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Repeated Causal Decision Making

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Many of our decisions refer to actions that have a causal impact on the external environment. Such actions may not only allow for the mere learning of expected values or utilities but also for acquiring knowledge about the causal structure of our world. We used a repeated decision-making paradigm to examine what kind of knowledge people acquire in such situations and how they use their knowledge to adapt to changes in the decision context. Our studies show that decision makers' behavior is strongly contingent on their causal beliefs and that people exploit their causal knowledge to assess the consequences of changes in the decision problem. A high consistency between hypotheses about causal structure, causally expected values, and actual choices was observed. The experiments show that (a) existing causal hypotheses guide the interpretation of decision feedback, (b) consequences of decisions are used to revise existing causal beliefs, and (c) decision makers use the experienced feedback to induce a causal model of the choice situation even when they have no initial causal hypotheses, which (d) enables them to adapt their choices to changes of the decision problem.

Keywords: decision making, causal learning, causal models, decisions from experience

In this article, we examine the interplay between causal learning and decision making. We are interested in two questions: Do decision makers acquire knowledge about causal structure when repeatedly acting upon a causal system in order to maximize the state of a target variable? And does this causal knowledge help people to adapt to changes in the decision task?

Our main hypothesis is that decision making is often contingent on causal considerations and that people rely on causal learning and reasoning to assess decision problems and adapt their choices accordingly. For example, cancer is often treated by chemotherapy, but the potential benefits of this treatment depend on a number of factors. When the patient's liver is working properly, chemotherapy is often the most promising treatment. However, when a patient suffers from liver dysfunction, which prevents metabolizing the chemicals, it may be necessary to switch to a different treatment, such as radiation therapy. Thus, the outcomes of the available courses of action depend on the characteristics of the underlying causal system. Causal knowledge enables the decision maker to determine which of the available options is most promising under the prevailing circumstances.

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Despite the importance of causal knowledge, most theories of decision making neglect the role of causal beliefs. The canonical normative model of decision making—expected utility theory (Savage, 1954; von Neumann & Morgenstern, 1944)—distinguishes between options, possible outcomes, and the associated probabilities. These accounts implicitly assume that likelihood estimates correctly mirror causal relations, although observable statistical ("evidential") relations may not necessarily reflect underlying causal processes (Hagmayer & Sloman, 2009; Joyce, 1999; Nozick, 1993; Sloman & Hagmayer, 2006). For example, there is a statistical relation between buying running shoes and being in good shape, but buying shoes does not causally affect a person's physical fitness. Only running (i.e., the common cause) will lead to being fitter. Thus, distinguishing between probabilities that reflect causal relations and probabilities referring to spurious associations is important for making good decisions.

Other theories acknowledge the importance of causal relations in decision making (e.g., Joyce, 1999; Nozick, 1993; Skyrms, 1980). A major goal of these approaches is to distinguish "evidential" from "causal" expected utilities. While evidential expected utilities are derived from probabilities that merely reflect statistical relations, causal expected utilities only take into account probabilities that reflect causal relations. A problem that these approaches encountered was the lack of a formal modeling framework to distinguish between causal and noncausal probabilistic relations in a rigorous manner. Recent progress in causal modeling based on graphical causal models and causal Bayes nets theory (Pearl, 2000; Spirtes, Glymour, & Scheines, 1993; see also Dawid, 2002) offers a potential solution to this problem.

The *causal model theory of choice* (Hagmayer & Sloman, 2009; Sloman & Hagmayer, 2006) is a descriptive model based on these theoretical advances, assuming that people often use the available information to induce a causal model representation of the decision situation. A causal model of a decision problem encompasses knowledge about the structure of the system targeted by the

intervention and the causal relations between actions, outcomes, and payoffs. Such a model enables decision makers to simulate the causal consequences of the available courses of action, thereby ensuring that decisions are based on causal and not merely on statistical relations.

Research has shown that people indeed use causal models and causal inference when making simple one-shot decisions (Hagmayer & Sloman, 2009). However, in these studies, participants only made decisions based on hypothetical scenarios without actually experiencing the consequences of the actions taken. Research on causal learning, reasoning, and decision making has not yet investigated to what extent people learn about causal structure and use causal knowledge when repeatedly making decisions in order to achieve a certain goal, such as maximizing a payoff.

Findings from research on dynamic decision making indicate that people are able to learn to successfully manipulate and manage complex causal systems (Osman, 2010). However, this research also indicates that decision makers usually acquire only limited knowledge about the causal structure of the system (Berry & Broadbent, 1995; but see Hagmayer, Meder, Osman, Mangold, & Lagnado, 2010). Instead, people seem to learn to control causal systems by strengthening the association between perceptual features of problems and successful actions (Dienes & Fahey, 1995) or by storing instances of previous actions and their outcomes in memory (Lipshitz, Klein, Orasanu, & Salas, 2001). One explanation for these findings is that the evidence available to participants in these studies was insufficient for causal learning (see the General Discussion for limits of causal induction).

Causal Learning and Decision Making

Several studies support the idea that people have the capacity to combine causal learning with decision making. People are able to induce a causal model of a system from cues like temporal order, covariation information, active interventions, and knowledge acquired through social learning (Lagnado, Waldmann, Hagmayer, & Sloman, 2007). There seems to be an advantage of causal learning from active interventions (i.e., self-selected actions upon a causal system) over learning from passive observations, which seems to be driven by the fact that interventions introduce a temporal asymmetry between cause and effect (Lagnado & Sloman, 2004, 2006; see also Greville & Buehner, 2010; Steyvers, Tenenbaum, Wagenmakers, & Blum, 2003). People also have the capacity to integrate separately learned causal relations into more complex models and to draw inferences from them (Ahn & Dennis, 2000; Baetu & Baker, 2009; Darredeau, Baetu, Baker, & Murphy, 2009; Hagmayer, Meder, von Sydow, & Waldmann, 2011). Causal knowledge is also used as a metacue for searching and selecting cues in multiple-cue judgment (Garcia-Retamero, Wallin, & Dieckmann, 2007). Finally, people are able to use their knowledge of causal structure to predict the consequences of novel interventions on a causal system (Meder, Hagmayer, & Waldmann, 2008, 2009; Sloman & Lagnado, 2005; Waldmann & Hagmayer, 2005).

This work shows that people have the capacity to derive causal model representations from experience when requested to do so and rely on causal knowledge when asked to make decisions pertaining to causal systems. However, it is unclear to what extent people induce causal models when they have other goals than revealing the causal structure of the environment. It could be that causal learning only takes place when this is the primary goal. But it could also be that there is a natural tendency to learn about causal relations, even when a decision maker pursues another goal.

Learning and Representation in Repeated Decision Making

In many real-world situations, people repeatedly have to choose among actions that constitute active interventions on a causal system (e.g., medical treatments). When making decisions, the goal usually is to achieve a certain outcome (e.g., to cure a disease) and not to learn about causal structure (e.g., the physiological setup of a particular patient). Nevertheless, the observed consequences of actions and their probabilities reflect the causal mechanisms within the system. In addition, observable cues (e.g., the temporal order of effects, direct vs. indirect effects of interventions) may provide hints to the underlying causal structure. Because repeated interventions may allow for the induction of a causal model of the decision problem, the question is what do people learn in such conditions.

Figure 1a depicts an example of a causal system used in our studies. It comprises three alternative actions (1, 2, 3), three outcome variables (*A*, *B*, *C*), and a final effect variable, which represent the decision maker's payoff. As can be seen from the model, the payoff is not directly influenced by any of the actions but only via the intermediate outcome variables *A*, *B*, and *C*. Of particular importance is that the intermediate variable *A* exerts a causal impact on variable *B*. By repeatedly choosing among the options, people can observe that Action 1 generates variable *A* and, in turn, *B*, while Actions 2 and 3 will generate only a single outcome variable (*B* and *C*, respectively). Thus, the experienced feedback comprises information about the statistical and causal

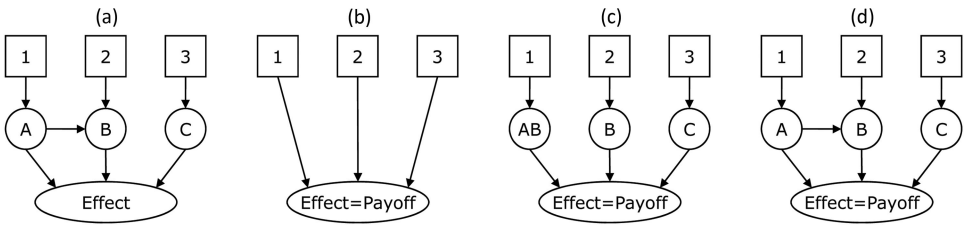


Figure 1. Possible representations of a decision problem. Squares represent possible actions, and circles represent domain variables: (a) true causal system, (b) representation of action-payoff contingencies, (c) representation of action-outcome-payoff contingencies, and (d) causal model representation.

relations of options, outcomes variables, and payoffs. Competing theories of (repeated) decision making and learning differ with respect to what kind of representation are assumed to result from the experienced feedback.

Action-Payoff (AP) Contingencies

Action-payoff representations include only relations between options (i.e., actions) and the received payoffs (see Figure 1b). The characteristic feature of these representations is that the intermediate outcomes resulting from the taken actions are not represented. In the animal learning literature, such approaches are sometimes called *habit learning* (e.g., Niv, Joel, & Dayan, 2006); in machine learning and cognitive neuroscience, they are often referred to as *model-free reinforcement learning* (Dayan & Niv, 2008; Sutton & Barto, 1998). Associative models of instrumental learning are a classic example from the human learning literature (cf. Pearce & Bouton, 2001; Shanks, 2010). An example from the judgment and decision-making literature is Barron and Erev's (2003) *value assessment model*, a reinforcement learning model that estimates the payoff distributions and expected values of the available options from feedback. Similar to habit learning models, the value assessment model only represents options and the expected value of the options.[1]

Action-Outcome-Payoff (AOP) Contingencies

Action-outcome-payoff representations encompass options, intermediate outcome variables, and the payoffs associated with these variables (see Figure 1c). Expected utility theories are the classical example from judgment and decision making; a similar approach from the animal learning literature is *goal-directed learning* (Niv et al., 2006) or *model-based learning* (Dayan & Niv, 2008; Sutton & Barto, 1998). The main difference to AP contingencies is that the outcomes resulting from an action and the value connected with these outcomes are represented separately. Therefore, such models can accommodate revaluations of outcomes by altering the values associated with the outcomes while preserving the action-outcome relations. However, these models have only a limited sensitivity to the causal structure underlying the decision problem (see our experiments for specific predictions).

