In particular, we presented participants with a change in the causal system's structure. Since different kinds of representations of the system entail different reactions to such a change, this modification taps into participants' knowledge. While purely instrumental knowledge only allows for very limited inferences about the changes in effectiveness of previously used interventions, causal knowledge enables the prediction of the consequences resulting from the modification.

### Method

#### *Participants and Design*

63 students from the University of Göttingen ( $n = 31$ , 28 females, 3 males mean age = 22.8) and various universities in Berlin ( $n = 32$ , 18 females, 14 males, mean age = 25.5) participated. Participants from Berlin were paid a small

amount of money (5€); participants in Göttingen could choose between being paid and receiving course credit. Participants were assigned to one of two counterbalanced conditions that only differed in the placement of one particular test question. In one condition, they were immediately queried about the underlying causal structure after the learning phases. In the other condition they were asked about causal structure after completing all other test questions. 27 participants were assigned to the condition in which they were immediately queried about causal structure after the learning phase whereas 36 participants were asked about causal structure after completing all other tasks (the unequal distribution resulted from a miscommunication between the two research locations).

### *Materials and Procedure*

We used a computer-based biological scenario in which participants were instructed to control the level of a certain neurotransmitter in the brain of mice. In order to do so, participants could stimulate the mice's brain with three different types of rays (labeled Alpha-, Beta-, and Gamma-rays). Participants were further told that the radiation might activate different brain areas, which in turn were responsible for the production of a certain amount of the transmitter (cf. Fig. 2). It was also pointed out that the brain areas could be interconnected. No further information was given about the relations between the different types of radiation and brain activation, or between the different brain areas and levels of neurotransmitter.

Next participants were informed that the specific level of the neurotransmitter was essential to the survival of the mice. There were two different kinds of mice, each of which needed a different neurotransmitter level. One of the mice needed a transmitter level of  $140 \pm 20$  points, while the other mouse needed a level of  $280 \pm 20$ . Then participants were asked to maintain the neurotransmitter level on a number of subsequent trials. To maintain the transmitter level, they could stimulate the brain with Alpha-, Beta- or Gamma-rays on each trial (henceforth denoted as 'do Alpha', 'do Beta', and 'do Gamma'). They also had the option to not stimulate the brain ('do nothing').

Fig. (1a) shows the causal system underlying the control task. The level of the neurotransmitter had a decay of 50% on each trial (i.e.,  $value_{t1} = value_{t0} \cdot .5$ ), regardless of the action taken. Alpha radiation activated one of the brain areas (area A), which in turn activated a second area (area B). The joint activation of these two areas resulted in a total increase of the transmitter level of 160 points. Beta radiation activated area B and resulted in an increase of 80 points. Finally, Gamma radiation activated the third brain area (area C), thereby increasing the transmitter level by 120 points. (The causal relations from radiation to brain areas on the one hand and brain areas and amount of produced transmitter were counterbalanced across participants.) Participants were never told the causal model's parameters or the functional characteristics of the system. Thus, the underlying causal relations had to be inferred on the basis of the consequences resulting from the interventions. The radiation as well as the subsequent activation of the brain areas were presented through animations (cf. Fig. 2). First, participants saw which brain areas became activated due to the chosen intervention. If





brain area  $C$  and the resulting value of the transmitter would be  $(200 \cdot .5) + C = 220$ . Since the value of the decay was known from the initial observation phase there is only one unknown in the equation. Thus, the equation can be solved for  $C$  to determine how much transmitter is produced by brain area  $C$  (i.e.,  $C = 220 - (200 \cdot .5) = 120$ ). To check whether participants learned to maintain the target level of the transmitter, we computed the distance from the target for each trial. Fig. (3b) depicts the resulting learning curve averaged over participants. It shows that participants learned to control the system, although there was little transfer between the two target levels (see the sharp increase in distance on trial 21 in Fig. 3b). A within-subjects ANOVA comparing the average distance of the first ten trials to the average distance of the last ten trials yielded a clear effect,  $F(1,62) = 10.5, p < .01, MSE = 277.4$ . Taken together, these results suggest that participants successfully learned to maintain the target value in the control task.

