Inductive Learning, Uncertainty and the Acquisition of Causal Models

Manfred Thüring (thuering@gp.tu-berlin.de)
Department of Cognitive Psychology, Berlin University of Technology,
Franklinstr. 5-7, 10587 Berlin, Germany

Uwe Drewitz (drewitz@gp.tu-berlin.de)
Department of Cognitive Psychology, Berlin University of Technology,
Franklinstr. 5-7, 10587 Berlin, Germany

Leon Urbas (leon.urbas@tu-berlin.de)
Center of Human-Machine-Systems, Berlin University of Technology,
Jebensstr. 1, 10623 Berlin, Germany

Abstract

Causal models can be regarded as fundamental knowledge bases consisting of rules for generating explanations and predictions. As we all know, such inferences are not free from uncertainty. In an experiment about acquiring causal models by induction, we investigate the impact of the validity of such models on the certainty of inferences. The results indicate that preliminary models are revised in the light of new information, and that the degree of validity considerably influences the certainty of predictions.

Keywords: Mental models; conditional rules; causal inferences; induction; uncertainty.

Introduction

Causal reasoning is one of the cornerstones of human cognition and seems as fundamental as our concepts of space and time. Explanations and predictions are crucial for everyday thinking as well as for scientific research. They heavily influence our understanding of the world and guide our judgments, decisions and actions in many respects.

Well aware of the central role of causality, philosophy and psychology have been exploring the mental mechanisms that underlie causal thinking for many years (White, 1990). Examples of research issues are the perception of causal relations, the structure of causal knowledge or the mechanisms of learning and reasoning in causal contexts.

In this paper, we address the issue of acquiring causal models by inductive learning. In particular, we investigate how a causal model may gradually evolve from observations and how its structure may influence the content and certainty of predictive inferences.

Basic causal models

To start with, what do we mean when we say "A causes Z", as in the statement "smoking causes lung cancer"? Mackie (1974) proposes four possible interpretations:

- A is a necessary and sufficient condition for Z. Smoking (A) is the only cause for lung cancer (Z) and always leads to that effect.
- A is a necessary but insufficient condition for Z. Only smokers get lung cancer, but some of them do not, because they are lacking the particular genetic predispositions (C) that cause lung cancer in conjunction with smoking.
- A is a sufficient but unnecessary condition for Z since other conditions lead to the same effect. Smoking causes lung cancer, but the inhalation of asbestos particles (B) does as well.
- A is an insufficient but necessary part of a condition which itself is unnecessary but sufficient for Z (i.e., either A and C or B cause Z). Smoking in conjunction with certain genetic predispositions causes lung cancer or – alternatively - cancer is caused by asbestos particles.

Mackie's taxonomy of causal statements points to four basic knowledge structures constituting the different meanings of "A causes Z". These structures can be regarded as causal models in the sense of Craik (1943) who coined the term "mental model" for domain specific knowledge of predictive and explanatory power. To represent the (in)sufficiency and (un)necessity of conditions for an effect, a causal model can be expressed in terms of conditional rules. Based on Mackie's definition, four types of models can be distinguished. In this paper, we address three of them.

Model of Unique Causation (MUC)

A is the only cause for Z. Whenever A occurs Z will occur, and whenever A is missing Z will be missing as well. This implies two conditional rules.

R_{MUC1}: A → Z
R_{MUC2}: -A → -Z

Model of Complex Causation (MCC)

A in conjunction with another condition C forms a complex cause that leads to Z. When A and C occur, Z will occur. When either A or C are missing, Z will not appear. This can be represented by three rules.

R_{MCC1}: (A & C) → Z
R_{MCC2}: -A → -Z
R_{MCC3}: -C → -Z

Model of Multiple Causation (MMC)

A or B cause Z. When A or B are present, Z will occur too. When both A and B are missing, Z will not appear either. Again, three rules express these relationships.

R_{MMC1}: A → Z
R_{MMC2}: B → Z
R_{MMC3}: (-A & -B) → -Z

Model four is a combination of MMC and MCC, called Model of Multiple Complex Causation (Thüring, 1991).

All these models represent different types of causal knowledge that people acquire by gathering experience in a domain and that are used to explain or to forecast events of interest. In our example for instance, a person who believes that a certain predisposition is necessary in conjunction with smoking for developing lung cancer (MCC) will tend to other predictions than a person who believes that smoking on its own will lead to this disease (MUC). Hence, the structure of a model and the contents of its rules determine the inferences that a persons draws about a case.