Causal Model Representations

A mental causal model of a decision problem comprises options, their causal relations to outcome variables, the causal relations among these variables, and their relations to the payoff. Figure 1d depicts such a model. Causal model theories (Sloman, 2005; Waldmann, Hagmayer, & Blaisdell, 2006; Waldmann & Holyoak, 1992) assume that learners acquire these models through individual or social learning. While some theories assume that the structure of the model is derived from cues like temporal order and interventions (Hagmayer et al., 2007; Lagnado et al., 2007), other accounts propose that people primarily use statistical properties of the observed data (Gopnik et al., 2004; Griffiths & Tenenbaum, 2005). Typically, causal model theories assume that the parameters of the model (e.g., the strength of the causal relations and base rates of causes) are estimated on the basis of the observed covariations. Causal Bayes nets theory (Pearl, 2000; Spirtes et al., 1993) provides a formal modeling framework for the induction of causal models.[2]

The advantage of causal knowledge is that it enables decision makers to immediately evaluate the implications of changes in the decision problem, such as the removal of a variable from the underlying causal system or the addition of a novel option. It therefore allows to flexibly respond to changes without requiring further learning input. For instance, knowledge about the causal relations among genes, proteins, and cancer enables personalized medicine.

Hypotheses and Overview of Experiments

Based on a causal model theory of decision making, we propose the following hypotheses. If decision makers have a causal model hypothesis prior to repeated decision making, this knowledge will guide decisions and the interpretation of experienced feedback. If the observed decision outcomes are not consistent with the initial causal hypothesis, the mental causal model will be revised accordingly. When decision makers have no initial causal hypothesis, they will use decision feedback to induce a causal model to the extent possible given the available cues to causal structure. If the decision problem changes, decision makers will rely on their mental causal models to evaluate possible actions and adapt their behavior accordingly.

We conducted three experiments to investigate these hypotheses. In each experiment, participants were asked to maximize a certain payoff through their decisions. They were never asked to engage in causal learning or reasoning. Experiment 1 examined how beliefs about causal structure guide repeated decisions. Therefore, we manipulated decision makers' beliefs about the causal structure underlying the decision problem while keeping the observable consequences constant. In Experiments 2 and 3, decision makers received no information about causal structure prior to decision making. Thus, they had no causal hypothesis to rely on at the start. Participants received decision feedback, enabling them to induce a causal model or to engage in mere instrumental learning, such as learning the expected value of actions (AP) or the relation among actions, outcomes, and payoffs (AOP). All approaches entail optimal (payoff-maximizing) choices as long as the decision

[1] In order to account for certain tendencies in human decision making, the value-assessment model may also include the associative learning of certain rules, such as loss avoidance (see Barron & Erev, 2003, for details).

[2] A formal theory of causal models in learning, reasoning, and decision making is offered by causal Bayes nets theories (for a detailed introduction, see Pearl, 2000; Spirtes et al., 1993; see also Dawid, 2002). Briefly, this account uses directed acyclic graphs to represent the structure of a causal system and connects the structural models with probability distributions over the domain variables. On a causal interpretation of these graphs (as opposed to a purely statistical semantics), the model can be used for reasoning about interventions on the causal system (see Pearl, 2000; Spirtes et al., 1993; see also Meder et al., 2008, 2009). In a decision-making context, it is useful to choose a more general modeling framework in which interventions are explicitly represented as exogenous cause variables (e.g., Dawid, 2002; Spirtes et al., 1993). This approach allows to model different types of interventions, such as actions that do not deterministically fix the value of a variable but only exert a probabilistic influence on the target (for a detailed discussion, see Eaton & Murphy, 2007; see also Meder, Gerstenberg, Hagmayer, & Waldmann, 2010).

situation does not change, but they make diverging predictions when the decision problem is altered.

Experimental Procedure

All experiments consisted of an initial *repeated decision-making phase*, in which participants were asked to maximize the state of a payoff variable by repeatedly choosing between different actions. This repeated decision-making phase was followed by a *test phase* in which the structure of the decision problem was modified. In the test phase, participants were asked to continue maximizing the payoff.

To evaluate whether learners spontaneously engaged in causal induction, we used two types of modifications in the test phase. The first manipulation was to introduce new options whose consequences had not yet been observed (Experiments 1 and 2). The rationale was that only knowledge about causal structure would allow participants to assess whether the novel options were superior or inferior to the existing courses of actions. The second manipulation was to change the structure of the causal system by removing one of the variables (Experiment 3). In this case, the available actions were identical to the initial decision-making phase, but their accurate reevaluation would require causal knowledge.

We used three different dependent variables to tap into decision makers' representations and decision-making processes. First, we examined their choice behavior after modifying the decision problem (i.e., after introducing new options or altering the causal structure of the decision problem). Second, we asked decision makers to provide estimates of the values of the different options, both for the original and modified choice situation. Third, we tried to directly elicit participants' representation of the decision problem, either by a free-elicitation procedure (Experiments 1 and 3) or by a forced choice among potential causal models (Experiment 2). To exclude the possibility that participants' choices in the test phase were contaminated by eliciting a causal model of the decision problem, actual choices were collected first, followed by the expected value estimates and the causal model task.

Model Comparisons

In all experiments, we compared the predictions of a causal model account to two other accounts. The first account, action-payoff (AP) learning, is representative for models from animal learning and decision making, assuming that decision makers merely learn about the payoffs resulting from actions (e.g., Dayan & Niv, 2008; Erev & Barron, 2005; Niv et al., 2006; Sutton & Barto, 1998). Therefore, we used the expected values of each action resulting from the initial repeated decision-making phase to derive model predictions for the test phase.

The second model, action-outcome-payoff (AOP) learning, assumes that learners represent the relations between actions and immediate outcome variables and the value of the outcome variables (cf. Dayan & Niv, 2008). A critical feature of the causal structures used in the present experiments was that some outcome variables were causally related to each other. Since research on associative learning has shown that learners are at least sometimes sensitive for the interrelation of predictive variables (Pearce & Bouton, 2001), we considered two variants of AOP learning. The first model (AOP1) assumes that people only learn about the relations of each action to all outcome variables and the relations of the outcome variables to the payoff. The second model (AOP2) assumes that people may also learn that outcome variables predict each other (e.g., that there is a statistical relation between variables *A* and *B* in Figure 1a). To derive the predictions of the first model (AOP1), we used the contingencies among actions and outcome variables and the expected values of the outcome variables under the assumption that their contributions to the payoff are linear-additive (e.g., Rescorla & Wagner, 1972)—an assumption also supported by empirical research (De Houwer, 2009; Waldmann, 2007). To derive the predictions of the second model (AOP2), we additionally took into account the contingencies between the outcome variables (specific predictions are presented in the description of the experiments below).

Finally, the causal model account assumes that learners use the available cues to infer the causal structure of the domain (Lagnado et al., 2007) and the observed statistical relations to infer the strength of the causal relations (Waldmann & Hagmayer, 2001), including the contribution of the outcome variables to the payoff. Note that this account allows that the strengths of causal relations may be learned through reinforcement learning. The critical claim is that the causal model inferred from cues like temporal order determines *which* relations are learned and represented (i.e., only direct causal relations) and *how* they are represented (i.e., in terms of causes and effects). These representations, in turn, guide assessments of changes in the decision problem. Following causal model theories (Waldmann et al., 2006), we assumed causal influences of different variables to be additive.

Experiment 1

The goal of the first study was to examine how repeated decision making is guided by beliefs about causal structure. We manipulated decision makers' causal hypotheses between subjects by suggesting different causal models prior to the initial repeated decision-making phase. The consequences of the available actions during this phase were identical for all participants. Thus, potential differences between conditions cannot be attributed to differences in feedback.

We hypothesized that participants would use the initially presented causal model hypothesis to evaluate the experienced feedback and revise their mental model if the feedback was inconsistent with the suggested structure. As the initial hypotheses differed between conditions and the observed data only partially confirmed them, participants should end up with different revised hypotheses, despite identical learning input. By contrast, the other models (AP and AOP) suggest that people would use the feedback to acquire knowledge about the contingencies among actions, outcomes, and payoffs. As the observed data were identical, no differences between conditions should be observed.

Method

Participants and design. Participants were 36 undergraduates from the University of Göttingen who were randomly assigned to two causal model conditions (causal chain vs. common cause). They received course credit or were paid 7€.

Materials and procedure. All participants were tested individually on a computer. The experiment consisted of a repeated

decision-making phase and a subsequent test phase. In both phases, participants were requested to maximize the state of a payoff variable by repeatedly choosing between different actions. They were not instructed to learn about causal structure.

We used a biological scenario in which three genes (outcome variables *A*, *B*, *C*) could be activated by three types of injections (actions do*U*, do*V*, do*W*). The genes allegedly influenced the level of a growth hormone, which constituted the payoff variable. Options do*U*, do*V*, and do*W*, as well as genes *A*, *B*, and *C,* were binary variables (present vs. absent and active vs. inactive); the payoff was a continuous variable ranging from 0 to 100 points.

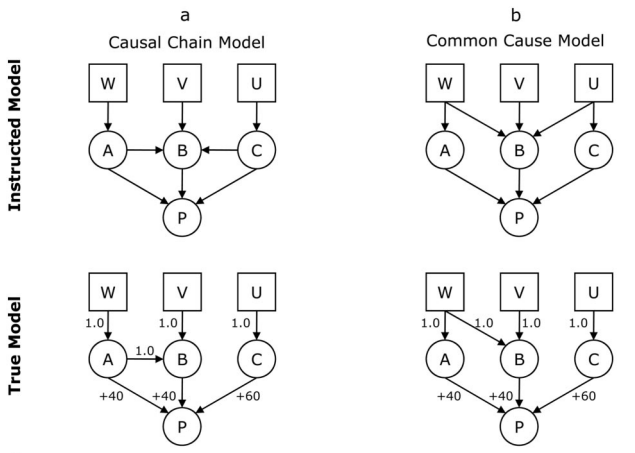
The instructions (see the Appendix) informed participants that scientists had bred mice whose growth hormone genes had been deactivated but which could be re-activated by injecting the mice with different types of "messenger-RNA." No information was given on how much hormone was produced by the individual genes. Participants then received a written description and a graphical representation of either a causal chain or a common cause model hypothesis. Thus, a qualitative hypothesis regarding causal structure was suggested to participants, but they received no information regarding the strengths of the causal relations. Note that participants were not informed about the subsequent test phase.