### Causal Model Selection Task

As noted previously, we expected that participants would correctly identify the causal structure of the system. This was in fact the case: 84% of participants correctly chose the causal chain model over the independent causes model, which is significantly higher than 50%,  $\chi^2(df = 1, N = 63) = 29.3, p < .01$ . This finding indicates that participants were sensitive to the structure of the causal system underlying the control task. Also, participants' model choices were not affected by the measurement point,  $\chi^2(df = 1, N = 63) = .04, p > .80$ . We therefore pooled the data for the subsequent analyses.

### Intervention Choices

Next we analyzed decision makers intervention choices in Test Phases 2a and 3a, in which they were asked to make four intervention decisions for the original system (comprising all three variable  $A, B$ , and  $C$ ) and the modified causal system in which variable  $B$  was removed. Fig. (4) depicts the results.<sup>1</sup> On the left hand side choices are depicted for the causal system participants acted upon in the control task. If participants correctly inferred the consequences of the different interventions, they should prefer intervention 'do Beta' for target level 140 since  $(current\ level \cdot decay) + Points/do\ Beta = (120 \cdot .5) + 80 = 140$  and  $(160 \cdot .5) + 80 = 160$ . For target level 280 'do Alpha' is the best option because the other options result in less points:  $(current\ level \cdot decay) + Points/do\ Alpha = (120 \cdot .5) + 160 = 220$  and  $(160 \cdot .5) + 160 = 240$ . Although not perfect, the most frequent choices conformed to these predictions. On the right hand side of Fig. (4) choices are depicted for the modified causal system in which area  $B$  (the area normally activated by Beta radiation) is removed. Based on the modified causal model, participants should now have preferred intervention 'do Alpha' for target level 140 because  $Points/do\ Beta_{B\_removed} = 0$  and  $Points/do\ Alpha_{B\_removed} = 80$ . For target level 280 'do Gamma' should now be chosen because  $Points/do\ Gam-$

$ma_{B\_removed} = 120$ , that is 'do Gamma' now has a higher impact than 'do Alpha'. Thus, participants should exhibit a differential choice pattern for the original and the modified causal system. In contrast to this prediction, Fig. (4) indicates that participants tended to stick with the choices they made for the system they initially acted upon. To test whether participants changed their preference we conducted a Bowker test for choices with respect to a target level of 140. A Bowker test investigates whether frequencies of different categories (i.e., chosen options) stay the same for two measurements. The test confirmed that a number of participants systematically switched away from choosing 'do Beta',  $\chi^2(df = 1, N=126) = 22.1, p < .001$ . However, not all of them turned to 'do Alpha' as we expected; a substantial number now preferred 'do Gamma'. For a target level of 280 a Bowker test could not be run due to low cell entries. Instead we conducted a McNemar test focusing on the 'do Alpha' and the 'do Gamma' choices. In line with the predictions derived above, participants switched away from choosing 'do Alpha' and now preferred more often 'do Gamma', but still most participants continued with 'do Alpha',  $\chi^2(df = 1, N=126) = 17.3, p < .001$ . Thus, it does not seem as if decision makers used a correctly parameterized causal model to make their choices.

### Predicted Values

In Test Phases 2b and 3b participants were asked to estimate the transmitter values resulting from all possible interventions (including the option of 'do nothing'). Estimates were requested for two starting values (140 vs. 280) for both the original and modified causal system. Table 1 depicts the results for the 16 estimates as well as the values entailed by the underlying causal model. For the system participants acted upon in the control phase, they generally underestimated the transmitter level resulting from the interventions. However, the rank order of the estimates corresponds to the order entailed by the underlying causal model.