Compared to the vast amount of causal relationships and their complex intertwining stored in human memory, the MUC, MCC and MMC are apparently simple and limited, and of course we do not assume that they represent comprehensive knowledge bases. Instead, we propose to regard them as basic structures from which such knowledge bases are built. The exact structure and size of an extensive causal model depends on the number of conditions it contains and the relations between these conditions (i.e. terms as "A", "B" and "C" in the rules of basic models are meant as placeholders for sets of conjunctively or disjunctively combined conditions). This conceptualization makes it is possible to describe elaborated causal models as combinations and extensions of the basic models given above.

Uncertainty of causal inferences

When the conditions of a rule in a causal model are matched by adequate information, it produces an inference predicting either the occurrence or the non-occurrence of the event the rule is about. From the perspective of propositional logic, such deductions leave no room for uncertainty. On the other hand, we all know that explanations and predictions are usually far from being certain. This raises the question what factors may lead to the uncertainty of a causal inference deduced from the rule of a mental model.

Two factors have been proposed to influence the uncertainty of explanations and predictions: (a) the ambiguity of the information available for a causal inference, and (b) the validity of the causal model (Thüring, 1991).

Ambiguity can be defined as the perceived amount of missing information (Frisch & Baron, 1988). This amount depends on the structure of the causal model and the extent to which given information matches the conditions of its rules. To illustrate this relationship, imagine a person who relies on the MCC to predict an event Z. When this person receives confirming or disconfirming information about "A" and "C" then the situation is unequivocal (i.e., all data are available required for the model to forecast the occurrence or non-occurrence of Z). On the other hand, imagine this person receiving only information confirming "A" while "C" remains unknown. This case is a typical example for an ambiguous situation, since the occurrence of Z depends on whether condition "C" is fulfilled or not. If "C" is true in addition to "A" the condition of rule R_{MCC1} is completely matched and "Z" should be inferred. If "not C" is true the condition of R_{MCC3} is fulfilled and "not Z" should be predicted.

The experienced validity of a causal model depends on the number of correct and false inferences derived from the model in the past. Imagine a person with rather limited causal knowledge as in the case of the mono-causal model MUC. Whenever this person receives the information "A" she will predict Z, and whenever she receives the information "not A", she will predict "not Z". In both cases, the confidence in the rules of the model should increase when the prediction is right. However, when the causal model does not represent all causal conditions which are really responsible for Z, such inferences may be wrong. Firstly, "A" may not be a sufficient condition for Z to appear. In this case, the "real" causal relationship may be that "A" in conjunction with another condition is required for Z. Secondly, "A" may not be a necessary condition for Z (i.e., although "not A" is true, Z appears). In this case, the real causal relationship may be that an alternative cause "B" may lead to Z, too. In both cases, the person's model lacks important knowledge and the wrong predictions should decrease her confidence in the rules she uses.

Contingency information as in this example has been investigated in a multitude of studies concerned with the "strength" of a causal relation, and a number of theories have been proposed to predict causal strength from the frequencies in the cells of a contingency table. Prominent approaches are the ΔP rule (Jenkins & Ward, 1965), the Power PC theory (Cheng, 1997), the pCI rule (White, 2004) and the Belief Revision Model (Catena et al., 1998). All of them focus on covariation and try to predict the perceived strength of a causal relationship. None of them addresses the problems of how contingency information might be understood on the basis of preexisting causal knowledge or how such information might influence the certainty of a prediction or explanation.

Causal models are a promising conceptual framework to tackle these problems. Since they capture both, the ambiguity of given data as well as the validity of causal rules, they can be used to predict the content as well as the certainty of a causal inference. In this respect, they are similar to causal networks (Pearl, 2000), but in contrast to this approach, they stress the heuristic, non-bayesian nature of human probability assessment. Based on theoretical assumptions about heuristics for judging the likelihood of events (Einhorn & Hogarth, 1986), Thüring (1991) proposes a theory which describes the processes by which inferences are derived from causal knowledge. This theory also specifies a formal algorithm to predict the certainty of causal inferences from the validity of causal rules represented in the model and the perceived ambiguity of the available data.