According to the suggested *causal chain model* (Figure 2, left), actions do*U*, do*V*, and do*W* directly activate only one of the three genes *A*, *B*, or *C*. However, the genes are also causally related to each other: An activation of *A* triggers an activation of *B* (do*W*→*A*→*B*), and *C* activates *B* (do*U*→*C*→*B*). By contrast, in the *common cause model* condition (Figure 2, right), participants were instructed that action do*W* directly affects genes *A* and *B* (*A*←do*W*→*B*) and that do*U* directly affects *B* and *C* (*B*←do*U*→*C*).

Both causal model hypotheses were only partially correct. The true underlying causal models are depicted in the middle row of Figure 2. Contrary to the suggested models, do*U* neither directly nor indirectly affected gene *B*, but only activated *C*. The table in Figure 2 specifies the feedback that decision makers experienced, which was identical in both conditions. Choosing do*W* resulted in an activation of both *A* and *B* and a payoff of +80 (i.e., the hormone level increased by 80 points). Actions do*V* and do*U* activated *B* and *C*, respectively, yielding payoffs of +40 and +60. Choosing not to take any action ("no do") left the genes inactive and generated no payoff. For each condition, two counterbalancing conditions were used in which the causal roles of do*U* and do*W* were switched.

In the instruction to the initial repeated decision-making (RDM) phase, participants were told the following: "Your task is to activate the genes by injecting individual mice with *U*-, *V*-, or *W*-RNA in order to maximize the animals' hormone level" (see the Appendix). Each of the 30 trials referred to a new mouse whose genes were inactive prior to intervention. After each decision, feedback was provided regarding which genes had been activated and the resulting level of growth hormone (Figure 3). Thus, participants received information on the state of the outcome variables and the final payoff. No further cues to causal structure (e.g., temporal order of events) were provided.

In the following test phase, which comprised 10 trials, a new set of options was introduced (see the Appendix for instructions). Participants were informed that they now had only "*A*-RNA" (= do*A*) and "*C*-RNA" (= do*C*) available, which deterministically activate genes *A* and *C*, respectively. Then they were given the following instruction: "Your task is to maximize the level of the growth hormone in the individual animals by applying these messenger-RNA." No feedback was provided in this phase. Thus, the consequences of the new interventions could not be observed.



Feedback Structure

Repeated Decision-Making Phase (identical across models)			Test Phase				
				Causal Chain		Common Cause	
Choice	Outcome	Points	Choice	Outcome	Points	Outcome	Points
do W	A & B	+80	do A	A & B	+80	A	+40
do V	B	+40	do C	C	+60	C	+60
do U	C	+60	–	–	–	–	–
no do	–	0	no do	–	0	–	0

Figure 2. Causal models and feedback structure in Experiment 1. Squares indicate possible actions, and circles indicate outcome variables. Numbers in graphs represent probabilities and generated payoffs, respectively. In the test phase, no feedback regarding outcome variables and payoff was given.

Following the test phase, participants were requested to estimate the expected payoffs of all actions (do*U*, do*V*, do*W*, do*A*, do*C*). Finally, participants were provided with a graph depicting only the variables (i.e., a graph similar to the one in Figure 2, middle row, without arrows) and were asked to insert all causal relations they assumed to hold between actions, intermediate outcome variables, and the payoff variable.

Model predictions. For the initial repeated decision-making phase, all accounts predict participants learning that option do*W* yields the highest payoff, followed by do*U* and do*V* (see Figure 2). Action-payoff (AP) learning suggests that decision makers merely learn the expected value of each action. When participants engaged in action-outcome-payoff (AOP) learning instead, they should also learn that do*U* is not related to *B*, and they should learn that *B* is present whenever do*W* is chosen (AOP1 and AOP2) or variable *A* is present (AOP2). A causal model account suggests that participants revise the initially suggested structure and remove the causal link between do*U* and *B* (common cause condition) and *C* and *B* (causal chain condition). Based on the assumption that the influence of the genes is additive, they should also learn how much hormone each of the genes produces (AOP1 and AOP2 and causal model theory).

For the test phase, causal model theory predicts that participants would rely on their revised causal model to infer the causally expected values of the new interventions (see the table in Figure 2). According

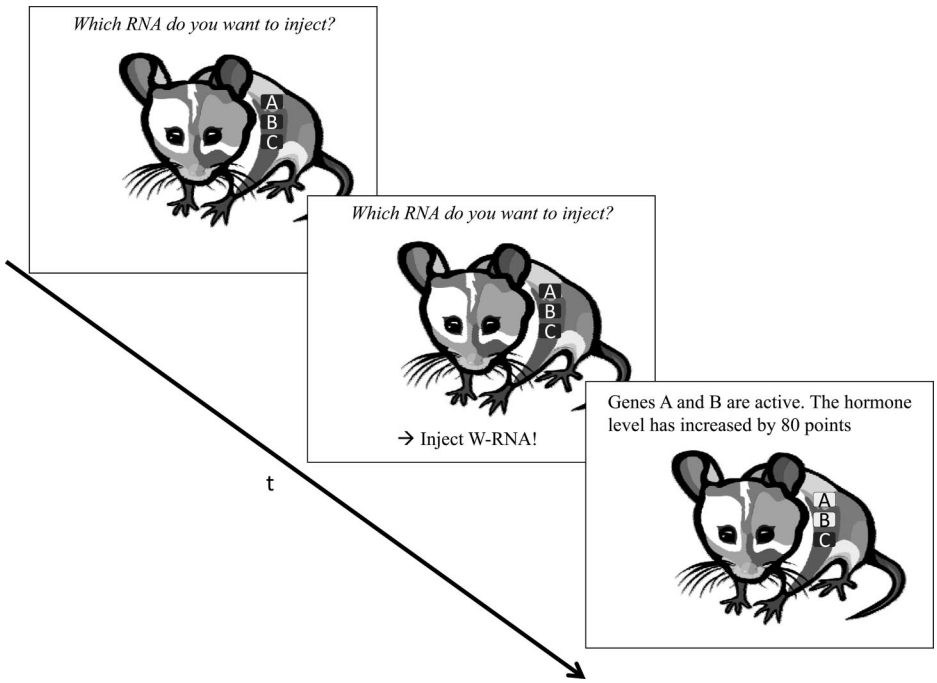


Figure 3. Stimuli and schematic outline of a trial during the initial repeated decision-making phase in Experiment 1.

to the revised causal chain model, do*A* would activate *A* and *B*, generating a total payoff of +80, whereas do*C* would activate *C*, yielding a payoff of +60. Hence, participants should prefer do*A*. The revised common cause model implies that do*A* would yield a payoff of +40 because variables *A* and *B* are not causally related. Accordingly, decision makers should opt for do*C*, which yields a payoff of +60. Thus, participants should exhibit diverging preferences in the test phase.

An AP learning model does not allow inferring the payoff of the novel option, as the acquired representations are restricted to the values of the experienced actions. Both AOP learning models entail that participants should be able to make an inference regarding the payoff of the novel options. According to the first model (AOP1), which only represents the values of the individual outcome variables, participants should infer that the expected payoff of an activation of *A* would be +40 and of *C* +60. Thus, they should prefer intervention do*C* over do*A*, regardless of condition. According to the second model (AOP2), the deterministic relation between variables *A* and *B* [$P(B|A) = 1.0$] is learned and taken into account. Hence, do*A* would have a higher expected payoff than do*C* [EV(do*A*) = +80, EV(do*C*) = +60]. Therefore, a preference for do*A* should result in both conditions. Note that although both AOP accounts make different predictions about what is learned and represented, they do not make differential predictions for the two causal model conditions.

Finally, when queried about the underlying causal model, we expected participants to describe the revised but not the initially instructed model (i.e., no causal link $C \rightarrow B$ in the chain model and no link do$U \rightarrow B$ in the common cause model). AP learning makes no specific predictions (or trivially only assumes links between actions and payoffs). AOP learning entails that participants should not include any relation among do*U* and *B*, but it makes no other particular predictions, as these models do not specify how an instructed causal model is modified by the observed statistical relations.[3]

Results and Discussion

Table 1 depicts participants' choices for the initial repeated decision-making phase and the subsequent test phase. In the first phase, participants exhibited a clear preference for do*W*, regardless of condition. Statistical analyses (between-participants *t* tests for do*U*, do*V*, do*W*, and "no do") revealed no reliable differences between conditions (all $ps > .19$).[4] By contrast, a clear difference between conditions was obtained for the test phase. In line with a causal model account, participants chose do*A* significantly more often in the causal chain than in the common cause condition, $t(34) = 3.16$, $p < .01$.

Participants' estimates of the expected values were consistent with their choices (see Table 1). Again, no differences between conditions resulted for the first phase (all $ps > .33$), but only for the test phase. In the causal chain condition, participants judged do*A* to have a higher expected value than do*C*, whereas they rated

[3] An alternative would be to assume that a model is derived from the observed contingencies. Several causal models are consistent with the data: Variable *A* could be a cause of *B*, *A* and *B* could be generated by a common cause, or there might be both a common cause and a direct causal relation. Thus, a data-driven account does not allow deriving specific predictions.

[4] As choices of the different options were logically dependent on each other, we compared the number of choices of each option separately between conditions.

Table 1
Mean Number of Choices (±SEM) and Expected Value Estimates (±SEM) in Experiment 1

		Repeated decision-making phase (30 trials)				Test phase (10 trials)		
Dependent variable	Condition	do*W*	do*V*	do*U*	no do	do*A*	do*C*	no do
Choices	Chain model	20.6 (1.65)	3.2 (0.62)	4.5 (0.72)	1.7 (0.43)	7.8 (0.65)	2.1 (0.63)	0.1 (0.06)
	Common cause model	18.9 (1.66)	4.1 (0.69)	5.5 (0.94)	1.3 (0.28)	4.3 (0.88)	5.4 (0.90)	0.1 (0.06)
Expected values	Chain model	78.9 (1.11)	43.3 (1.81)	55.6 (2.58)		70.0 (3.70)	54.4 (3.81)	
	Common cause model	80.0 (0.00)	43.3 (2.43)	54.4 (2.17)		42.4 (2.59)	46.3 (4.02)	

Note. Actions "do (●)" refer to the choices people could take (see Figure 2).

do*C* slightly higher than do*A* in the common cause condition. An analysis of variance (ANOVA) with actions do*A* versus do*C* as a within-participants factor, and condition (causal chain vs. common cause) as a between-participants factor, resulted in the expected interaction effect, $F(1, 34) = 11.5$, $p < .01$, $MSE = 148.1$. Follow-up analyses confirmed that participants gave higher ratings for do*A* in the causal chain than in the common cause condition, $t(34) = 5.96$, $p < .001$, whereas there was no significant difference with respect to do*C*, $t(34) = 1.47$, $p = .15$. Decision makers in the chain condition seemed to have realized that do*A* would activate *B*, thereby entailing a higher expected payoff.