The crucial analyses concern the comparison of 'do Alpha' and 'do Beta' before and after removing variable  $B$  from the causal system. A causal model analysis entails that the ratings for 'do Alpha' and 'do Beta' should decline. The qualitative pattern of estimates conformed to these predictions. Multiple planned comparisons revealed significant differences for 'do Alpha' ratings before and after the modification for both starting values of 140 and 280 (cf. Table 1). The difference for 'do Beta' ratings before and after the modification was only significant for a starting value of 140. The difference for starting value 280 ( $p = .055$ ) missed our error type I corrected criterion of significance. However, the obtained mean estimates also indicate that participants did not decrease their estimates as much as entailed by the underlying causal model. Since the removal of area  $B$  completely wipes out the causal impact of 'do Beta' on the transmitter level the predicted values of 'do Beta' should equal the ratings for 'do nothing'. However, only 16 out of 63 participants made this inference at a current value of 140, and only 20 participants at a current value of 280. In line with our predictions the small differences obtained for the estimates of 'do Gamma' and 'do nothing' failed to reach significance for both starting values. Overall, the findings indicate some evidence for participants' sensitivity to the causal model modification, but the results also show that

<sup>1</sup>We ran tests to check whether asking participants first about causal structure affected their choices. In order to avoid low cell entries we classified participants' choices as predicted vs. not-predicted for all eight test cases. Then we conducted  $\chi^2$ -test for each test case separately comparing the choices in the two counterbalancing conditions. Only one significant difference resulted. Participants more often chose do Alpha after the modification given a starting value of 120 and a target of 140 when they were first asked about causal structure ( $\chi^2 = 8.82, p < .01$ ). As this was the only difference, we report the choices for both conditions together in Fig. (4).





pha', +20 for 'do Beta', '+70 for 'do Gamma', and -75 for 'do nothing'. The mean change heuristic then predicts that the decision maker would choose 'do Beta', since  $120 + 20$  comes closest to the desired target value.

To capture the implications of the removal of area *B* we assumed that decision makers simply subtracted the experienced mean change of 'do Beta' from the experienced mean change for interventions 'do Beta' and 'do Alpha'. Thus, participants following this encoding strategy should assume that the intervention 'do Beta' is not any longer effective after removing *B* from the causal system (Fig. 1b). Note that subtracting the mean change of 'do Beta' from 'do Alpha' does not imply sensitivity to causal structure. It merely assumes that participants were sensitive to the fact that a choice of 'do Alpha' was followed by an activation of areas *A* and *B*.

### Exemplar-Based Strategy

The second strategy is an implementation of the generalized context model [22]. The generalized context model is one of the dominant exemplar models in categorization, but it can also be used to model repeated decision making. We chose this model because it has been widely used and proved highly successful in the area of judgment and decision making [23]. The exemplar-based strategy assumes that participants encode each trial they encounter. More specifically, for each trial the starting value of the transmitter, the action chosen, the activated areas and the resulting change in the outcome value are assumed to be stored. To predict the consequences of an option on a new trial, the starting value of this particular trial is compared to the starting values of all previous trials in which the same option had been chosen. Previous outcome changes are weighted depending on the similarity of each previous starting value to the starting value of the current trial. Trials resembling the current trial are weighted more. Identical trials receive the maximum weight. By integrating the weighted changes of outcomes from previous trials an estimate of the outcome value for the considered option for the current trial is derived. Estimates for all options are compared to the target value to identify the best option.

To model this strategy, we first computed the change participants observed for each type of intervention (i.e., do Alpha, do Beta, do Gamma, and do nothing) on each trial,  $change_{j, do(x)}$ . Then we computed the absolute distance of each starting value of each test trial to the starting values of all trials seen before [22]. For current trial *i* and previous trials  $j_1 \dots j_n$  the distance was defined as

$$d_{ij} = |value_{i,t_0} - value_{j,t_0}| \quad (2)$$

Next, we transformed absolute distances into psychological distances reflecting perceived similarities. To do so we used the exponential transformation proposed in the generalized context model [22]:

$$\eta_{ij} = e^{-d_{ij}} \quad (3)$$

These similarities were then used to weight the observed outcome change on each trial on which the same option was chosen. By summing up over the weighted observed outcome changes and adding the current starting value the outcome value for each option was predicted. The following formula was used:

$$value_{i,do_x,t_1} = \sum_{j=1}^{j=n} \eta_{ij} \cdot change_{j,do_x} / \sum_{j=1}^{j=n} \eta_{ij} + value_{i,t_0} \quad (4)$$

