Using Causal Persuasive Arguments to Change Beliefs and Teach New Information: The Mediating Role of Explanation Availability and Evaluation Bias in the Acceptance of Knowledge

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In this article processes by which causal arguments effect change in established beliefs were explored. The hypothesis that explanation availability mediates belief change in response to causal arguments was tested in 2 experiments. Persuasive communications used causal evidence, noncausal (statistical) evidence, or both to argue that AIDS is not transmissible by casual contact. Results supported the authors' hypothesis. Causal arguments produced the greatest belief change, with the effect mediated by explanation availability. Causal arguments were also less subject to evaluation bias, consistent with a cognitive interpretation of the biased assimilation phenomenon (in which evidence in favor of one's position is evaluated more favorably than evidence in opposition). Experiment 2 replicated the basic effects with dependent measures (including a measure of behavioral commitment) obtained 3 weeks after presentation of the communications. These results suggest that a causal component strengthens the educational potential of persuasive arguments.

Persuading an audience to accept new information seems particularly difficult when audience members can explain why they hold their current beliefs. The persuasion task then goes beyond simply convincing an audience of the correctness of the new information; persuasion must also overcome forces that maintain established beliefs. For example, in teaching social psychology, we have seen students resist ideas that challenge their common sense understanding of social behavior. Of course, common sense is derived from life experience and is rich with explanations. Researchers studying learning in children have observed a similar phenomenon. In several studies, incorrect prior knowledge interfered substantially with the learning of correct scientific information (Alvermann & Hague, 1989; Alvermann, Smith, & Readence, 1985; Lipson, 1982), particularly when the incorrect knowledge was activated. As Lipson (1982) noted, "Subjects . . . were remarkably impervious to changing their schemata regarding familiar topics" (p. 258).

Health officials have faced a similar formidable challenge educating the public about AIDS. In relation to AIDS, everyone has experienced common cold or flu viruses and witnessed the spread of these viruses through casual everyday interactions between people. It should not be surprising then that people believe that HIV (the so-called AIDS virus) is spread in similar ways. People can easily (though incorrectly) explain why casual interaction with a person with AIDS is dangerous or why insects such as mosquitoes pose a hazard in spreading a blood-related disease. To understand why such beliefs are difficult to change, and what kind of information could be most effective in prompting change, we consider in this article the causal nature of beliefs and the role of causal arguments in promoting change.

Argument in Persuasion

In introducing the availability heuristic, Tversky and Kahneman (1973) defined availability as "the ease with which instances or associations could be brought to mind" (p. 208; also in Kahneman, Slovic, & Tversky, 1982, p. 164). The availability of causal arguments (explanation availability) has been demonstrated to play a key mediating role in the perseverance of beliefs (Anderson, New, & Speer, 1985) but has not received detailed study in the literatures that focus on belief change. To be sure, much has been written concerning the importance of arguments per se in both the group polarization literature (e.g., Burnstein & Vinokur, 1977) and the persuasion literature, in which it has been shown that highly involved audiences attend to the quality of arguments; whereas less involved audiences may "count" arguments as a heuristic in determining the validity of a message (Petty & Cacioppo, 1984). Strong arguments are more persuasive than weak arguments if the audience is sufficiently involved to think about the arguments. However, such findings leave open questions of what constitutes a strong or effective argument or by what processes arguments instill belief change. Past research on argument
strength has operationalized the concept of strong arguments only in terms of the favorability of audience elaboration (see O'Keefe, 1990, p. 104 for a critique of research on argument strength in persuasion) but has "postponed the question of what specific qualities make arguments persuasive" (Petty & Cacioppo, 1986, p. 32). We propose that a causal component to such arguments can change beliefs by altering the balance of explanations available in support of alternative beliefs, and we report on two experiments that show that this may be a significant process for belief change.

Beliefs relating variables in the social environment (known as social theories) have a fundamentally causal nature that contributes to the stability and continuity of beliefs (e.g., Anderson, 1983). Social theories are not simply beliefs about whether variables are related; they are beliefs about why they are related, and people tend to seek such causal information (Nisbett & Ross, 1980; Ross, 1977; Ross & Anderson, 1982).

In numerous studies, researchers (cf. Alvermann et al., 1985; Jelalian & Miller, 1984; Lipson, 1982; Ross & Anderson, 1982; Ross & Lepper, 1980) have documented the perseverance of beliefs, including a variety of social theories (Anderson, 1982, 1983; Anderson, Lepper, & Ross, 1980; Anderson & Sechler, 1986). Most relevant here is work demonstrating that causal thinking mediates the perseverance of social theories (see Anderson, 1989, for a brief review). Anderson et al. (1980) found that students who explained a belief supported by weak case study data generally continued in that belief even after being informed that the case studies were fictitious, suggesting that explanation availability was mediating perseverance. Indeed, the effect occurred only for students whose explanations included causal components. Anderson (1983) demonstrated that such concrete case study data stimulated spontaneous causal processing, making beliefs based on that form of data particularly tenacious. Statistical data that actually produced stronger initial beliefs produced significantly less spontaneous causal thought and significantly less perseverance. Anderson et al. (1985) measured availability of competing arguments at the time final beliefs were assessed and found that perseverance was substantially accounted for by the relative availability of supporting explanations.

Increasing the availability of counterexplanations (i.e., explanations supporting alternative relations) has been shown to decrease perseverance. Anderson (1982) required participants to write explanations for alternative relations either before or after seeing case study data. Both procedures reduced perseverance, suggesting that causal thought may be the key to changing as well as maintaining beliefs. Anderson and Sechler (1986) and Lord, Lepper, and Preston (1984) produced similar counterexplanation effects.