The influence of ambiguity on the certainty of causal inferences could be demonstrated in a number of experiments (Thüring, 1991; Thüring & Jungermann, 1992; Jungermann & Thüring, 1993). Participants learned models of fictional diseases which were structured as the MCC, MMC or MMCC. After ensuring that the diseases were properly understood, sets of data about hypothetical patients were presented. While the validity of the models was held constant, the data about the cases differed with respect to

ambiguity (i.e., the degree of matching between data and causal rules was varied.) Data sets contained either positive evidence – for example information matching rules with positive conditions as in R_{MUC1} - or they contained negative evidence, thus matching rules with negative conditions, such as R_{MUC2}. For both types of evidence and for explanations as well as predictions, the experiments showed a pronounced impact of ambiguity on the certainty of causal inferences. Increasing the degree of matching (and thus decreasing ambiguity) led to higher subjective probabilities of any prediction or explanation derived from a rule.

Further evidence comes from experiments reported by Molz (2002) who also found an increase of the certainty of causal inferences when the ambiguity of information decreased. Moreover, he detected an influence of the validity of causal models on the subjective probabilities of inferences. Conclusions based on models of low validity were considered as less certain than conclusions derived from models of high validity. This influence, however, was much less pronounced than assumed. A reason for this unexpected result may lie in the way subjects were trained in these experiments. Causal models as well as their validity were not acquired by observing sets of data, but were described in texts. Information about validity was given by sentences, such as "this diagnosis proved right in one of three cases". Since this information was only given once per model, its influence might be rather minor compared to ambiguity which had to be accounted for in each judgment about a patient.

To summarize, the knowledge base of explanations and predictions can be conceptualized as a causal model consisting of a number of rules. These rules represent knowledge about positive and negative evidence with respect to the occurrence or non-occurrence of the phenomenon the model is about. The ambiguity of data and the validity of rules are regarded as factors which influence the certainty of inferences derived from the model. Both factors can be expressed as model parameters. While the extent of ambiguity is given by the degree of matching between data and rule conditions, the validity depends on the relation between correct and incorrect inferences that were deduced from the rules of the model in the past. While the influence of ambiguity on the certainty of causal inferences has been documented convincingly, the influence of validity needs further clarification.

Experiment

To investigate the influence of validity on the certainty of causal inferences, we set up an experiment in which causal models were gradually acquired by inductive learning. The goal of the experiment was to answer three questions:

(1) Does the certainty of a causal inference increase with growing validity of the rule it is derived from?
(2) What happens to the certainty of inferences drawn from a well proven model when the validity of the model starts to decrease?
(3) In which way do people revise the rules of their model when its validity is diminishing?

The task for the participants of the experiment was to acquire knowledge about a technical system in order to predict its state. The system was a fictional pump system regulating the cooling of a power plant. In each trial, participants received information about the states of four components of the pump system and had to predict the future state of the overall system. In addition, they had to judge the certainty of their prediction. At the end of each trial, they were informed about the correctness of their forecast.

The experiment consisted of three successive blocks of trials. The first block endorsed a simple causal model (the MUC), the second block provided evidence against that model to reduce its validity, and the third block offered information to revise the MUC by transforming it into a more complex model, such as the MMC or the MCC. This fixed sequence of blocks corresponds to a common situation in inductive learning where people start with a simple hypotheses which proves as deficient and must be modified in the face of new evidence.

Participants

Forty two participants were recruited for the experiment, twenty four of them were women. Only lay people with respect to the technology of power plants were chosen. All persons were paid for their participation.

Materials

The instruction informed people that they were participating in a learning experiment. A short scenario was presented in which the pump system was characterized as an important part of a power plant. People were told that it consisted of four subsystems and that its state depended on the states of these components – but they were not informed about the specific causal relationships between those states. Instead, they were told that their task was to find out which states of the subsystems entailed a proper functioning of the whole system, and which states led to a malfunction.

The state of each subsystem was indicated on a dial (see figure 1). Each dial could be "on" (subsystem A and C) or "off" (subsystem B and D). Only if the dial was on, the state of the subsystem could be read. In this case, the needle of the dial was either on the right side - indicating that the subsystem was "up" and functioning properly (subsystem A) - or the needle was on the left side – indicating that the subsystem was "down" (subsystem C). If the dial was off, no information about the corresponding subsystem was available. In this case, the dial was shown in grey (subsystem B and D). Altogether, the state of each subsystem was either "up", "down" or "unknown".