Finally, we analyzed the causal models drawn by participants. In the common cause condition, all participants judged do*W* to be the common cause of *A* and *B*, 94% judged do*V* to be the cause of *B*, and 67% judged do*U* to be the cause of variable *C* only. In the chain condition, 71% assumed do*W* to be the indirect cause of *B* (do*W*→*A*→*B*), 24% regarded do*W* to be the common cause of *A* and *B*, 88% recognized do*V* as being the cause of *B*, and, finally, 82% judged do*U* to be neither a direct nor indirect cause of *B*. These results show that most participants revised the initially suggested causal model in accordance with the experienced feedback.

Overall, the findings indicate that most participants represented the decision task in terms of mental causal models and used the experienced feedback to estimate parameter values and to revise their hypotheses. The causal hypotheses, in turn, affected participants' choices in the test phase. Data-driven models like AP and AOP cannot account for the obtained differences because neither the learning input nor participants' choices during the initial decision-making phase differed between conditions.

Experiment 2

The first goal of Experiment 2 was to examine whether decision makers would spontaneously induce a causal model representation during repeated decision making. This would demonstrate that people not only use existing causal beliefs (like in Experiment 1) but also show a natural tendency to infer the causal structure underlying a decision problem. Therefore, no causal hypotheses were suggested. Instead, learners were provided with options that would enable them to infer the underlying causal structure from the experienced feedback. As cues to causal structure were available, we expected participants to induce a causal model representation and to use it to make decisions in the test phase.

The second goal was to show that causal induction during repeated decision making is not limited to simple tasks with deterministic causal links. We increased task complexity by using a probabilistic causal system and actions that only probabilistically generated their effects.

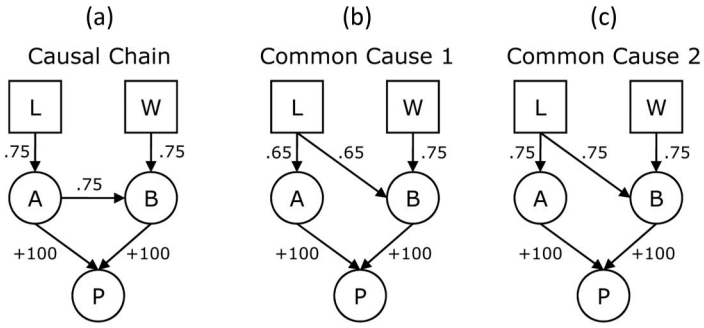
Method

Participants and design. Sixty undergraduate students from the University of Göttingen participated for course credit or were paid 7€. They were randomly assigned to one of three causal model conditions (causal chain, common cause 1 [CC1], common cause 2 [CC2]).

Materials and procedure. Participants were tested individually on computers in small groups of up to six people. Figure 4 shows the causal chain and common cause structures underlying the decision problem in the experimental conditions and the respective feedbacks. In the causal chain condition, option do*L* influenced variable *B* only by way of *A* (do*L*→*A*→*B*), whereas in the two common cause conditions, do*L* independently affected *A* and *B* (*A*←do*L*→*B*). We examined two different common cause conditions. In one condition (CC1; see Figure 4b), the available options (do*L*, do*W*) had the same expected values as in the causal chain condition. This manipulation, however, required different probabilistic relations than in the chain condition [e.g., $P(A\&B \mid \text{do}L)_{\text{Chain}} > P(A\&B \mid \text{do}L)_{\text{CC1}}$; see Figure 4b]. We therefore designed a second common cause condition (CC2; see Figure 4c) in which $P(A\&B|\text{do}L)$ and $P(A|\text{do}L)$ were identical to the chain condition. As a consequence, the expected value of do*L* was higher than in the chain condition, but the rank order of the actions' expected values was identical in all conditions [i.e., EV(do*L*) > EV(do*W*); see Figure 4].

We used a biological scenario according to which certain bacteria produce a vaccine against diseases (see the Appendix for instructions). All participants received the same instructions informing them that the production of the vaccine is regulated by two genes, *A* and *B*, that are inactive by default. Their task was to produce as much vaccine as possible by activating the genes with "trigger substances" *L* and *W*. Participants were not informed of how the trigger substances related to the activation of the genes, but it was pointed out that the two genes may be causally interrelated. The instructions also indicated that new trigger substances might become available after the initial repeated decision-making phase. Figure 5 outlines stimuli and procedure. Recall that, in contrast to Experiment 1, no specific causal model was suggested.

The initial repeated decision-making phase consisted of 100 trials. Each decision referred to bacteria whose genes were inactive prior to applying a trigger substance. Participants could choose between three



		Chain			Common Cause 1			Common Cause 2		
Choice	Events	Prob	Points	EV	Prob	Points	EV	Prob	Points	EV
do L	A & B	0.56	200	131	0.42	200	130	0.56	200	152
	A	0.19	100		0.23	100		0.19	100	
	B	0.00	100		0.23	100		0.19	100	
	—	0.25	0		0.12	0		0.06	0	
do W	B	0.75	100	75	0.75	100	75	0.75	100	75
	—	0.25	0		0.25	0		0.25	0	
no do	—	1.00	0	0	1.00	0	0	1.00	0	0
do A	A & B	0.75	200	175	0.00	200	100	0.00	200	100
	A	0.25	100		1.00	100		1.00	100	

Figure 4. Causal models and feedback structure of Experiment 2. Squares indicate possible actions, and circles indicate outcome variables. Numbers in graphs represent probabilities and generated payoffs, respectively. Option do*A* was only available in the test phase. This action has the highest causally expected value in the causal chain condition but not in the two common cause conditions. In the test phase, no feedback regarding outcome variables and payoff was given. Prob = probability; EV = expected value.

options (do*L*, do*W*, no do). After making a decision, participants first observed which genes became active and then received information on the total amount of vaccine produced (payoff). If on a given trial only one gene became active, the activation was illustrated by slowly changing the color of the gene. If on a particular trial both *A* and *B* were activated, the temporal order of the outcome variables conformed to the underlying causal structure. In the chain condition, participants first observed that gene *A* became active and then, with a delay of 1 s, that gene *B* also became active. In the common cause condition, both genes became active simultaneously. After the animation, information regarding the payoff was given along with a statement on which genes had been activated (see Figure 5).

The subsequent test phase consisted of 10 additional decision trials. In this phase, a novel option was introduced (see the Appendix for instructions). Decision makers were informed that a new trigger substance had been developed, which reliably activated gene *A* (i.e., with a probability of 1). Then, participants could choose between the two known options (do*L*, do*W*) and the new option do*A* to maximize the payoff variable. No feedback was provided in this phase (i.e., participants did not observe any activation of the genes, and they did not receive feedback regarding the produced amount of vaccine).

Upon completion of the test phase, participants were asked to estimate the expected values of all options. Then, they were queried about their assumptions about the causal structure underlying the decision task. We used a forced-choice task in which participants were presented with graphs of a causal chain and a common cause model (similar to the ones shown in Figure 4, but without the numbers), and they had to indicate which of the two models would correctly describe the causal relations between actions, intermediate variables, and payoff.

Model predictions. For the initial repeated decision-making phase, all models predict participants learning that option do*L* has a higher expected value than do*W* and that they adjust their choices accordingly. Based on a causal model account, we also expected them to infer the underlying causal model from temporal information (e.g., sequential vs. simultaneous activation of genes *A* and *B*) and statistical properties of the learning data.

For the test phase, the causal model account predicts that participants would use their causal model to infer the expected payoff of the novel option do*A*. In the chain condition, the new option do*A* should be preferred as it has a higher expected value than do*L* (see Figure 4). By contrast, in the common cause conditions, intervening on *A* would not affect *B*; therefore, do*L* should be preferred over do*A*.

An AP learning model assuming that only contingencies among options and payoffs are represented does not yield any predictions with respect to the novel option, because the intermediate outcome variables are not separately represented. However, an AOP model would be able to make predictions. The first model (AOP1), which does not represent relations among the outcome variables, entails that the expected payoff of do*A* would be 100, as this is the value