Belief Perseverance and Change: Learning New Information

From these findings, a model of belief perseverance and belief change emerges. All else being equal, a social theory will persist to the extent that there are relatively more explanations available to the believer to support the original theory than to oppose that theory or to support an alternative theory (Anderson et al., 1980; Anderson et al., 1985). By the same token, a social theory should be vulnerable to change when causal arguments shift the balance in available explanations in favor of a new belief. Noncausal arguments, which do not contain explanations, should not have this effect on explanation availability and so would not be expected to be as effective in promoting belief change and the learning of new information. As we use the term here, a causal argument contains information regarding an underlying mechanism that could account for the relationship embodied in a theory. (When such mechanism information can be brought to mind by an individual, it is said to be an available explanation for the theory.) A noncausal argument does not contain such mechanism-related information but may instead support a theory with direct evidence that the relationship embodied in a theory has been observed in the covariation of the variables that make up the theory.

We should note that it is possible for there to be a true causal relationship underlying a theory without an individual knowing the mechanism involved. That would not affect our distinction between a causal and a noncausal argument, however. A causal argument conveys information regarding a mechanism, whether that mechanism is the true cause of the relationship, or even whether there is a true causal relationship. A noncausal argument cites the degree of covariation as evidence for or against a theory, without citing a mechanism, even when the theory represents a true causal relationship (in which, for example, a carefully controlled experiment might have indicated that two variables are causally related).

Of course, the relative availability of explanations is not completely a function of explanations generated in the external environment, such as those provided in a causal argument. Cognitive response theory argues that belief change occurs primarily as a function of internal thoughts and elaborations provoked by stimuli in the external environment (for a review, see Fiske & Taylor, 1984, Chapter 12). The degree of internal elaboration depends on the characteristics of both the person (e.g., Bransford et al., 1982; Cacioppo & Petty, 1982; Cacioppo, Petty, Kao, & Rodriguez, 1986; Petty & Cacioppo, 1981) and the external stimulus (e.g., Anderson, 1983; Chaiken, 1980; Sawyer, 1981; Wood & Eagly, 1981). Thus, people may differ in their tendency to generate explanations, even in response to noncausal arguments. For this reason, as we compared the effectiveness of causal and noncausal arguments, we expected that explanation availability should predict final beliefs within groups of adults responding to a certain form of argument, just as we predicted that it will provide the mechanism for producing differences between groups. In short, explanation availability should mediate the effect of arguments on participants' final beliefs, influencing how much the audience actually learns from the new information presented.

Other research has suggested additional reasons to question the value of noncausal arguments. Although no re-
searchers in previous studies have directly compared the impact of causal arguments with the impact of noncausal statistical arguments, many studies have shown that people undervalue statistics. People underuse base-rate information in making judgments (e.g., Kahneman & Tversky, 1973), and pallid statistical information has less impact on people than vivid concrete information (Nisbett, Borgida, Crandall, & Reed, 1976). Ahn, Kalish, Medin, and Gelman (1995) showed that people typically do not seek out covariation information in making causal attributions for events. Most important for the current discussion, Lord, Ross, and Lepper (1979) demonstrated that people discount noncausal statistical evidence that is in disagreement with their current position. People strongly favoring or opposing capital punishment examined two statistical studies: one supporting and one disputing the death penalty's deterrent effect. Participants in their study did not accept all of the information at face value, but they selectively criticized the study that opposed their view and found this opposing evidence less convincing.

Should this bias in evaluating evidence be less pronounced for causal evidence? If the bias is purely a result of motivational factors leading people to reject opposing evidence, then the bias should be equally present in causal and noncausal evidence. However, Lord et al. (1984) argued that the bias arises from errors in cognitive processing, not as a direct result of motivational commitment to a given position. As support, they showed that the evaluation bias decreased when people considered how they would evaluate the capital punishment studies had their results come out the opposite way. This cognitive manipulation effectively counteracted the evaluation bias. The similarity of this “consider-the-opposite” manipulation to Anderson's (1982) counter-explanation technique is suggestive. Perhaps as people considered how a study could produce opposite results, they spontaneously generated causal explanations in support of each result, producing an effect similar to that which would occur if the evidence had been causal in the first place. If this analysis is correct, causal arguments should be less subject to negative evaluation by opponents than noncausal arguments, representing another significant process by which causal arguments are more effective than noncausal arguments.

Overview

In summary, we propose two processes by which causal arguments may be more effective than noncausal arguments in changing beliefs, leading to the acceptance of new information. Causal arguments may act directly on the relative availability of explanations, shifting the balance of available explanations in favor of the new belief and thus producing belief change. In addition, causal arguments may counteract whatever cognitive processes result in the biased evaluation of noncausal evidence.

We investigated these processes in two experiments. In each experiment we presented to the participants the public health education message that AIDS is not transmitted by casual contact (including nonsexual household contact or contact through mosquitoes), as some people have feared. Evidence substantiating this message was either causal (biological reasons why AIDS is not spread this way) or noncausal (statistical epidemiological patterns that show AIDS is not spread this way). Participants evaluated this evidence, indicated how available explanations were for them to understand why AIDS is not spread this way, and indicated their resultant beliefs about this issue. The second experiment incorporated a longer time interval separating the educational message and belief measurements and included a measure of behavioral commitment.