Participants could judge the state of the whole system by pressing one of two buttons labeled "ok" or "malfunction". For estimating the certainty of their prediction, they had to use the scale shown in figure 1. The state of the whole system was indicated in a separate field displaying either the message "ok" or "malfunction".

All materials were evaluated in three pretests with a total number of 20 persons. Instruction, technical scenario and graphics were revised twice according to the participants' comments. The third pretest finally ensured that the scenario provided a plausible cover story and that instruction, task as well as all materials were precise and comprehensible.

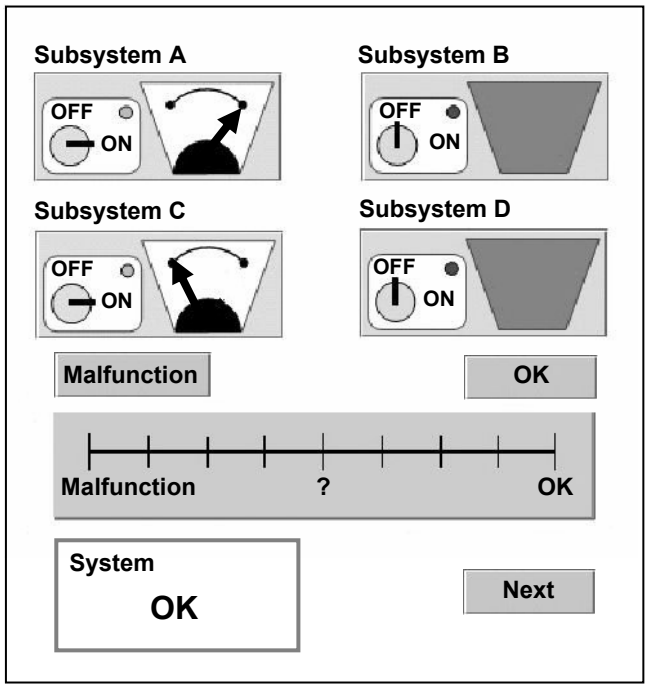

Figure 1: Overview of materials used in all three blocks.

Procedure

The experiment was run on an IBM PC under Windows XP. Each participant read the instruction and could ask any questions before the experiment began. In each trial, the participant was first shown the dials of the subsystems. The field indicating the state of the whole pump system was grey. To predict the state of the whole system, the participant pressed either the button labeled "ok" or the button labeled "malfunction". Then she judged the certainty of her prediction by adjusting the slide shown in figure 1. After this judgment, she was informed about the correctness of her response by displaying either the message "ok" or "malfunction" in the field labeled "system". After this feedback, she started the following trial by pressing a "next" button.

The experiment consisted of three blocks of trials in a fixed sequence. Within each block, trials followed a random order. The first block aimed at inducing a model of unique causation (MUC) in which the proper functioning of subsystem A appeared as a sufficient and necessary condition for the proper functioning of the whole pump system. The block consisted of twelve trials per rule. The combination of "subsystem A is up" and "the whole system is functioning properly" was presented to induce and reinforce the rule R_{MUC1}. The combination of "subsystem A is down" and "the whole system has a malfunction" was presented to induce and reinforce the rule R_{MUC2}. To support an early formation of rules, the dials of the other systems were turned off during the first three trials for both rules. This was changed for the subsequent trials. The dials of other systems were turned on as well, but the information they provided was always consistent to the rules of the MUC. Although this consistency ensured that no evidence was presented conflicting to the MUC, information about the other three subsystems are distractors in this context.

The second block consisted of six trials per rule and was designed differently for two groups of our participants. The first group received information contradicting the *sufficiency* of the MUC. People were provided with data which were inconsistent with R_{MUC1} (i.e., the whole system was not "ok" although subsystem A was "up"). The second group received information contradicting the *necessity* of the MUC. People were provided with data which were inconsistent with R_{MUC2} (i.e., the whole system was "ok" although subsystem A was "down").

The third block had ten trials per rule and was again different for the two groups. The first group was given information that could be used to reestablish the *sufficiency* of the causal model by expanding the model of unique causation (MUC) into a model of complex causation (MCC). To do so, people had to recognize that subsystem A had to be "up" in conjunction with subsystem C. To represent this relation, the three rules of the MCC had to be acquired. Only data were provided which were in accordance with these three rules. The second group was given information that could be used to reestablish the *necessity* of the causal model by expanding the MUC into a model of multiple causation (MMC). To build up that model, people had to recognize that a failure of subsystem A was compensated when subsystem B was "up". Hence, proper functioning of subsystem B was an alternative cause for the proper functioning of the whole system. The representation of these relations required the generation of the three rules of the MMC. For this group, only data were presented which were in accordance with these three rules.