For example, suppose a participant chose 'do Beta' twice, once with a starting value of 100 points resulting in a change of +30 points and once with a starting value of 150 resulting in a change of -15 points. Now the participant is confronted with a starting value of 140 and tries to predict the resulting value using these two previous trials. The distances of the new starting value to the previous starting values are 40 and 10 points respectively, therefore similarities are  $4.25 \cdot 10^{-18}$  and  $4.54 \cdot 10^{-5}$ . Thus the predicted value is  $(4.25 \cdot 10^{-18} \cdot 30 + 4.54 \cdot 10^{-5} \cdot (-15)) / (4.25 \cdot 10^{-18} + 4.54 \cdot 10^{-5}) + 140 = 125$ . As the example shows, closer previous trials exhibit a much stronger influence on the prediction. Predicted values for each option were then compared to the target value. The option whose value comes closest to the desired target value is chosen.

To capture the implications of the removal of area *B* we assumed that decision makers would use the knowledge they had about cases in which only area *B* was activated before. These were the cases in which the option 'do Beta' was chosen. Therefore the removal of *B* can be accounted for by subtracting the predicted value of option 'do Beta' from the predicted value of 'do Alpha' and 'do Beta' for each particular test case. Thus,

$$value_{do\_Alpha|B_{removed},t_1} = value_{do\_Alpha,t_1} - value_{do\_Beta,t_1} \quad (5)$$

Imagine that the value of 'do Alpha' has to be predicted for a starting value of 160 points after area *B* had been removed. Then the predicted values for both 'do Alpha' and 'do Beta' are computed. Let's assume that the resulting values are +80 for 'do Alpha' and -1 for 'do Beta'. Hence the predicted value of 'do Alpha' given a removal of area *B* would be  $160 + 80 + 1 = 241$ . Note that this subtraction does not imply that participants are sensitive to causal structure. Like for the mean change strategy it merely entails that participants are sensitive to the fact that 'do Alpha' is followed by an activation of areas *A* and *B*.

### Normative Causal Model Strategy

The third strategy assumes that participants use the information about the decay and the feedback on the interventions to infer the structure and the parameters of the underlying causal system. Hence, it presupposes that the actual parameters of the causal system are inferred. Choices and estimates are then assumed to be based on the correctly parameterized causal model. Predictions derived from this normative causal model have been presented in the previous sections (cf. Table 1). We decided to include this strategy as a benchmark, although the analyses presented above indicate that participants in general did not follow this strategy.

### Subjective Causal Model Strategy

The fourth strategy assumes that participants engaged in causal learning and correctly inferred the underlying causal structure, but failed to derive correct parameter estimates. Thus, it may be the case that participants engaged in *causal* decision making, but based their choices and predictions on their *subjective* estimates of the causal model's parameters. These subjective parameter estimates may deviate from the

actual parameters since extracting the causal model's parameters required participants to disentangle the impact of the different interventions from the influence of the decay.

To model this strategy, we used participants' estimates for the consequences of the interventions to derive their individual parameter estimates of the causal model. We first reconstructed participants' assumptions about the decay by dividing their individual predicted value estimates for the consequences of 'do nothing' before the causal structure modification by the respective starting value (140 or 280) and then taking the mean of these two estimates. Assume a participant estimated that 'do nothing' at current transmitter values of 140 and 280, respectively, would result in values of 90 and 160. The subjective estimates for the decay would then be  $(90/140 + 160/280) / 2 = .61$ .