Experiment 1

We assessed in this study pre- and postcommunication beliefs in a 2 × 2 factorial design, the independent variables being the presence or absence of causal arguments and presence or absence of noncausal arguments. After indicating initial beliefs about the spread of AIDS, participants were randomly assigned to one of four conditions. In three conditions we presented a message asserting that AIDS is not spread through casual contact and substantiated this message with causal, noncausal, or both types of evidence. The fourth (control) condition involved an unrelated task. Participants evaluated evidence they read and indicated their beliefs once again. Finally, participants indicated the availability of reasons to explain why AIDS is not spread by casual contact.

Method

Participants

Of 167 adult participants, 144 (69 males and 75 females) completed all items and were included in the analyses. To obtain sufficient numbers of participants, we recruited at a large public university (n = 108, participating for class credit) and at a large protestant church (n = 36, participating without compensation), both in Houston, Texas. Participants were assured of anonymity. University participants signed consent forms; church participants consented by volunteering. Sessions at the university attracted from 1 to 20 participants; participants from the church were given booklets to work on at their own convenience, which were returned within 2 weeks. All four conditions were randomly assigned at each recruitment location and within each experimental session. All analyses were conducted on the total sample.

Procedure

Materials. All materials were in booklets (titled Beliefs about AIDS). Participants worked at their own pace. The three message conditions differed only in the type of evidence presented. The causal condition (n = 38) explained why AIDS is not spread by casual contact, the noncausal condition (n = 35) conveyed statistical arguments to show that AIDS is not spread by casual contact, and the composite condition (n = 35) included both kinds of arguments. The control condition (n = 36) did not include any message about AIDS but involved participants with an unrelated task of similar length (a self-monitoring scale and an attributional
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Participants provided demographic information before proceeding. We also measured attitudes toward gay men as an individual difference variable that can be expected to affect participants' responses in this belief domain (Herek, 1989; Herek & Glunt, 1988). Participants responded on 9-point scales, 1 (strongly disagree) to 9 (strongly agree), to the 10 items of the Attitudes Toward Gay Men Scale (ATG; Herek, 1987), with internal consistency (alpha) of .93, and higher scores indicating more negative attitudes.

Belief scale. Participants were instructed as follows:

Now that you have given us some general information about yourself and your beliefs, we are interested in exploring some of your beliefs about Acquired Immune Deficiency Syndrome (AIDS). AIDS is a disease caused by a virus that attacks and destroys the body's immune system. Persons with AIDS are then likely to come down with a variety of life-threatening illnesses.

The AIDS virus was identified in 1983. Since that time, much research has looked at how this virus can be spread from one person to another. On the next three pages, we will ask you some questions about how you believe this virus might be spread and about how you might act toward a person carrying this virus. Please answer these questions according to your personal opinion, based on your own beliefs at this time.

Using 9-point response scales, 1 (strongly disagree) to 9 (strongly agree) participants responded to 48 items constituting the belief scale, assessing beliefs about the spread of AIDS through casual contact (examples of items included "A person can safely work beside a person with AIDS," "I would not be afraid to hug a person with AIDS," "It is not safe to use a toilet after a person with AIDS" [reverse scored], "LIVING in the same room with a person with AIDS is dangerous" [reverse scored], and "AIDS can be spread by mosquitoes in swampy areas" [reverse scored]). Two random orders were used. Item responses were averaged to yield belief scores in the range of 1 to 9 (a procedure used for all scales in this study). Higher scores indicated more message-congruent beliefs. The belief scale had an internal consistency (alpha) of .98.

Communications. After indicating their initial beliefs, participants (except in the control condition) read a communication conveying the message that AIDS is not spread by casual contact. The following instructions introduced the reading-learning assignment:

Now we would like you to read some information related to the spread of AIDS. For this study, we collected a number of newspaper articles that have appeared in recent months. One of those articles is on the next page. Different people in this study will be reading different articles, chosen at random from those that we collected. Please read the article on the next page carefully and try to understand the major points. You will not be asked to remember specific details from the article, but we will be interested in how much you learn about the main ideas presented.

The "articles" were fabricated to convey causal, noncausal, or both, arguments gleaned from actual newspaper articles and from public health sources. (For ethical reasons, it was important that substantive information included in all of the articles be true and accurate. Given the research findings on belief perseverance, inaccurate information could have a lasting negative impact on participants.) They were written in parallel form at a high school reading level, with the causal and noncausal articles equivalent in length, and the composite article was about 50% longer.

Both causal and noncausal articles stated that "there are only three ways the AIDS virus is transmitted in the United States—through blood contamination, sexual contact and birth to an infected mother." The articles differed, however, in the nature of the arguments given as reasons why one should believe that AIDS is not transmitted by casual person-to-person contact or by insects such as mosquitoes.

In the noncausal article, an "epidemiologist with the Centers for Disease Control in Atlanta" reports on

A series of studies looking at the interactions between AIDS patients and their families and close friends to see whether AIDS is passed on through casual person-to-person contacts. Statistics revealed clear evidence that such casual contacts do not play a part in the spread of this disease.

Such statistics cited in the article include the fact that "none of the identified cases of AIDS in the United States have been actually proven to have been transmitted in schools, day care centers, or through casual person-to-person contact" and

A study of more than 100 people in families where there was a person with AIDS without the knowledge of the family and in which normal family interactions such as hugging, kissing, eating together, sleeping together, etc., took place revealed not a single case of AIDS transmission.