Throughout the whole procedure, two basic conditions were fulfilled. Firstly, it was ensured from the beginning that no data sets were presented which were inconsistent to the final model which had to be acquired by each group. This means that even the data sets of the first trials aiming at building up the preliminary model of unique causation were chosen in a way that neither contradicted the MCC nor the MMC. Secondly, no ambiguous data sets were used. This is important for block three because this block was the first one where ambiguity could arise. For the MMC, an ambiguous situation would arise if rule R_{MCC1} was only partially matched by the information of a data set. For the MCC, data would be ambiguous which do not match rule R_{MMC3} completely. Since any partial matching of that kind was completely avoided, ambiguity – as defined above – was ruled out in this experiment.

Design

Different independent variables were relevant for the three blocks. For the first block, a 2-factorial design with repeated measurement was realized. The first factor was called "rule" with the treatments "ok" (for rules predicting a proper functioning of the system) and "malfunction" (for rules predicting a failure of the system). The second factor was the number of reinforcing trials, called "reinforcement", with twelve treatments. For block two, the factors were "rule" (as in block 1) and "discrediting" with six treatments (i.e., the number of trials with information inconsistent to

the rules of the MUC). For the third block, the factors were "rule" with two treatments and "reinforcement" with ten treatments. All designs were designs of repeated measures and used certainty judgments as major dependent variable.

Results

Figure 2 to 5 show the mean certainty judgments as well as the percentage of participants producing the inference predicted by the causal model which was supposed to be used. An analysis of the percentages revealed that an inference opposite to the one proposed by the model was drawn in four trials (i.e., in trial 6 of block one, in trial 4 of block two and in trial 4 in block three for the MMC as well as for the MCC). Since these trials failed to reinforce or to discredit the model as planned we decided to exclude them from the statistical analysis. In each case, the reason for the opposite inference is a distractor which will be explained in the discussion.

First block: Reinforcing the MUC

Figure 2 illustrates the gradual increase of certainty with the growing number of reinforcing trials for both rules. This trend is interrupted in trial 6 where certainty judgments suddenly drop. As mentioned above, this dramatic change is caused by a distractor which led 74% of the participants to an opposite inference.

An ANOVA revealed a significant main effect of the factor "reinforcement" ($F(10,410) = 12.00$, $p < 0.001$, $\eta^2 = 0.226$) and a significant interaction of "reinforcement" and "rule" ($F(10,410) = 4.51$, $p < 0.001$, $\eta^2 = 0.099$). The factor "rule" had no significant effect.

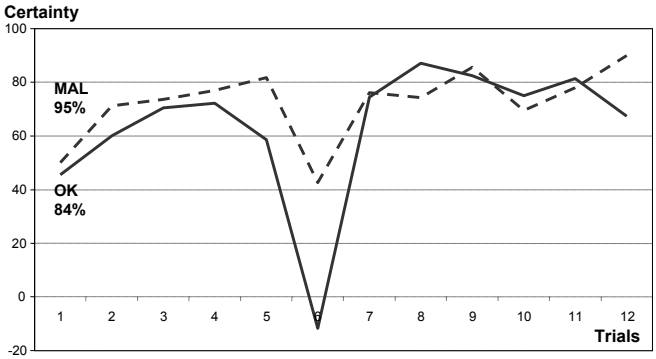

Figure 2: Mean certainty judgments for the MUC depending on the number of reinforcing trials.

Second block: Discrediting the MUC

Figure 3 shows the percentage of predicted inferences and the development of mean certainty judgments over six trials discrediting the rules of the MUC. This time, the fourth trial produced an opposite inference.

Data were analyzed by an ANOVA. The factor "discrediting" had a significant effect on mean certainty judgments ($F(3.3, 65.9)=15.70$, $p < 0.001$, $\eta^2 = 0.440$. Moreover, a significant interaction between "discrediting" and "rule" was shown $F(3.3, 62.6)=3.42$, $p < 0.021$, $\eta^2 = 0.146$). There was no main effect of the factor "rule".