Next we calculated the supposed influence for the other interventions from the predicted value estimates given for the original causal model. To do so, we multiplied the starting values by the participants' subjective estimate of the decay and then subtracted the result from the participants' subjective estimate for the resulting transmitter value for this intervention. Assume a participant predicts that the resulting transmitter level would be 220 for 'do Alpha' at a current value of 140. Her subjective estimate of the decay was .6. The subjective estimate for the impact of 'do Alpha' would then be  $220 - (140 \cdot .6) = 136$ . We again averaged the results for each intervention across the two starting values (140 and 280). Note that the resulting parameter estimates were based on the ratings participants gave before the causal system was modified. In order to predict the parameters of the modified causal system, the parameter for 'do Beta' was subtracted from the derived estimates of 'do Alpha' and 'do Beta'. Hence, the parameter values decreased to zero for 'do Beta' and remained unchanged for 'do Gamma' and 'do nothing'. These individual parameter estimates (including the decay) were then used to predict the individual choices in Test Phases 2a and 3a (i.e., the intervention choices made for the original and modified causal system) and the predicted values for each intervention after area B had been removed (Test Phase 3b).

### Subjective Causal Model Without Decay

A fifth possible strategy is that participants induced a causal model and estimated its parameters, but assumed that no decay would be present once an intervention was taken. That is, decision makers may have assumed that stimulating the brain by means of radiation eliminates the transmitter's decay. Causal Bayes net theories in fact make the assumption that certain ('strong') interventions eliminate all other causal influences on the variable intervened on [24]. Another reason for not assuming decay when acting upon the system might have been that the impact of the decay was not directly observable during the control task. The decay was only explicitly observed when the 'do nothing' option was chosen, otherwise the transmitter level was merely observed to change up- or downward. The assumption of no decay given interventions entails different parameters for the causal model. To calculate these parameters we again used participants' ratings of predicted values in Test Phase 2b (i.e., the predicted value estimates for the unmodified causal system). The only difference to the previous model is that the decay is ignored when the impact of the three

interventions is calculated. Otherwise the calculation is identical to the previous strategy. The same holds for the derivation of the predictions.

### Evaluating the Decision Models

To evaluate the different strategies we compared the models' predictions with participants' individual choices and ratings. For the choice task, the number of correctly predicted interventions was computed separately for each participant.<sup>3</sup> The number of correct predictions ranged from zero (none of the choices was predicted) to four (every choice made was predicted by the model). The resulting distributions for all strategies are depicted in Fig. (6). For example, the normative causal model correctly predicted all four choices made by a participant with respect to the system observed during learning (i.e., before modification) for 15 participants, it predicted 3 out of 4 choices for another 34 participants, 2 out of 4 for ten participants, and only 1 out of 4 for 4 participants. There were no participants for which the normative causal model failed completely. In Fig. (6) also the prospective results from a random choice model are depicted. Random choices were modeled by a binomial distribution with  $n = 4$  choices and a probability of  $p = .25$  to predict the actual choice by chance. If participants chose randomly, then none of a participant's choices should be correctly predicted for 20 participants, only one choice for 27 participants, two choices for 13 participants and 3 out of 4 choices for 3 participants. Given random choices no complete matches are expected (see Fig. (6) black lines and diamonds).