Regarding transmission by insects, the article argues that "if mosquitoes were spreading AIDS, we would expect school-age children, who are frequently outdoors and are subject to mosquito bites, to be getting the disease. This hasn't happened." Also cited are studies from Haiti and Central Africa revealing that

AIDS is largely a disease of sexually active young adults living in the cities. The disease is much less common in rural areas. In Zaire, the AIDS virus has been found in only 0.8% of healthy people in rural villages, where mosquitoes would be most likely to be spreading the disease. In contrast, studies done in cities have found the virus in more than 27% of the sexually active young adults. This pattern is quite different from malaria . . . a disease known to be carried by mosquitoes.

Note that these arguments support the article's conclusions with direct evidence related to covariation of contact and disease transmission, without suggesting any mechanisms to account for the level of covariation observed.

In the causal article, the same epidemiologist with the Centers for Disease Control in Atlanta reports on

A series of studies looking at the interactions between AIDS patients and their families and close friends to see why AIDS is not passed on through these contacts. Case studies revealed clear reasons why such casual contacts do not play a part in the spread of this disease.

Causal arguments cited in this article include the fact that

The AIDS virus is not concentrated in saliva . . . The virus has to be present in high concentration to infect another person, and even then, it must get into that person's bloodstream . . . This virus is very fragile . . . [Stomach juices would kill the virus . . . ] Workout partners did not get the virus because it was not present in sweat.

Regarding transmission by insects, the article argues that
this particular virus does not reproduce inside mosquitoes, bedbugs, or any other insect that we've found . . . [It] may remain for a short time in the blood in the mosquito's stomach, but it can't be given to another person since it's not in the mosquito's saliva . . . A mosquito's mouth is too small to carry enough virus to infect anyone . . . Mosquitoes rarely move from person to person in the middle of a meal, and between feedings, the virus quickly dies.

Note that in this communication, the arguments do suggest the mechanisms that could account for the conclusions reached in the article, but they do not provide the kind of covariation evidence set forth in the noncausal article.

One anonymous reviewer correctly pointed out that causal arguments invariably contain statistical information. It is important to note that such statistical information does not fall under our definition of noncausal arguments, and so causal arguments and noncausal arguments remain conceptually distinct entities. We previously defined a noncausal argument as one that supports a theory “written to evidence that the relationship expressed in the theory has been observed in the covariation of the variables that comprise the theory.” The arguments in our noncausal communication fit this definition, citing evidence directly relating to whether there is covariation between casual contact and AIDS cases. On the other hand, a causal argument such as “stomach juices would kill the virus” also conveys covariation information (being essentially a statement that the presence of stomach acid is associated with the absence of live virus), but it is information on the covariation of “stomach juices” and “live viruses” and not on the covariation of “casual contact” and “AIDS cases.” (Why people perceive the causal argument as informative with regard to the latter covariation, and may change their beliefs about that covariation in response, is the key issue in this article.) Thus, this information does not fit our definition of a noncausal argument. Ahn et al. (1995) noted that mechanism-based explanations for an event convey covariation information at a different “level of abstraction” from the event itself. That is evident in this example. Therefore, it would be a mistake to say that causal arguments “contain” noncausal arguments, as we use the terms here, or that the strength of statistical information in the causal and noncausal arguments can meaningfully be compared—the statistical information in each refers to a different set of variables.

Evaluation of evidence. Participants evaluated the information that they had read. Separate questions referred to studies concerned with (a) casual contact between people or (b) with the threat of mosquitoes. Responses were made on 9-point scales. Questions asked how well the studies were conducted, 1 (very poorly done) to 9 (very well done), how convincing the evidence was for the conclusions given, 1 (completely unconvincing) to 9 (completely convincing), how satisfied the participant was with the evidence presented, 1 (completely unsatisfied) to 9 (completely satisfied), and whether the evidence gathered had been appropriate, 1 (not at all appropriate) to 9 (very appropriate). These 8 items formed the evaluation scale, with an internal consistency (alpha) coefficient of .95. Higher scores indicated more positive evaluations. Participants in the control condition did not complete the evaluation scale.

Our design and use of the evaluation scale did not presume that there was enough information available to make a definitive judgment of quality of the studies. People must frequently make judgments on the basis of incomplete information. Our participants apparently had enough information to form an opinion about the studies, and given the high value of alpha, it seems they tended to make global judgments evaluating the evidence. (No participants mentioned difficulty in performing these judgments as a problem during debriefing.) The specific questions on the evaluation scale were patterned after those used by Lord et al. (1979).

Postcommunication beliefs. Immediately following the evaluation scale, the belief scale was readministered. Participants were reminded that they had answered these questions earlier, but they were not to go back and look at their previous answers. They were also instructed,

Although you may want to consider the information we gave you in the article, we are not asking you to recall the beliefs or conclusions stated there. Rather, we are asking that you answer these questions according to your own personal opinion based on your own beliefs at this time.

Participants in the control condition answered the questions again, but the instructions did not mention any article.

Explanation availability. Two measures of explanation availability were included in this experiment. In the first measure, participants were asked to think of reasons why AIDS is not spread by casual contact or mosquitoes and to “write down as many of those reasons as come to mind.” The number of reasons produced by each participant was coded into a variable labeled reasons by a rater who was unaware of the experimental condition of each participant.

Subjective ease of generation is also an important indicator of explanation availability (Nisbett & Ross, 1980, p. 23). After the listing task, participants rated the ease with which explanations could be brought to mind to support the belief that AIDS is not spread by casual contact. Two questions (“How easy is it for you to think of reasons why AIDS is not spread by [casual contact/ mosquitoes]”) were answered on 9-point rating scales, 1 (very difficult) to 9 (very easy). These items formed the explanation availability (EXP) scale, with a coefficient alpha of .82.