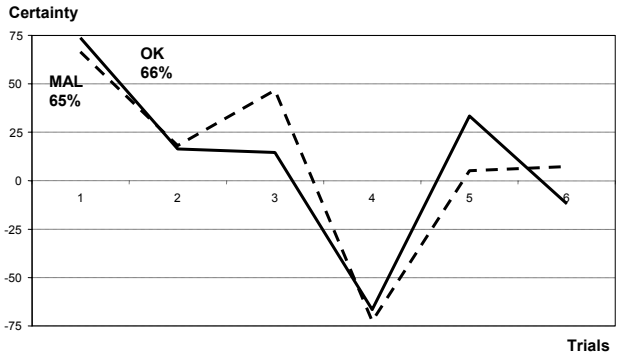

Figure 3: Mean certainty judgments for the MUC depending on the number of discrediting trials.

Third block of trials: Reinforcing MCC and MMC

Percentages of predicted inferences and mean certainty judgments given by participants who learned the MCC are shown in figure 4. An opposite inference was drawn in the fourth trial. An ANOVA revealed two significant effects: a main effect of the factor "reinforcement" ($F(9, 180)=9.15$, $p < 0.001$, $\eta^2 = 0.314$) and an interaction effect of "reinforcement" and "rule" ($F(9,180)=4.75$, $p < 0.001$, $\eta^2 = 0.192$).

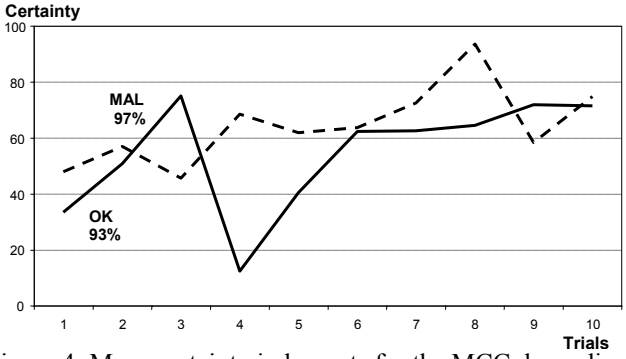

Figure 4: Mean certainty judgments for the MCC depending on the number of reinforcing trials.

The other group of participants who learned the MMC produced mean certainties that are illustrated in figure 5. Again, an inference opposite to the model appeared in trial 4.

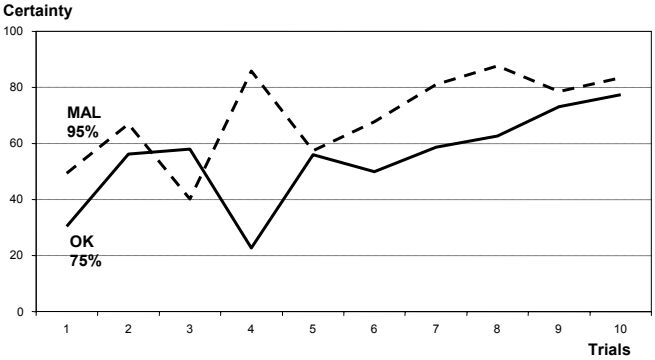

Figure 5: Mean certainty judgments for the MMC depending on the number of reinforcing trials.

The ANOVA of the data of this group showed an effect of "rule" ($F(1,20)=10.29$, $p < 0.004$, $\eta^2 = 0.340$) and of "reinforcement" ($F(9,180)=8.96$, $p < 0.001$, $\eta^2 = 0.309$) as

well as a significant interaction of these two factors ($F(9,180)=7.59$, $p < 0.001$, $\eta^2 = 0.275$).

Discussion

Let us now return to the three questions raised at the beginning of this chapter.

The results of the first experimental block support the assumption that people can easily build up a basic causal model, such as the MUC. As the high percentage of inferences in concurrence with the causal model show, in most people infer the states of the technical system as predicted. The certainty of these judgments increases with the number of trials confirming the rules of the model. This can be equally observed for rules whose conditions are matched by positive evidence, such as R_{MUC1}, and for rules employed to evaluate negative evidence, such as R_{MUC2}.