As Fig. (6a) shows, all strategies predicted participants' choices before the modification of the causal system better than the random choice model. The resulting distributions all deviated significantly from the random binomial distribution ( $\chi^2_{\text{mean\_change}} = 1963.0, p < .001$ ;  $\chi^2_{\text{exemplar}} = 1179.0, p < .001$ ;  $\chi^2_{\text{causal\_normative}} = 1250.9, p < .001$ ;  $\chi^2_{\text{causal\_subjective+decay}} = 1239.7, p < .001$ ;  $\chi^2_{\text{causal\_subjective+no\_decay}} = 1260.2, p < .001$ ). Hence, all strategies can predict the decisions made by the participants with respect to the system they intervened upon in the initial control task. A different picture arises for the intervention choices made regarding the modified causal system in which variable B was removed. As shown in Fig. (6b) only the mean change heuristic, the exemplar-based strategy and the subjective causal model without decay were able to predict participants' choices, although the predictions from the other strategies also deviated from the random choice model ( $\chi^2_{\text{mean\_change}} = 179.4, p < .001$ ;  $\chi^2_{\text{exemplar}} = 123.3, p < .001$ ;  $\chi^2_{\text{causal\_normative}} = 17.0, p = .002$ ;  $\chi^2_{\text{causal\_subjective+decay}} = 61.2, p < .001$ ;  $\chi^2_{\text{causal\_subjective+no\_decay}} = 190.6, p < .001$ ). Thus, only strategies not explicitly taking the system's momentum (i.e., the decay of the outcome value) into account were able to predict participants' choices before and after the causal modification. Moreover, the three strategies had almost the same predictive power (cf. Fig. 6b).

<sup>3</sup>We decided to include all participants in the evaluation of the five models. Although it would be interesting to see whether participants who correctly identified the underlying causal model used different strategies, we decided not to split up participants into subgroups. The reasons are that (i) participants had a probability of 50% to pick the true model in the model selection task by chance, and (ii) all other conceivable indicators of causal assumptions would have to be derived from the variables that are used in modeling.







**Table 2. Mean Estimates of Effects Resulting from Interventions in Experiment 2. Numbers in Italics are Predictions Derived from a Correctly Parameterized Causal Model, Numbers in Brackets are Standard Errors**

	Target & Start =140		Target & Start = 280	
	Before	After	Before	After
do Alpha	<i>230</i>	<i>150</i>	<i>300</i>	<i>220</i>
	199.8	155.6	315.2	274.8
	(4.82)	(4.98)	(5.18)	(8.57)
	$F(1, 59) = 66.5; p < .001^{**}$		$F(1, 59) = 32.4; p < .001^{**}$	
do Beta	<i>150</i>	<i>70</i>	<i>220</i>	<i>140</i>
	150.9	111.0	248.4	215.1
	(4.37)	(5.07)	(6.03)	(8.51)
	$F(1, 59) = 53.8; p < .001^{**}$		$F(1, 59) = 14.3; p < .001^{**}$	
do Gamma	<i>190</i>	<i>190</i>	<i>260</i>	<i>260</i>
	163.3	156.4	269.1	266.1
	(4.17)	(3.92)	(4.77)	(5.29)
	$F(1, 59) = 4.33; p = .04$		$F(1, 59) < 1; p = .58$	
do Nothing	<i>70</i>	<i>70</i>	<i>140</i>	<i>140</i>
	74.3	76.2	155.2	156.6
	(1.81)	(2.22)	(4.69)	(4.63)
	$F(1, 59) = 1.79; p = .19$		$F(1, 59) < 1; p = .44$	

Note. For all interventions and target values separate ANOVAs were calculated with counterbalancing condition (causal structure test first vs. causal structure test last) as a between-participants variable and modification (before vs. after) as a within-participants variable. To correct for accumulation of type 1 error the significance level was set to  $\alpha = .00645$ . It turned out that neither the counterbalancing condition nor the interaction of the two factors reached significance. Therefore only the main effects of the modification of the causal system are reported in Table 2. Results marked with an asterisk are significant.