Debriefing. All participants were debriefed on the purposes of the experiment and the nature of the articles they had read. For ethical reasons, it was stressed to participants that although the articles had been prepared especially for the study and some details (such as researchers' names) had been fabricated, the studies presented were based on actual scientific research, and the conclusions presented reflected the best scientific knowledge available at the time.

Results and Discussion

Initial-belief scores were subtracted from final-belief scores to yield a belief change score for each respondent. (Thus, positive-change scores are message congruent and indicate learning and accepting new information). Comparing the mean belief change scores for various experimental conditions indicated the relative effectiveness of causal and noncausal arguments in this study. Cell means for belief change scores are presented in Figure 1.

Effectiveness of Arguments

Overall effectiveness. For belief change scores, the 2 X 2 analysis of variance (ANOVA; Type 3 sum of squares) yielded a highly significant main effect of causal evidence. $F(1, 140) = 41.10, p < .0001, MSE = 0.56$, a nonsignificant main effect of noncausal evidence, $F(1, 140) = 2.76, p > .05$, and a significant interaction, $F(1, 140) = 7.89, p < .01$. The interaction reflected the absence of a significant effect of noncausal evidence in the presence of causal
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0.6 Noncausal Evidence

0.8 Not Presented

III Presented

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::Q 0.4 0.2 0.0 0.2 0.2

Not Presented

Presented

Causal Evidence

Figure 1. Mean belief-change scores as a function of the presence or absence of causal and noncausal arguments.

evidence, \( t(71) = -0.64 \), along with the presence of an effect in the absence of causal evidence, \( t(69) = 5.22, p < .0001 \). The effect of causal evidence was significant in the presence or absence of noncausal information, \( t(68) = 2.52, p < .02 \), and \( t(72) = 6.60, p < .0001 \), respectively. Direct comparison of the causal-only and noncausal-only conditions showed significantly greater belief change in the causal condition, \( t(71) = 3.25, p < .002 \). This overall pattern of change supports our expectation that causal arguments would be more effective than noncausal arguments, although the results also show that belief change was significantly greater in the noncausal-only condition, relative to the control condition.

Effectiveness on skeptics. It is important to consider the effectiveness of causal and noncausal arguments in teaching new information to those people who initially have the most discrepant beliefs. To examine this, we repeated the ANOVA on belief change scores using only the third of the participants (\( n = 48 \)) with the lowest (most message-discrepant) initial-belief scores. Mirroring the overall results, the model yielded a significant main effect of causal evidence, \( F(1, 44) = 25.36, p < .0001, MSE = 0.76 \) (unweighted marginal means, \( M = 0.11 \) and \( M = 1.40 \)), no main effect of noncausal evidence, and a significant interaction, \( F(1, 44) = 5.68, p < .05 \). Once again, change was greater in the causal-only condition (\( M = 1.74, n = 12 \)) than in the noncausal-only condition (\( M = 0.38, n = 11 \)), \( t(21) = 3.83, p < .001 \), showing causal evidence to be more effective than noncausal evidence among those with the most message-discrepant initial beliefs.

The impact of causal arguments was clear as we observed that 21% of the participants in the causal condition initially scored at or below the midpoint of the belief scale, but only 8% scored in this range after reading the causal evidence. In contrast, 26% of people in the noncausal condition fell in the lower half of the scale initially, and there was no change in this percentage for final beliefs.

Process Analyses

Causal evidence and explanation availability. To examine the hypothesis that causal evidence affected beliefs by first affecting explanation availability, we conducted three additional analyses on both measures of explanation availability. First, the zero-order correlation between EXP and final beliefs was positive, \( r(142) = .52, p < .0001 \). Second, the \( 2 \times 2 \) ANOVA, with EXP as the dependent variable, yielded a significant main effect of causal evidence, \( F(1, 140) = 20.03, p < .0001, MSE = 6.20 \), with no other significant effects (\( ps > .30 \)). Mean EXP scores are shown in Figure 2.

Third, a mediation regression analysis, with final beliefs as the dependent variable and EXP as the mediator, confirmed the mediation prediction. When EXP was not in the statistical model, the causal evidence manipulation had a significant impact on final beliefs, \( F(1, 142) = 5.32, p < .03, MSE = 2.95 \). However, partialling out EXP effects reduced the causal evidence effect on final beliefs to non-significance, \( F(1, 141) < 1 \). Interestingly, the effect of EXP on final beliefs was significant even when the effect of the causal evidence manipulation was partialed out, \( F(1, 141) = 45.29, p < .0001, MSE = 2.25 \).

Parallel analyses on the reasons measure of explanation availability produced parallel findings. First, the zero-order correlation between reasons and final-belief scores was positive, \( r(142) = .43, p < .0001 \). Second, the \( 2 \times 2 \) ANOVA, with reasons as the dependent variable, yielded a significant main effect of causal evidence, \( F(1, 140) = 24.39, p < .0001, MSE = 4.64 \), with no other significant effects (\( ps > .25 \)). Mean reasons scores essentially mirrored those for EXP (shown in Figure 2) for the control, noncausal, causal, and composite conditions, being 1.33, 0.77, 2.71, and 2.94, respectively.

Third, the mediation regression analysis, with final beliefs
as the dependent variable and reasons as the mediator, also mirrored the EXP results. As noted with the EXP analysis, the causal evidence manipulation had a significant impact on final beliefs when no mediator was in the model, $F(1, 142) = 5.32, p < .03, MSE = 2.95$. However, partialling out the reasons effect reduced the causal evidence effect on final beliefs to nonsignificance, $F(1, 141) < 1$. As with EXP, the effect of reasons on final beliefs was significant even when the effect of the causal evidence manipulation was partialled out, $F(1, 141) = 25.91, p < .0001, MSE = 2.51$. Thus, although the reasons and EXP measures operationalized explanation availability in very different ways, the regression findings for both are consistent with each other and with our proposal that causal evidence operates on beliefs through its effect on explanation availability.