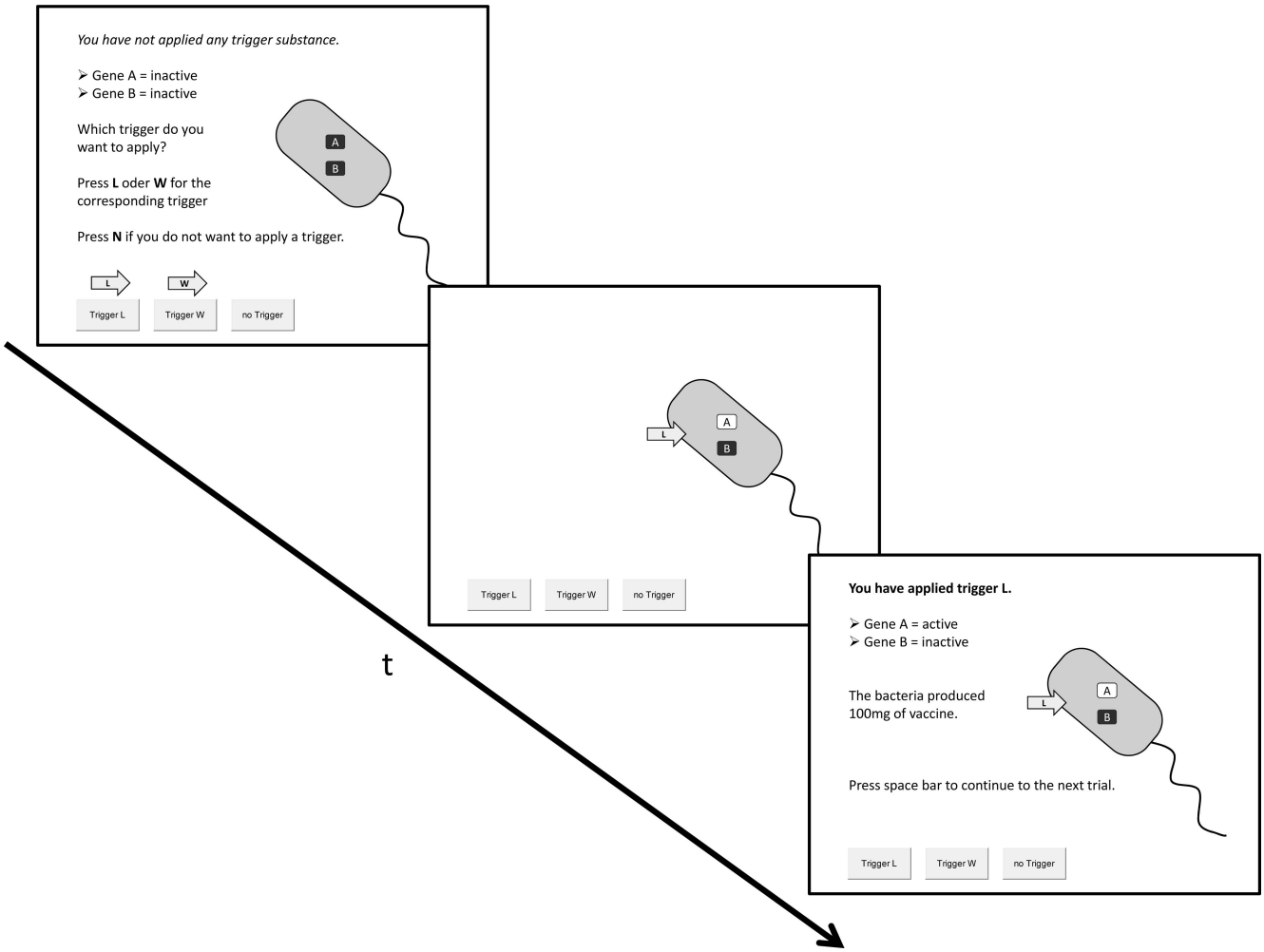


Figure 5. Stimuli and schematic outline of a trial in the repeated decision-making phase in Experiment 2. After making a decision, an animation was shown, illustrating which genes had been activated by the intervention. Finally, information on the generated payoff (amount of produced vaccine) was provided, along with information regarding which genes had been activated.

associated with outcome variable *A*. Therefore, option do*L* should be preferred over do*A*, as the expected value of do*L* is higher in all conditions (see Figure 4). The second model (AOP2) is sensitive to the statistical relation between variables *A* and *B*. The observed probabilities $P(B|A)_{\text{Chain}} = 0.75$, $P(B|A)_{\text{CC1}} = 0.65$, and $P(B|A)_{\text{CC2}} = 0.75$ entail the following expected values: $\text{EV}(\text{do}A)_{\text{Chain}} = 175 > \text{EV}(\text{do}L)_{\text{Chain}} = 131$, $\text{EV}(\text{do}A)_{\text{CC1}} = 165 > \text{EV}(\text{do}L)_{\text{CC1}} = 130$, and $\text{EV}(\text{do}A)_{\text{CC2}} = 175 > \text{EV}(\text{do}L)_{\text{CC2}} = 152$. Thus, a preference for do*A* should result in all conditions.

In summary, a causal model account predicts different choices and estimates of expected values, depending on the underlying causal model, whereas the other accounts predict no differences between conditions.

Results and Discussion

Tables 2 and 3 show the results of Experiment 2. In the initial decision-making phase, decision makers had a clear preference for option do*L*, regardless of condition (see Table 2). Two ANOVAs with the three conditions as a between-participants variable and choices of do*W* and do*L*, respectively, as dependent variables revealed no statistical differences between conditions (do*W* $F < 1$ and do*L* $F < 1$). By contrast, participants' choices during the test phase differed (see Table 3). Decision makers in the chain condition exhibited a strong preference for the new option do*A*, indicating that they realized that the novel option had a higher expected value. In the two common cause conditions, participants preferentially chose do*L*. Significantly more do*A* choices were obtained in the chain condition than in conditions CC1, $t(38) = 3.50$, $p < .001$, and CC2, $t(38) = 5.23$, $p < .001$. The two common cause conditions did not differ from each other, $t(38) = 0.93$, $p = .35$.

Estimated payoffs for do*L* and do*W* closely resembled the actual values, though participants in the CC2 condition underestimated the expected value of do*W*, and participants in the chain condition slightly underestimated the value of do*A*. An ANOVA with option

Table 2
Mean Number of Choices (±SEM) and Received Payoffs (±SEM) During the Initial Repeated Decision-Making Phase in Experiment 2

	Choices		
Condition	do*L*	do*W*	no do
Causal chain	74.8 (3.6)	21.8 (3.1)	3.5 (0.8)
Common cause 1	77.7 (2.6)	20.3 (2.4)	2.0 (0.4)
Common cause 2	73.5 (4.2)	23.7 (3.7)	2.9 (0.9)

Note. Actions "do*L*" and "do*W*" refer to the choices people could take (see Figure 4); "no do" denotes the decision not to take any action.

(do*L*, do*W*, do*A*) as a within-participant factor, and causal model (causal chain vs. common cause) as a between-participants factor, yielded the expected interaction effect, $F(1, 114) = 3.93$, $p < .01$, $MSE = 945.3$. The crucial analyses concern option do*A*, whose actual consequences people never observed. Estimates in the chain condition were significantly higher than in condition CC1, $t(38) = 2.4$, $p = .02$, and condition CC2, $t(38) = 2.3$, $p = .03$. The two common cause conditions did not differ from each other, $t(38) = 1.3$, $p = .19$.

To assess decision makers' beliefs about causal structure, they were asked to choose between graphs of a causal chain and a common cause model. In all three conditions, the majority of participants chose the correct model: 85% in the chain condition, and 70% and 75% in common cause conditions 1 and 2, respectively.

Overall, there was a strong concordance between the preferences that decision makers revealed through their choices, their expected value estimates, and their subjective beliefs about the causal structure of the decision problem: Participants assuming a chain model (regardless of true underlying model) chose do*A* more often than participants assuming a common cause model ($M_{\mathrm{do}A|\mathrm{chain}} = 6.93$, $SEM = 0.57$; $M_{\mathrm{do}A|\mathrm{CC}} = 3.41$, $SEM = 0.56$), $t(58) = 4.37$, $p < .01$, and gave higher estimates ($M_{\mathrm{do}A|\mathrm{chain}} = 138.6$, $SEM = 6.87$; $M_{\mathrm{do}A|\mathrm{CC}} = 107.2$, $SEM = 5.78$), $t(58) = 3.52$, $p < .01$.

Taken together, participants seemed to have induced a causal model based on the observed feedback and later used these models to infer the value of the new option. This resulted in differential preferences for the novel option. The chain and the first common cause condition provided identical feedback with respect to the payoffs of both conditions. Therefore, a learning model encoding only contingencies among options and payoffs would yield identical representations and cannot explain the diverging preferences. As outlined above, the findings also cannot be accounted for by models learning about the contingencies among options, outcome variables, and payoffs, which would also predict no differences between conditions.

Experiment 3

The goal of Experiment 3 was to extend the findings of Experiment 2 by using a different manipulation in the test phase. Instead of adding a novel option, the causal structure was modified by removing one of the variables from the causal system. Causal relations were again probabilistic, and interventions were imperfect.

Method

Participants and design. Forty-eight undergraduate students from the University of Göttingen participated for course credit or were paid 7€. The factor causal model (causal chain vs. common cause) was varied between conditions.

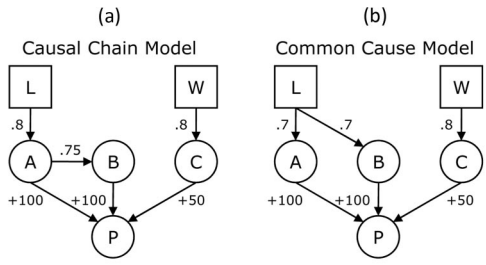
Materials and procedure. Participants were tested individually on computers. Figure 6 shows the two experimental conditions, causal chain and common cause, and the associated feedback structures. We used the same materials and procedure as in Experiment 2 (i.e., the bacteria stimuli and the task to maximize the amount of produced vaccine; see Figure 5 and the Appendix for instructions). As in the previous study, participants were not provided with a causal model hypothesis prior to the decision-making phase. The only difference to Experiment 2 was that there were three genes (*A*, *B*, *C*) instead of two. Figure 6 shows the two conditions. In the causal chain condition, option do*L* influenced *B* only by way of *A* (do$L \rightarrow A \rightarrow B$). In the common cause condition, do*L* was directly related to both *A* and *B* ($A \leftarrow \mathrm{do}L \rightarrow B$). Thus, whereas in the chain condition the presence of *A* was a necessary event for the occurrence of *B* [i.e., $P(B|\neg A) = 0$], this was not the case in the common cause condition [i.e., $P(B|\neg A) > 0$]. Despite this difference, the expected values of the available options were identical across conditions: EV(do*L*) = 140, and EV(do*W*) = 40.

The initial repeated decision-making phase consisted of 100 trials, with the temporal order of events conforming to the under-

Table 3
Mean Number of Choices (±SEM) and Mean Expected Values Estimates (±SEM) in the Test Phase of Experiment 2

Dependent variable	Condition	do*L*	do*W*	do*A*
Choices	Causal chain	2.2 (0.4)	0.1 (0.6)	7.8 (0.7)
	Common cause 1	5.4 (0.8)	0.5 (0.8)	4.2 (0.8)
	Common cause 2	6.2 (0.9)	0.5 (0.7)	3.3 (0.6)
Expected value estimates	Causal chain	134.8 (8.0)	75.0 (4.0)	140.0 (8.8)
	Common cause 1	141.3 (6.4)	68.8 (4.8)	112.0 (7.8)
	Common cause 2	157.0 (10.1)	59.6 (7.3)	113.5 (7.5)

Note. Actions "do*L*," "do*W*," and "do*A*" refer to the choices people could take (see Figure 4). Option "do*A*" was only available in the test phase.