number of choices matching to the actual choices was counted. The resulting distribution of matches was again compared to a binomial distribution modeling random choices (binom( $n = 4, p = .25$ )). Fig. (9) shows the distributions of the strategies and the random choice model. For the original system, all strategies achieved a better fit than the random choice model ( $\chi^2_{\text{mean\_change}} = 1565.4, p < .001; \chi^2_{\text{exemplar}} = 1846.2, p < .001; \chi^2_{\text{causal\_normative}} = 1252.3, p < .001; \chi^2_{\text{causal\_subjective+decay}} = 1675.4, p < .001; \chi^2_{\text{causal\_subjective+no\_decay}} = 371.5, p < .001$ ). This was also true for the modified causal system:  $\chi^2_{\text{mean\_change}} = 56.0, p < .001; \chi^2_{\text{exemplar}} = 106.0, p < .001; \chi^2_{\text{causal\_normative}} = 142.5, p < .001, \chi^2_{\text{causal\_subjective+decay}} = 280.9, p < .001; \chi^2_{\text{causal\_subjective+no\_decay}} = 113.1, p < .001$ . Looking at the distribution and the  $\chi^2$ -values, the subjective causal model with decay strategy seems to have done best. It predicted at least 3 out of the 4 choices for 16 of the participants; the subjective causal model without decay did the same for 8 participants, the mean change heuristic for 7, and the exemplar based strategy for 4. For the remaining participants the models either did not yield differential predictions or were not able to account for at least 3 out of the 4 predictions. These results indicate that participants probably used a variety of strategies. However, most seem to have preferred a causal strategy.

In a second step, we also evaluated the models' predictions for the predicted value estimates. We again computed normalized root mean squared errors (NRMSE). The results mirror the findings obtained for the intervention choices,  $\text{NRMSE}_{\text{mean\_change}} = .393, \text{NRMSE}_{\text{exemplar}} = .304, \text{NRMSE}_{\text{causal\_normative}} = .229, \text{NRMSE}_{\text{causal\_subjective+decay}} = .214, \text{NRMSE}_{\text{causal\_subjective+no\_decay}} = .261$ . The subjective causal model with decay strategy fitted participants' ratings significantly better than the mean change heuristic ( $t(60) = 5.85, p < .01$ ), the exemplar-based strategy ( $t(60) = 3.25, p < .01$ ), and the subjective causal model without decay ( $t(60) = 2.80, p < .01$ ). There was no difference to the normative causal strategy ( $t(60) = 1.18, p = .24$ ).

**Summary**

Participants were confronted with the same causal system in both Experiment 1 and 2. The only difference was that in Experiment 2 information about the causal impact of the decay was provided when decision makers intervened on the system. As before, the majority of participants was able to correctly infer the structure of the underlying causal system. In contrast to the results of Experiment 1 we now found that participants systematically adapted their choices and estimates when the structure of the causal system was modified. In accordance with the predictions of a causal model ap-



change heuristic) or base new decisions on memory retrieval (e.g., the exemplar based strategy) cannot account for our results. These approaches cannot explain how people made correct causal model judgments about the intermediate variables, nor why they changed their choices and estimates systematically after the structure of the system had been modified. The modeling results showed that these alternative models can account for participants' choices from the first experiment, but not from the second. Moreover, participants' estimates of the values resulting after the modification were best predicted by a causal model strategy in both experiments (without decay in Exp. 1 and with decay in Exp. 2). Thus, although some participants probably employed alternative strategies, the induction of causal models seems to be the strategy favored by the majority of participants.

Consistent with the findings presented here is a set of recent studies showing that people tend to induce causal model representations when repeatedly intervening on a static causal system in order to maximize a specific payoff [15, 16]. For example, using a non-dynamic causal system Meder and Hagmayer showed that participants spontaneously induced causal models from probabilistic data and made appropriate inferences for both novel options and the removal of variables [16]. The experiments were designed to pit causal model theory against instrumental learning models, which entails that decision makers only acquire action-outcome contingencies. The results showed that decision makers induced a causal model representation of the choice task and acquired causal knowledge that went beyond mere action-outcome contingencies.

### Conditions Promoting Causal Learning During Control Tasks

The present experiments indicate that decision makers have the capacity to engage in causal learning during control tasks. What are the conditions that enable and support such learning? Generally, it has been argued that the lack of evidence for causal learning found in previous studies results from methodological rather than psychological factors [18]. This argument also receives empirical support. For example, when decision makers are probed about their structural knowledge during the control task, they show improved insight into the underlying causal structure and capitalize on this knowledge for regulating the system [18]. Thus, in contrast to the traditional view on control learning, it seems that decision makers can go beyond solely procedural skills and acquire explicit causal knowledge.