Path analyses. To get a clearer picture of the mediating role of explanation availability, as operationalized by both EXP and reasons measures, we conducted two path analyses on participants’ final beliefs. The models we predicted, along with the results of each path analysis, are presented in Figures 3 and 4, in which EXP and reasons as the measure of explanation availability were used, respectively. Two factors were expected to affect explanation availability for each participant: an external factor (the information presented) and an internal factor (elaborations by the participant in response to the information). Of primary importance to this research was the prediction that the causal persuasive communication (the external factor) would lead to more accurate final beliefs by the mediating variable of explanation availability. However, we also had to take into account the internal factor. On the basis of Herek and Glunt’s (1988) findings connecting attitudes toward gay men to beliefs about AIDS, we expected a direct effect of ATG on final beliefs and we also expected ATG to influence the participants’ elaborations, causing ATG to have a direct effect on explanation availability as well.

As can be seen in both Figures 3 and 4, the paths predicted to be significant did yield significant path coefficients. The causal evidence communication (coded as follows: causal = 1 and not causal = 0) directly influenced explanation availability ($p < .0001$ for EXP, $p < .001$ for reasons). Those exposed to the causal communication held more accurate final beliefs about the transmissibility of AIDS by mosquitoes and casual contact than did those not exposed to the causal communication. As expected, there was no direct effect of the communication manipulation on final beliefs in either model, once the indirect (mediated) path via explanation availability was in the model.

Prior attitudes toward gay men yielded significant direct negative effects on both explanation availability ($p < .01$ for EXP and reasons) and final beliefs ($p < .0001$ with EXP in the model, $p < .001$ with reasons in the model). In other words, people with negative attitudes toward gay men found it relatively difficult to think of explanations for why AIDS is not transmitted by mosquitoes or casual contact. Even when this explanation availability effect was controlled statistically, those with prior negative attitudes still held less accurate final beliefs about the transmissibility of AIDS by mosquitoes or casual contact.

Overall, the models displayed in Figures 3 and 4 fit the data quite well. $Q = .997$ with EXP in the model and $Q = .992$ with reasons in the model. (It should be emphasized, of course, that these models are conceptually the same, only with different measures operationalizing explanation availability.) The pattern of interrelations among these variables did not depart significantly from the predicted pattern, $W(1) = 0.36, p > .5$ for EXP, and $W(1) = 1.15, p > .25$ for reasons. Pedhazur (1982, pp. 619–623) discussed the $Q$ and $W$ statistics. Briefly, $Q$ is a measure of the proportion of variance in a fully recursive model (i.e., with all the paths included) that is accounted for by the target model. A $Q$ of 1.0 would indicate that dropping a path (in our case, the direct effect from causal evidence to final beliefs) produced no drop in explained variance. Our $Q$ values are very close to 1.0, indicating that our mediation models fit the data quite well. $W$ is distributed approximately as a chi-square variable and is a test of fit. If the hypothesized model fits well, the $W$ will not be significantly different from chance. According to this measure, our models, with either EXP or reasons as the mediating variable, fit quite well.

Causal evidence and biased evaluation. We have also proposed that causal arguments should be more effective because cognitive biases lead people to discount noncausal evidence discrepant with their beliefs. We found evidence of a cognitive basis for evaluation bias in the current experiment as we regressed evaluation scores on initial-belief scores, $F(1, 106) = 48.00, p < .0001$, and on ATG scores, $F(1, 106) = 15.59, p < .0001$. Participants with message-incongruent initial beliefs and negative attitudes toward gay men tended to evaluate evidence in this belief domain more negatively. Consistent with Lord et al.’s (1984) cognitive formulation of evaluation bias, initial beliefs predicted evaluation scores better than did the attitude measure. In fact, ATG accounted for no unique variance in evaluation scores.
aside from variance shared with the initial belief scores \((F < 1)\). In contrast, belief scores accounted for unique variance when in combination with ATG, \(F(1, 105) = 29.14, p < .0001\). This is consistent with a model in which attitudes affect initial beliefs (in this case, negative attitudes toward gay men were strongly associated with low initial-belief scores, \(F(1, 142) = 73.72, p < .0001\)), but then those beliefs directly affected evaluations through cognitive processes.

Furthermore, we found evidence that people discount noncausal evidence discrepant with their beliefs. We classified participants as having message-congruent or message-incongruent initial beliefs (scoring above or below the midpoint of the belief scale). Using evaluation scores as our dependent variable, we conducted a \(2 \times 2\) (Congruent-Incongruent × Causal-Noncausal) SAS Type 3 sum-of-squares ANOVA that revealed a significant interaction, \(F(1, 69) = 4.65, p < .05\). In the noncausal condition, evaluation scores differed for participants with congruent \((M = 6.90, n = 26)\) and incongruent \((M = 4.79, n = 9)\) initial beliefs, \(t(33) = 4.21, p < .0002\). Participants with message-incongruent initial beliefs evaluated noncausal evidence more negatively. In contrast, causal evidence was not subject to this bias. In the causal condition, evaluation scores did not differ for participants with congruent \((M = 7.30, n = 30)\) and incongruent \((M = 6.81, n = 8)\) initial beliefs, \(t(36) = 0.88\). This pattern of means shows that the causal evidence was not discounted by those with incongruent beliefs the way that the noncausal evidence was, which is consistent with our prediction that causal evidence would be less subject to evaluation bias.