Repeated Decision Making Phase

		Causal Chain			Common Cause		
Choice	Events	Prob	Points	EV	Prob	Points	EV
do L	A & B	0.60	200	140	0.49	200	140
	A	0.20	100		0.21	100	
	B	0.00	100		0.21	100	
	—	0.20	0		0.09	0	
do W	C	0.80	50	40	0.80	50	40
	—	0.20	0		0.20	0	
no do	—	1.00	0	0	1.00	0	0

Test Phase

		Causal Chain			Common Cause		
Choice	Events	Prob	Points	EV	Prob	Points	EV
do L	A & B	0.00	200	0	0.00	200	70
	A	0.00	100		0.00	100	
	B	0.00	100		0.70	100	
	—	1.00	0		0.30	0	
do W	C	0.80	50	40	0.80	50	40
	—	0.20	0		0.20	0	
no do	—	1.00	0	0	1.00	0	0

Figure 6. Causal structures and feedback in Experiment 3. Squares indicate possible actions, and circles indicate outcome variables. Numbers in graphs represent probabilities and generated payoffs, respectively. In the test phase, variable *A* is removed from the causal system. In the causal chain condition, the causally expected value of action do*L* decreases to zero, making do*W* the better option. In the common cause condition, the causally expected value of action do*L* also decreases, but it remains the option with the highest expected value. No feedback about outcomes and payoff was provided in the test phase. Prob = probability; EV = expected value.

lying causal model (i.e., genes *A* and *B* became either sequentially or simultaneously active). The test phase comprised 10 additional decisions. The instructions to the test phase informed decision makers that they would be presented with bacteria that did not possess gene *A*. Accordingly, the graphical representation of the bacteria in the test phase was lacking one of the genes. No feedback with respect to the activation of the gene or the amount of vaccine was provided in the test phase. Then, participants were requested to estimate the expected payoffs of all options in both decision-making phases (i.e., with and without variable *A*). Finally, participants were presented with an empty graph depicting the variables and were asked to indicate the hypothesized causal relations by drawing arrows between options, outcome variables, and payoff.

Model predictions. For the initial repeated decision-making phase, all models again predict participants learning about the payoffs of the available options and prefering do*L* over do*W*. The causal model account also predicts that they would use the available cues (i.e., the observable statistical and temporal relations) to infer the underlying causal model.

Different predictions are entailed for the test phase. A causal model account implies that the removal of variable *A* should result in different choices in the two conditions. In the chain condition, participants should have induced a model according to which do*L* affects *B* only by way of *A*. Therefore, a removal of *A* renders do*L* ineffective, making do*W* the better option. In the common cause condition, the removal of *A* also decreases the expected value of do*L*. However, because do*L* directly influences *B*, do*L* remains the better option (see Figure 6).

An AP model would build up the same representation in both conditions and would not be able to differentially adapt to the removal of *A*. AOP learning models, by contrast, would be sensitive to the modification. According to both models AOP1 and AOP2, participants should infer from the repeated decision-making phase that EV(*B*) = 100. As the contingencies between do*L* and *B* were highly similar [$P(B|\text{do}L)_{\text{Chain}} = .6$, and $P(B|\text{do}L)_{\text{CC}} = .7$], the removal of *A* would have the same qualitative implications for the expected values of do*L*: EV(do*L*|*A* removed)$_{\text{Chain}} = 60$, and EV(do*L*|*A* removed)$_{\text{CC}} = 70$. As these expected values are higher than the expected value of do*W* (40), a preference for do*L* is predicted in both conditions. Note that there is no difference between AOP1 and AOP2, as *B* is the only outcome variable after the removal of *A*.

Results and Discussion

The upper left part of Table 4 depicts participants' choices for the repeated decision-making phase. Decision makers exhibited a clear preference for option do*L* in this phase. Separate ANOVAs for options do*L* and do*W* revealed no differences between conditions (both ps $> .20$). A clear difference between conditions was observed in the test phase after the removal of *A* (see Table 4, upper right part). Participants chose do*W* more often in the chain condition than in the common cause condition, $t(46) = 2.83$, $p < .01$. Conversely, the mean of do*L* choices was higher in the common cause condition than in the causal chain condition, $t(46) = 2.72$, $p < .01$. The fact that participants in the common cause condition exhibited only a slight preference for do*L* over do*W* might be due to a trade-off between mean and variance (i.e., opting for do*L* gives 100 points with $p = .7$, whereas do*W* results in 50 points with $p = .8$).

Participants' expected value estimates were consistent with their choices (see Table 4, middle part). An ANOVA for the estimates in the test phase with option (do*L* vs. do*W*) as a within-participants factor, and causal model (causal chain vs. common cause) as a between-participants factor, resulted in the expected interaction effect, $F(1, 46) = 5.69$, $p < .05$, $MSE = 768.3$. In both conditions, participants realized that the removal of variable *A* would decrease the expected value of option do*L*. Crucially, they were sensitive to the fact that the amount of decrease depended on the underlying causal structure. An ANOVA for ratings of do*L* with phase (before vs. after removal of *A*) as a within-participants factor, and condition (causal chain vs. common cause) as a between-participants factor, resulted in a significant main effect of removal, $F(1, 46) = 198.6$, $p < .01$, and a significant interaction, $F(1, 46) = 10.6$, $p < .01$, $MSE = 996.5$. Participants gave lower estimates for do*L* in the causal chain than in the common cause condition, $t(46) = 2.69$, $p = .01$. There were no differences between conditions with respect to do*W* ($F < 1$).

Table 4
Mean Number of Choices (±SEM), Expected Value Estimates (±SEM), and Causal Model Drawings in Experiment 3

Dependent variable	Condition	Repeated decision-making phase (100 trials)			Test phase (10 trials)		
		do*L*	do*W*	no do	do*L*	do*W*	no do
Choices	Causal chain	73.6 (3.4)	21.7 (2.8)	4.7 (1.2)	2.8 (0.7)	7.2 (0.7)	0.07 (0.06)
	Common cause	79.3 (3.0)	17.9 (2.7)	2.8 (0.9)	5.5 (0.7)	4.5 (0.7)	0.0 (0.0)
Expected values	Causal chain	149.4 (5.4)	45.6 (3.8)		37.6 (7.4)	40.6 (4.5)	
	Common cause	137.1 (5.9)	44.1 (2.5)		67.3 (8.2)	43.3 (2.8)	

		Causal chain	Common cause	Common cause + chain	Other model
Causal model task	Causal chain	10	6	4	4
	Common cause	4	6	9	5

Note. Actions "do*L*" and "do*W*" refer to the choices people could take (see Figure 6); "no do" denotes the decision to not take any action.

Finally, we analyzed the causal models drawn by the participants (see Table 4, lower part). Unexpectedly, a number of participants indicated that do*L* is a common cause (i.e., $A \leftarrow \text{do}L \rightarrow B$) *and* that there is a direct relation $A \rightarrow B$ (i.e., causal overdetermination). Although not very parsimonious, this hypothesis is actually not inconsistent with the observations in the common cause condition. The exactly correct model was drawn by 33% of the participants. Together with the participants assuming causal overdetermination in the common cause condition, 52% of the decision makers induced a causal model that was consistent with the obtained feedback. The rest, however, drew a model that was inconsistent with the observations made. This finding indicates that not all participants inferred the correct causal model underlying the decision problem (see the General Discussion). However, we also suspect that the free elicitation procedure was more difficult for participants than the forced-choice task used in Experiment 2.

Follow-up analyses revealed a substantial convergence between participants' choices, expected value estimates, and causal model hypotheses. Participants assuming a model with a common cause (regardless of the true underlying model) chose do*L* more often than participants assuming a causal chain model ($M_{\text{do}L|\text{CC}} = 5.48$, $SEM = 0.66$; $M_{\text{do}L|\text{Chain}} = 1.43$, $SEM = 0.64$), $t(46) = 4.25$, $p < .01$, and they assumed do*L* to be more effective despite the removal of *A* ($M_{\text{do}L|\text{CC}} = 68.2$, $SEM = 4.02$; $M_{\text{do}L|\text{Chain}} = 9.32$, $SEM = 6.15$), $t(46) = 6.66$, $p < .01$.

Overall, these results demonstrate that participants were sensitive to the causal structure of the decision problem. Many decision makers seemed to have spontaneously induced a causal model representation, which allowed them to adapt their choice behavior when variable *A* was removed from the system. Like the results of Experiment 2, the differential choices and judgments cannot be explained by accounts assuming that people merely learn and represent contingencies observed in the data.

General Discussion

In the present experiments, we studied the role of causal inference in repeated decision making. The results provide strong evidence that decision makers are sensitive to the causal texture of decision problems, induce causal models, and use this knowledge to adapt their choice behavior when being confronted with changes in the decision context. They did so even though they were never instructed to engage in causal reasoning but only to maximize a certain payoff variable.

Experiment 1 demonstrated how the consequences of decisions are evaluated relative to existing beliefs about causal structure. After making a number of decisions whose feedback could be used to estimate the causal models' parameters and revise structure hypotheses, participants were confronted with a new set of options. Despite identical learning experience, they chose different options, estimated different expected values, and made different causal model assumptions. Experiments 2 and 3 showed that many participants spontaneously induced a causal model of the decision problem, although their instructed goal was to maximize the state of a certain payoff variable. They acquired causal knowledge and adapted their choice behavior when the decision task changed, even though interventions and causal relations were probabilistic.

However, the results also show that not all decision makers spontaneously induce causal model representations. For example, the causal models drawn by participants in Experiment 3 indicate that only about half of them acquired a causal model that was a veridical representation of the underlying causal structure. Some seem to have acquired a causal model representation that was wrong—but that still guided their decisions. For about 40% of participants, their assumed causal model and their choices as well as their estimates about expected values were not coherent with each other. Three of these people neither changed their choices in the test phase nor did they revise their expected values, indicating that they engaged in mere action-payoff learning. The remaining participants reacted to the removal of a variable from the system by shifting their behavior and reducing their estimated payoffs for the affected action. There was no apparent difference between conditions for these people, which indicates that they probably engaged in some kind of action-outcome-payoff learning.