Another factor that might promote causal learning is the kind of system acted upon. The simulated environments used to study control tasks often comprise several continuous input variables that are connected to several continuous outputs. Typically, inputs and outputs are linked by complex, nonlinear functions. In such situations, the precise form of the functional relations may simply be underdetermined by the observable data. In this case, knowledge of previously successful actions with respect to certain circumstances allows for better decisions. Unfortunately, this strategy may further constrain the observed data. For example, once a learner has achieved a specific equilibrium and knows how to maintain it, she will probably refrain from exploring other actions that may lead her away from the goal state [27]. As a

consequence, the chosen inputs and observed outputs are likely to stay in a certain range of values, thereby making it even more difficult to reveal the underlying functions. A particular kind of underdetermination of causal relations by data is also found in our studies. Although decision makers knew that a decay took place when they did not intervene on the system, the feedback they received in Experiment 1 did not allow them to infer whether the decay still maintained its influence once they acted upon the system. As a consequence, they learned little about the causal parameters. By contrast, when participants were provided with information about the impact of the decay while learning to control the system (Experiment 2), they acquired more causal knowledge.

Finally, it is probably crucial that the number of causal hypotheses is constrained by cues to causality, such as background knowledge about causal mechanisms, and the time course of events. The more knowledge a person can bring to bear, the easier it is to pinpoint the underlying causal system [8, 28, 29]. In addition, the feedback provided to participants is critical for causal induction. For example, if in our studies the status of the intermediate variables subsequent to an intervention had not been observable, it would have been impossible to infer the structure of the system and to evaluate any structural changes. In such a situation causal learning would be restricted to mere action-outcome contingencies. Imagine you turn the key of your car and it does not start. Without further information about the state of the intermediate causal variables (e.g., fuel, battery, ignition) you will be unable to identify the problem.

When these prerequisites are met, other conditions may further enhance causal induction. First, free exploration of the causal system without any specific goal has been shown to improve structure learning [20]. While in control tasks the acquisition of knowledge about the system conflicts with keeping the system in a particular state, free exploration allows the system's behavior to be scrutinized under a wide range of circumstances. The empirical evidence shows that participants who are instructed with a specific goal (i.e., maintaining a certain equilibrium state) perform worse than participants who are allowed to freely explore the dynamic system [20]. Second, motivation may also affect causal learning. Personal involvement and high stakes create the motivation to scrutinize information more carefully [30]. Third, personality factors like "need for cognition" and intelligence contribute to successfully controlling a system and learning about its structure [31, 32]. Fourth, expertise can both hamper and promote causal induction. Expertise is in general connected to elaborate background knowledge, which constrains possible causal hypotheses, so that causal learning becomes easier. On the other hand, experts have a huge knowledge base of previously encountered cases, which often allows for exemplar-based decision making. Research on naturalistic decision making has shown that experts tend to use their exemplar knowledge when a given case resembles previously encountered situations, but rely more on causal induction and mental simulations when they cannot retrieve an appropriate case from memory [33]. Finally, knowing in advance about possible changes of the causal system may create additional motivation to engage in causal learning. As pointed out before, causal representa-

tions, but not other forms of knowledge, allow people to quickly react to changes in the causal system, including the arrival of new options [15, 16].

## CONCLUSION

The findings reported in this paper indicate that people spontaneously induce causal models when they make judgments and decisions about a dynamic system. They do this even when simpler learning and decision making strategies would have sufficed for the control task assigned to them. We take this as evidence that people prefer to induce causal models so long as the situation allows for causal learning. Only when the task structure does not allow them to engage in causal learning will they resort to simpler strategies. Hence, we do not claim that when people control a dynamic system they will always succeed in learning the underlying causal model, but we suspect that they will often try.

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