**Limitations**

The expected effects of causal arguments were generally confirmed by the results of Experiment 1. However, there were several aspects of the experiment that limit the applicability of those results to the more general educational context. First, the presence of a pretest for relevant beliefs may have affected the processing of the communications, possibly sensitizing participants to issues that they might otherwise have overlooked. Second, this experiment assessed final beliefs within minutes of exposure to the information and provided no information on the duration of belief changes. Third, this experiment was conducted primarily in closely monitored small groups, a factor that may have increased participants' attention to materials that would have been ignored under more anonymous or natural conditions. Fourth, the control condition in Experiment 1 differed from other conditions in two ways—providing no arguments related to AIDS transmission, but also stating no conclusions on the topic—an approach that could have produced confounded results. Finally, Experiment 1 gave no indication whether the communications produced any commitment to changed behaviors.

In Experiment 1, there was also an indication that noncausal arguments were somewhat effective in changing beliefs. Because this finding ran counter to our expectations, we wanted to see whether this result would replicate. To investigate all of these issues further, we conducted a second experiment.

**Experiment 2**

In the second experiment we utilized essentially the same design as in Experiment 1. Differences included the absence of an initial-belief measure, exposure to materials in an extremely large and anonymous group session, and the inclusion of a measure of behavioral commitment. Also, the control condition was changed to include a communication asserting the message about AIDS transmission, but without support of causal or statistical evidence. Most important, beliefs were measured in a separate session 3 weeks after participants read the communications.

**Method**

**Participants**

Participants from the introductory psychology class at a large midwestern university participated for class credit. Both sessions of the experiment were conducted during class meetings. Altogether, 307 students participated in Session 1, and 272 participated in Session 2, with 216 people attending both sessions. Because of missing data or other participant errors, the numbers in the subsamples for various analyses varied somewhat.

**Procedure**

**Session 1.** Materials for each session of the experiment were contained in separate booklets. The independent variables were manipulated in Session 1. Participants received a booklet titled *AIDS Information Update* that explained that we were interested in "your personal reaction to information about AIDS." Initial questions included the ATG scale (using 5-point response scales), but there were no questions that assessed beliefs about the transmission of AIDS. As in Experiment 1, "newspaper articles" manipulated the presence or absence of causal or noncausal information in a \(2 \times 2\) factorial design. Within the causal, noncausal, and composite conditions we used the same types of communications as in Experiment 1. The control condition was modified in Experiment 2 to include a communication stating the message that AIDS is not spread by casual contact or mosquitoes, but without the citation of either causal or noncausal evidence in support. Participants were randomly assigned to the experimental conditions. From the participants' perspective, the experiment ended with completion of the evaluation scale. Ethical considerations impelled us to provide an oral debriefing to explain that the articles were based on fact but were themselves fabrications.

**Session 2.** Session 2 occurred 3 weeks after Session 1. Participants completed the ATG scale, a 25-item version of the belief scale (incorporating items with the highest item total correlations), and the EXP scale as part of a "Social Issues Survey." (The reasons measure of explanation availability was dropped from Experiment 2 because it had yielded results similar to those of EXP in Experiment 1.) Responses were made on 9-point agree--disagree scales. Finally, an addendum to the survey introduced a measure of behavioral commitment. It was explained that a local AIDS charity needed volunteers and that participants could express interest by responding on the survey. One item asked for volunteers to help
with phone calls, literature distribution, and campus fundraisers. A second item asked for volunteers to actually work with people with AIDS. Participants could make one of four responses to each item with responses ranging from 1 (signifying definite interest), 2 (probable interest), 3 (possible interest), to 4 (no interest).

Every effort was made to dissociate the second session from the first, including the presence of different experimenters for the two sessions. No mention of the earlier session was made during Session 2 (although the questions making up the ATG scale were administered in both sessions) until the presentation of a written debriefing that explained the true nature of the entire experiment. Also during the debriefing, participants were informed that the volunteer opportunity had been an experimental measure and that the organization mentioned was fictitious. However, they were then given the name and phone number of an actual local AIDS services agency, with the suggestion that they call that number if they wished to volunteer their services. Participants were identified by the information they gave to receive credit for participation, allowing us to know in which condition each student had been.

Results and Discussion

In the absence of initial-belief measures, analyses for this experiment focused on the final beliefs assessed in the second session. Other dependent measures included EXP and a Behavioral Commitment score devised from the two behavior-related items. As it turned out, most participants indicated no interest in the volunteer work outlined, so the original 4-point scales were dichotomized and coded as follows: 1 (some interest) and 0 (no interest) for each activity. Summing the items formed a 3-point scale with responses ranging from 0 (no willingness to engage in behaviors) to 2 (indicating some willingness to engage in both behaviors).

Not all participants in Session 2 were present for the first session. This was actually fortunate, in that it allowed us to compare directly participants (n = 56) attending only Session 2 (who received no AIDS-related communication, making them comparable in some ways to the control condition in Experiment 1) with participants (n = 54) in the control condition (who received the communication stating only conclusions without supporting evidence of any kind). Both groups completed dependent measures in the absence of either causal or noncausal evidence. These groups did not differ on any of the dependent measures (all ps < .25), demonstrating that the message alone, as presented in the control condition, was ineffective in influencing beliefs. This result also allowed us to treat both groups of participants as controls, thus making use of all the data from participants who responded to dependent measures in the absence of causal or noncausal arguments. Because the analyses now included two control groups, all main and interaction effects from the basic 2 x 2 design (causal arguments present or not by noncausal arguments present or not) were assessed by contrast analyses in which equal weights were assigned to the two control groups (see Winer, 1962).