For inferences deduced from such rules, a relatively high level of certainty is reached pretty fast and can be preserved as long as no "noise" is generated by information about irrelevant conditions. In block one, such information is provided in the sixth trial and concerns subsystem D which is causally irrelevant for the MUC. In the preceding trials, the display for D had always been turned off with the exception of one data set, where subsystem D had been "up" together with subsystem A. Since in that case the resulting state of the entire pump system had been "ok", people might have formed the hypothesis that D in *conjunction* with A was necessary for this state (i.e., that the pump system would malfunction if either A or D were "down"). This could explain why our participants drew an inference opposite to the MUC in the sixth trial, where D was presented as "down", and why their certainty judgments dropped accordingly. Very similar data constellations and corresponding hypotheses were probably responsible for the other conspicuous values mentioned in the result section, such as the mean certainties of trial four in figure 3, 4 and 5. Such deviations from the expected subjective certainties hint at the existence of more than one cognitive mechanism for inductive rule formation and the derivation of certainties. While the validity of a rule might heavily influence the certainty of predictions under regular circumstances, competing rules obviously come into play when data constellations leave room for new hypotheses.

At the end of block one, the MUC seems to be pretty stable with respect to the certainty of inferences. Although the data sets remain "noisy", judgments of certainty are mostly close to 0.80 or above and do not fall below 0.73. This changes considerably in the second block which aims to discredit the MUC. As soon as data sets occur, which violate the rules of the model, the certainty of inferences starts dropping. Moreover, the percentage of people producing the predicted inference decreases as well. These observations provide the answer to our second question. A decrease of the validity of a causal model has two effects. Firstly, the certainties of inferences deduced from the model decrease. Secondly, people seem to start forming rules producing alternative conclusions even though conclusive information for revising the model is still lacking.

Such information is not provided before the third block which aims at substituting the simple MUC by a MCC for one group of persons and by a MMC for the other group. In answer to our third question, this block suggest that people use such information to revise and extend the model they started with. In the course of inductive learning, a significant increase of the certainty of predictions is found with a growing number of trials reinforcing the models.

Although the development of certainty judgments over all three blocks leaves the impression of a regular pattern, it should not be forgotten that a variety of deviations occurred from what we had expected. This not only concerns the influence of distractors, but also the interaction between the factors "rules" and "reinforcement" respectively "discrediting". This interaction shows that some trials led to certainty judgments departing from the general trend induced by reinforcing or discrediting a model. It may indicate that people do not value the information about different cases equally and that they start looking for alternative rules much earlier than expected. Further research is required to investigate the cognitive processes underlying these phenomena and to describe them in terms of rule-based causal models.

References

Catena, A., Maldonado, A. & Cándido, A. (1998). The effect of the frequency of judgement and the type of trial on covariation learning. *Journal of Experimental Psychology: Human Perception and Performance*, 24, 481-495.

Cheng, P. W. (1997). From covariation to causation: a causal power theory. *Psychological Review*, *104, 2*, 367-405.

Craik, K. (1943). *The nature of explanation*. Cambridge, MA.: Cambridge University Press.

Einhorn, H. J. & Hogarth, R. M. (1986). Judging probable cause. *Psychological Bulletin*, *99, 1*, 3-19.

Frisch, D. & Baron, J. (1988). Ambiguity and rationality. *Journal of Behavioral Decision Making*, 1, 149-157.

Jenkins, H.M. & Ward, W.C. (1965). Judgement of contingency between responses and outcomes. *Psychological Monographs: General and Applied*, 79, 1, 1-17.

Jungermann, H. & Thüring, M. (1993). Causal knowledge and the expression of uncertainty. In G. Strube & K. F. Wender (Eds.), *The cognitive psychology of knowledge*. Amsterdam: Elsevier.

Mackie, J. L. (1974). *The cement of the universe.* Oxford: Clarendon Press.

Molz, G. (2002). Knowledge reliability and information ambiguity: two determinants of causal inferences. *Cognitive Processing*, *3-4*, 31-42.

Pearl, J. (2001). *Causality: Models, reasoning, and inference*. Cambridge: Cambridge University Press.

Thüring, M. (1991). *Probabilistisches Denken in kausalen Modellen*. Weinheim: Psychologie Verlags Union.

Thüring, M. & Jungermann, H. (1992). Who will catch the Nagami Fever? Causal inferences and probability judgment in mental models of diseases. In D. A. Evans & V. L. Patel (Eds.), *Advanced models of cognition for medical training and practice* (pp. 307-325). Berlin: Springer.

White, P.A. (1990). Ideas about causation in philosophy and psychology. *Psychological Bulletin*, 108, 1, 3-18.

White, P.A. (2004). Causal judgement from contingency information: A systematic test of the pCI rule. *Memory & Cognition*, 32, 3, 353-368.