In sum, the results show that a majority of participants engaged in causal induction during repeated decision making, but a considerable number of participants acquired other types of representations. The reluctance of these participants to induce a causal representation might be rooted in explicitly instructing participants to maximize the state of the payoff variable. It simply may not have occurred to these participants that acquiring a causal repre-

sentation may turn out to be useful later on. In addition, attentional factors may have contributed to their reluctance. Being focused on maximizing the payoff, they may not have paid attention to the cues indicating the underlying causal structure.[5]

Theoretical Implications

Our findings challenge accounts that neglect the role of causal inference in decision making. Models that only encode the expected values of options cannot account for the results, since the intermediate outcome variables that make the crucial difference between the conditions are not represented. Accounts that represent the outcome variables and their associations to options, on the one hand, and payoffs, on the other hand, are sensitive to changes in causal structures and predict that participants will react to them. However, as detailed in the specific predictions for each experiment, such accounts cannot explain the diverging choices and estimates participants made.

A propositional account of associative learning (De Houwer, 2009; Mitchell, De Houwer, & Lovibond, 2009), which assumes that the formation of associative links is mediated through propositions, may be extended to explain these findings. According to this approach, associative learning is not automatic, but effortful, and requires conscious awareness and cognitive resources. In contrast to classical associative learning models, such an account allows learning to be influenced by verbal instructions and abstract knowledge. Applied to the current experiments, such an approach may assume that people use the available cues to causal structure (e.g., the instructed causal model in Experiment 1 or the temporal and statistical information in Experiments 2 and 3) to induce propositions referring to the causal relations underlying the decision problem. Such an account would be very similar to a causal model account, as it also assumes that the formation of links between events results from controlled reasoning processes.

It is also important to note that reinforcement learning models allow to describe repeated decision making in many areas (see Newell, Lagnado, & Shanks, 2007, for an overview). For example, choices between gambles with different payoff distributions can be captured by Barron and Erev's (2003) value assessment model. Decisions based on multiple cues can be modeled as reinforcement-based learning of predictive validities of cues (Newell et al., 2007). Reinforcement learning can also be applied to learning among decision-making strategies (e.g., decisions based on single vs. multiple cues; Erev & Barron, 2005; Rieskamp & Otto, 2006). Decision making with respect to causal systems, however, seems not to be adequately captured by these models, as they lack the expressive power to represent causal relations in the environment.

Our findings support causal model theories of learning and decision making (Sloman, 2005; Sloman & Hagmayer, 2006; Waldmann, 1996; Waldmann & Holyoak, 1992) and causal Bayes net accounts (Griffiths & Tenenbaum, 2005; Pearl, 2000). The results complement previous findings, demonstrating that decision makers use causal models and causal inference when making simple one-shot decisions (Hagmayer & Sloman, 2009) and when predicting the effects of causal interventions (Meder et al., 2008, 2009; Sloman & Lagnado, 2005; Waldmann & Hagmayer, 2005).

The Role of Causal Models in Decision Analysis

We have argued that people develop an intuitive notion of the causal structure of decision problems and induce causal model representations of a choice task to support decision making. Similar ideas are found in other fields, including philosophy, artificial intelligence, and decision theory.

One common approach in decision analysis is to construct decision trees representing the available courses of action, their consequential outcomes and the associated probabilities, and the resulting payoffs. But more important is what such trees do *not* represent, namely, the exact nature of the causal dependencies in the domain and the causal relations between actions and domain variables. Consider Experiment 3, which contrasted a causal chain with a common cause model. Figure 7a shows two decision trees that one could construct from the feedback that participants experienced in the initial repeated decision-making phase (cf. Figure 6). While such trees can sometimes provide a compact graphical representation of the decision task, they lack the expressive power and inference mechanisms to evaluate changes of the decision problem. Since the trees do not encode how option do*L* leads to the occurrence of both *A* and *B*, it is not clear how we can construct the truncated trees (see Figure 7b) from the original trees (see Figure 7a). A causal model representation provides the necessary semantics to express causal interdependencies, thereby enabling inferences about novel situations. For example, one may argue that the decision tree of the chain model should express the dependency between *A* and *B* (e.g., by having separate nodes for *A* and *B*, where *A* is followed by *B*), but to generate such a tree we must have already engaged in causal learning in order to realize this particular causal dependency. In short, we can construct decision trees from causal models, but not vice versa.

While classical decision trees do not explicitly take causal structure into account, other approaches bear a closer resemblance to our ideas. In particular, so-called *influence diagrams* (Dawid, 2002; Howard & Matheson, 1984/2005; see also Pearl, 2005) have been used to introduce causal knowledge to decision analysis. Classical influence diagrams are similar to Bayesian networks in that they are also based on a graphical modeling approach using directed acyclic graphs. However, typically, the focus is not on probabilistic inferences but is on modeling various types of decision problems with the goal of evaluating the consequences of decisions in terms of expected utilities. Influence diagrams can contain different types of nodes, including decision nodes, and the arcs connecting these nodes do not necessarily represent causal relations (Howard & Matheson, 1984/2005). Thus, contrary to causal Bayesian networks, influence diagrams do not aim to model the causal structure of a domain, and the decisions do not necessarily refer to *causal* interventions.

More recent research along these lines (Dawid, 2002; see also Pearl, 2000; Spirtes et al., 1993) has aimed to connect causal models with influence diagrams to provide general semantics for

[5] Several studies have shown that awareness and learning are tightly connected to each other (see Shanks, 2010, for a recent review). As attentional factors seem to influence even very basic learning processes (e.g., classical conditioning), it is plausible that the induction of rather complex causal models cannot take place without the allocation of attentional resources.

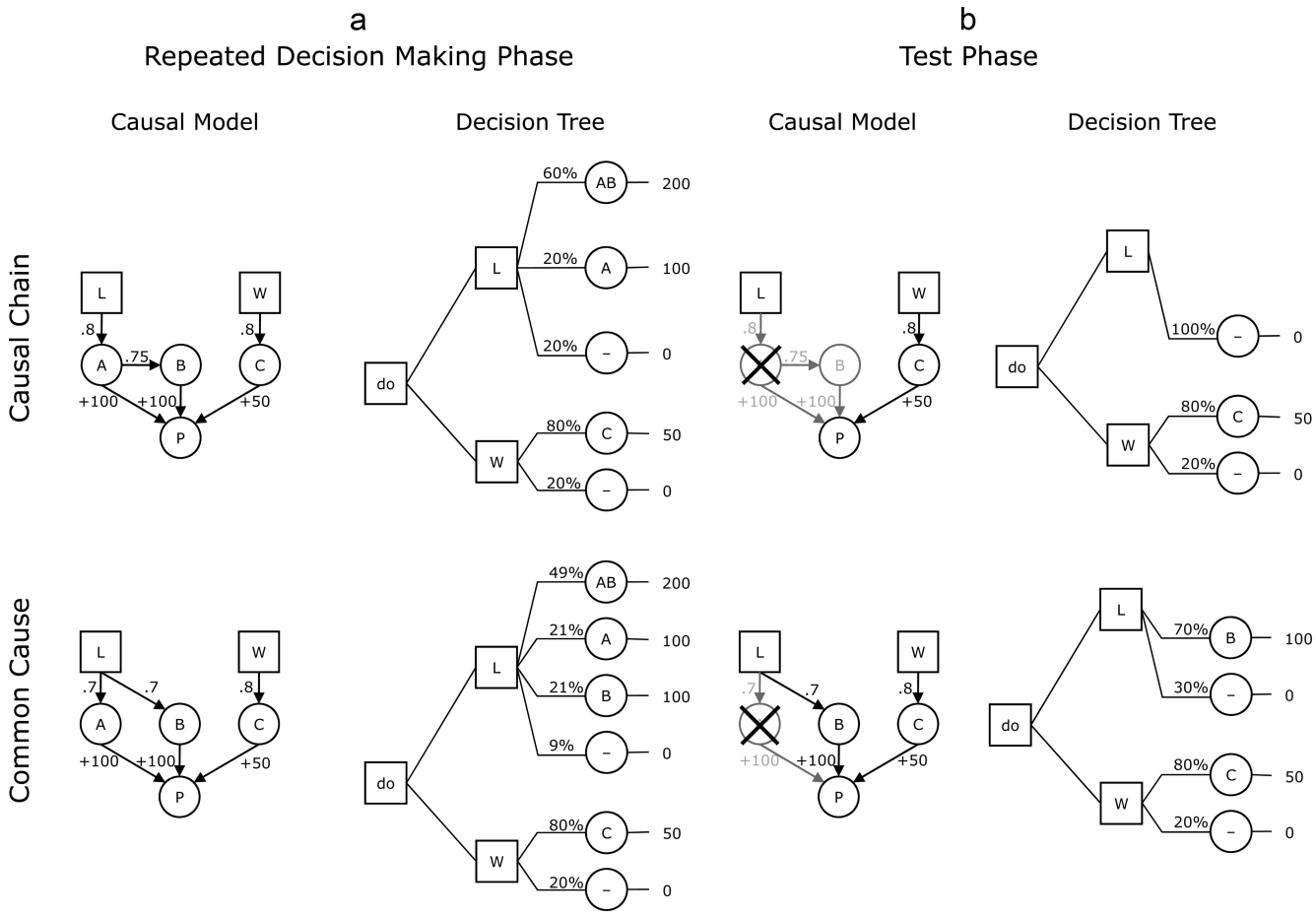


Figure 7. Causal models and possible decision trees in Experiment 3: (a) causal models and decision trees of the initial repeated decision-making phase and (b) modified causal models and decision trees in the test phase, in which variable *A* is removed from the causal system.

causal decision problems. Dawid (2002) has provided a detailed treatment of how causal Bayesian networks can be augmented with decision nodes and strategy variables for causal modeling of decision problems. The advantage of this approach is that we preserve the characteristic feature of causal models, namely, the capacity to encode conditional independence properties of joint probability distributions, while, at the same time, broadening the models' scope to decision analysis.

Limits of Causal Induction in Decision Making

Causal induction during repeated decision making may be limited by a number of factors. First, the experienced feedback must enable the decision maker to discover the underlying causal structure. Thus, feedback on the state of the variables within the system and cues to causal structure must be available (Lagnado et al., 2007). Impoverished outcome feedback pertaining only to statistical relations among actions and outcomes is not sufficient to build up elaborate causal model representations that go beyond action-outcome-payoff contingencies. This idea is consistent with research on control tasks (cf. Osman, 2010), which usually found that people mainly learned to control the system by learning contingencies between actions and outcomes.

Second, the causal model representation must be useful for the decision maker. For example, you can learn to use your washing machine or TV set without inducing a sophisticated causal model of the internal structure of the machine. In this case, learning action-outcome contingencies is sufficient. However, the situation is different when something happens to the causal system, such as a breakdown of a mechanical or electronic part. In this case, achieving a goal, such as repairing the machine, will require knowledge of the causal structure. Thus, the usefulness of causal model representations is contingent on an agent's goals and task demands.

Third, with an increasing complexity of the decision problem, the induction of causal models becomes more difficult, and data alone are rarely sufficient. In these cases, previous causal knowledge about the domain becomes crucial. Given a certain amount of prior knowledge, even sparse and noisy data may be sufficient to determine the underlying causal structure (Griffiths, Baraff, & Tenenbaum, 2004). In addition, learners may exploit a number of cues to causality, such as temporal order or knowledge communi-

cated through social learning. For example, fortunately, young doctors must not learn about the causal structure of the human body by experience, but they receive this information from teachers and textbooks.

Directions for Future Research

One may suspect that the use of causal knowledge in decision making is limited to the relatively simple problems examined here. However, there is a growing body of evidence indicating that this is not the case. For example, causal considerations seem to also play an important role in psychodiagnostic decision making (Ahn, Proctor, & Flanagan, 2009; de Kwaadsteniet, Hagmayer, Krol, & Witteman, 2010; Kim & Ahn, 2002; Kim & LoSavio, 2009). Also, when experts cannot identify the best solution immediately, they tend to construct simplified models of the domain to evaluate potential courses of action (Klein, 1998). Thus, the flexibility and adaptivity of causal model representations seems to pay off in naturalistic decision-making contexts. Future research will need to further explore the conditions under which causal models are acquired, revised, and used in decision making.

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(*Appendix follows*)

Appendix

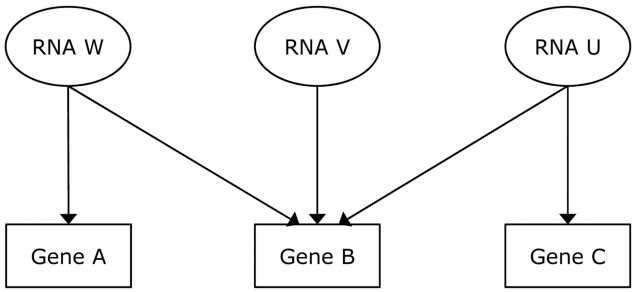
Instructions for Experiments 1–3 (Translated From German)

Experiment 1

Instructions initial repeated decision-making phase: Common cause condition. Imagine the following situation: An animal protection organization has stolen mice from a genetic engineering laboratory. The mice were genetically modified to investigate the relation between certain genes and growth. To this end, mice were bred whose growth genes are inactive.

However, the genes can be activated through so-called "messenger-RNA." You have three different types of messenger-RNA available: *W*-, *V*-, and *U*-RNA. The messenger-RNA can be used to activate genes. It is possible that the messenger-RNA can activate multiple genes or that the three genes can activate each other.

Pilot work suggests that the messenger-RNA influences the genes as follows: Injecting *W*-RNA activates genes *A* and *B*. Injecting *V*-RNA activates gene *B*. Injecting *U*-RNA activates genes *B* and *C*. However, this model is only a first hypothesis and has not been validated yet.



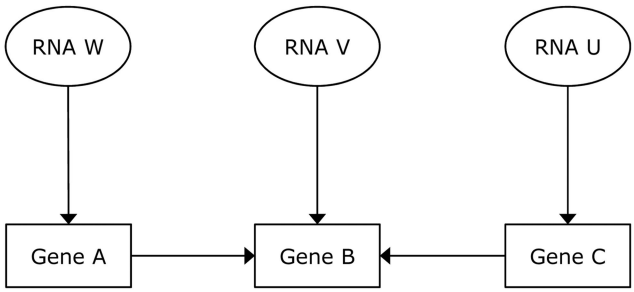
Your task is to activate the genes by injecting individual mice with *U*-, *V*-, or *W*-RNA in order to maximize the animals' hormone level. The higher the hormone level, the larger the animal will grow and the longer it will live.

You have to decide, for 30 animals, whether and what type of messenger-RNA you want to apply. At the beginning, you will have to rely on trial and error to find out how the messenger-RNA influences growth. For each animal, you will receive feedback on which genes have been activated and how the hormone level changed. Soon you will be able to choose the messenger-RNA that will maximally increase the growth-hormone level of an animal.

Instructions repeated decision-making phase: Causal chain condition. Imagine the following situation: An animal protection organization has stolen mice from a genetic engineering laboratory. The mice were genetically modified to investigate the relation between certain genes and growth. To this end, mice were bred whose growth genes are inactive.

However, the genes can be activated through so-called "messenger-RNA." You have three different types of messenger-RNA available: *W*-, *V*-, and *U*-RNA. The messenger-RNA can be used to activate genes. It is possible that the messenger-RNA can activate multiple genes or that the three genes can activate each other.

Pilot work suggests that the messenger-RNA influence the genes as follows: Injecting *W*-RNA activates gene *A*, which, in turn, will activate gene *B*. Injecting *V*-RNA directly activates gene *B*. Injecting *U*-RNA activates gene *C*, which, in turn, will activate gene *B*. However, this model is only a first hypothesis and has not been validated yet.



Your task is to activate the genes by injecting individual mice with *U*-, *V*-, or *W*-RNA in order to maximize the animals' hormone level. The higher the hormone level, the larger the animal will grow and the longer it will live.

You have to decide, for 30 animals, whether and what type of messenger-RNA you want to apply. At the beginning, you will have to rely on trial and error to find out how the messenger-RNA influences growth. For each animal, you will receive feedback on which genes have been activated and how the hormone level changed. Soon you will be able to choose the messenger-RNA that will maximally increase the growth-hormone level of an animal.

Instructions test phase. (The test phase instructions were identical for both conditions.) After you have successfully treated the initial 30 mice, your task is to treat 10 further mice, which have also been stolen from the genetic engineering laboratory. Unfortunately, you have exhausted your supplies of *U*-, *V*-, and *W*-RNA. However, a colleague provides you with *A*-RNA, which specifically activates gene *A*, and *C*-RNA, which specifically activates gene *C*. Your task is to maximize the level of the growth hormone in the individual animals by applying these messenger-RNA.

Attention: You will receive no feedback regarding the outcome of your decision on the hormone level. Nevertheless, you should maximize the hormone level of each animal.

(*Appendix continues*)

Experiment 2

Instructions were identical in all three conditions (common cause 1, common cause 2, causal chain).

Instructions initial repeated decision-making phase. Imagine the following situation: You are working in a laboratory producing vaccine against deadly diseases. These vaccines are produced with the help of genetically modified bacteria. The bacteria have been equipped with two yeast genes (genes *A* and *B*). If these genes are activated, the bacteria produce the vaccine.

For safety reasons, the genes are inactive by default. The people working in the laboratory have to activate the genes through so-called "trigger substances." These trigger substances can activate individual genes as well as multiple genes. Recently, it has also been discovered that the activity of a gene can activate other genes. In this case, the trigger substance activates one individual gene, which then triggers the activation of the other gene.

At this point in time, two trigger substances are available: *Trigger U* and *Trigger W*. However, the trigger substances do not always activate the genes. Other substances are currently under development, which will enable a more reliable and specific activation of genes. These substances might be available at a later point in time.

Your task is to maximize the amount of vaccine through applying the trigger substances.

To this end, you will be presented with 100 petri dishes one after another, with each petri dish containing the same number of bacteria. For every dish, you have to decide whether and which trigger substance to apply. After making a decision, you will receive feedback regarding which genes have been activated and how much vaccine has been produced.

In the beginning, you will have to rely on trial and error to find out how the trigger substances affect the genes, whether the genes are interrelated, and how much vaccine is produced. However, after some time, you will be able to choose the trigger that will maximize the amount of vaccine.

After you have made your decisions for the 100 petri dishes, new trigger substances will become available.

Instructions test phase. After you have successfully handled the 100 petri dishes, a new trigger substance, *Trigger A*, is at your disposal. This trigger activates, with 100% certainty, gene *A*. That means: If gene *A* also influences the other gene, the trigger will first activate gene *A*, which, in turn, might activate gene *B*. If gene *A* does not influence gene *B*, then the trigger will only activate gene *A*.

Your task is to decide for 10 additional petri dishes, which of the three trigger substances you would like to apply: *U*, *W*, or *A*. As before, your decision should maximize the amount of produced vaccine. Thus, carefully consider which trigger to apply.

Attention: You will receive no feedback regarding the outcome of your decision on the hormone level. Nevertheless, you should maximize the hormone level of each animal.

Experiment 3

Instructions were identical in both conditions (common cause, causal chain).

Instructions initial repeated causal decision-making phase. Imagine the following situation: You are working in a laboratory producing vaccine against deadly diseases. These vaccines are produced with the help of genetically modified bacteria. The bacteria have been equipped with three yeast genes (genes *A*, *B*, and *C*). If these genes are activated, the bacteria produce the vaccine.

For safety reasons, the genes are inactive by default. The people working in the laboratory have to activate the genes through so-called "trigger substances." These trigger substances can activate individual genes as well as multiple genes. Recently, it has also been discovered that the activity of one gene can activate another gene. In this case, the trigger substance activates one individual gene, which then triggers the activation of the other gene.

At this point in time, two trigger substances are available: *Trigger L* and *Trigger W*. However, the trigger substances do not always activate the genes.

Your task is to maximize the amount of produced vaccine through applying the trigger substances.

To this end, you will be presented with 100 petri dishes one after another, with each petri dish containing the same number of bacteria. For every dish, you have to decide whether and which trigger substance to apply. After making a decision, you will receive feedback regarding which genes have been activated and how much vaccine has been produced.

In the beginning, you will have to rely on trial and error to find out how the trigger substances affect the genes, whether the genes are interrelated, and how much vaccine is produced. However, after some time you will be able to choose the trigger that will maximize the amount of produced vaccine.

After you have made your decisions for the 100 petri dishes, you will receive new instructions.

Instructions test phase. After you have successfully handled the 100 petri dishes, your task is to apply the trigger substances to some further bacteria. In contrast to the bacteria you have seen before, these bacteria do not possess gene *A*. This gene is not present in these bacteria and therefore cannot be activated. However, the bacteria possess genes *B* and *C*.

Your task is to decide for 10 additional petri dishes with these new bacteria, which trigger substance (*L* or *W*) you would like to apply. As before, your decision should maximize the amount of produced vaccine. Thus, carefully consider which trigger to apply.

Attention: You will receive no feedback regarding the outcome of your decision on the hormone level.

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