Effectiveness of Arguments

As expected, the 2 x 2 ANOVA on the belief scores yielded a significant main effect for causal evidence, F(1, 258) = 4.73, p < .05, MSE = 2.98. Even after the 3-week delay, beliefs were more message congruent in the presence of causal evidence (M = 6.60) than in the absence of causal evidence (M = 6.13). Neither the main effect of the noncausal information nor the interaction was significant (both Fs < 1).

Additional results again supported the hypothesis that explanation availability mediates the effect of causal communication on beliefs. First, the zero-order correlation between EXP and final-belief scores was positive, r(259) = .69, p < .0001. Second, the main effect of the causal evidence manipulation on the EXP measure of availability was quite strong, F(1, 263) = 9.97, p < .002, MSE = 4.46. Those who had seen the causal evidence had higher EXP scores than those who had not (Ms = 6.41 and 5.57, respectively). The main effect of noncausal evidence and the interaction of causal and noncausal evidence were both nonsignificant, Fs(1, 263) = 2.11 and .04, respectively, ps > .14.

Finally, the mediation regression analysis on data from participants who had nonmissing scores on the final–belief and EXP measures confirmed the mediation prediction. When EXP was not in the statistical model, the causal evidence manipulation had a significant impact on final beliefs, F(1, 256) = 4.60, p < .04, MSE = 2.96. However, partiaiting out EXP effects reduced the causal evidence effect on final beliefs to nonsignificance, F(1, 255) < 1. Interestingly, and as in Experiment 1, the effect of EXP on final beliefs was significant, even when the effect of the causal evidence manipulation was partialed out, F(1, 255) = 228, p < .0001, MSE = 1.57.

The behavioral commitment measure further demonstrated the effectiveness of causal arguments. The correlation between EXP and behavioral commitment was positive, r(255) = .23, p < .001. Behavioral commitment scores yielded a significant effect of causal evidence, F(1, 251) = 4.37, p < .05, MSE = 696, and no other effects (ps > .25). In the presence of causal evidence, behavioral commitment was higher (M = .68) than in its absence (M = .46), though in both cases, the level of commitment was quite low (below the scale midpoint of 1.0). Thus, it appears that causal arguments not only affect subsequent beliefs but also have some effect on willingness to act on those beliefs.

Path Analysis

The path analysis model we predicted for this study is similar to that presented for Experiment 1, but with the addition of the behavioral commitment variable. Figure 5 presents the model and the results of the path analysis. Once again, all of the paths that we expected to be significant did yield significant path coefficients; whereas those expected to be small or nonsignificant yielded nonsignificant path coefficients. Of primary interest to this article was the
finding that the experimental manipulation of causal persuasive evidence yielded a significant direct effect on explanation availability ($p < .02$), with those exposed to the causal communication having higher explanation availability scores than those who had not received the causal communication. Also as expected, the direct effects of the causal evidence manipulation on final beliefs and on behavioral commitment were nonsignificant, supporting our theoretical view that causal persuasion effects on beliefs and on behavior are mediated by explanation availability.

As in Experiment 1, this study also showed strong effects of prior attitudes toward gay men on all three downstream variables. People with more negative prior attitudes toward gay men scored lower on the explanation availability measure ($p < .0001$), indicating that they found it relatively difficult to think of explanations for why AIDS is not spread by mosquitoes or casual contact. The direct effect of ATG on final beliefs ($p < .0001$) was of a similar nature, with those holding negative attitudes having less accurate beliefs about the transmissibility of AIDS by mosquitoes or casual contact, even when explanation availability was statistically controlled. The direct effect of ATG on behavioral commitment ($p < .005$) revealed that those with negative attitudes toward gay men were less likely to commit to work on AIDS-related projects, even when final beliefs and explanation availability were held constant statistically.

It is also important to note that the path analysis showed the direct link between explanation availability and behavioral commitment to be nonsignificant. As expected, the effects of explanation availability were largely mediated by final beliefs. Finally, the predicted causal model in Figure 5 fit the obtained data quite well, $Q = .986$. The pattern of interrelations among these variables did not depart significantly from the predicted pattern, $W(3) = 3.45$, $p > .3$.

In summary, Experiment 2 replicated the major findings of Experiment 1. The dependent variables responded as expected to the presence of causal information, despite the fact that the information was presented 3 weeks earlier in the context of a seemingly unrelated experiment. Causal evidence not only had an impact on subsequent beliefs but also affected commitment to act on those beliefs. Finally, a path analysis for data from Experiment 2 supported our proposed model that the impact of causal evidence on beliefs occurs through the mediation of explanation availability.

**General Discussion**

Results of the current experiments provide good support for our hypotheses. When causal arguments were presented, beliefs changed by an increase in the availability of explanations. Noncausal evidence was discounted by those with discrepant beliefs and did not effectively increase explanation availability to produce belief change.

**Causal and Noncausal Arguments**

Why should a person believe a particular social theory, accepting that information as true? We have proposed that two types of arguments logically bolster this belief: one type addressing whether the relationship embodied in the theory is true (the noncausal arguments), and one type addressing why the relationship is true (the causal arguments). (Imagine teaching the fact that “The sun always rises in the East.” This assertion could be supported by pointing out that the sun has always risen in the East each morning, a noncausal argument, or it could be supported with an explanation of the movements of our planet with respect to the sun, a causal argument. Both types of argument should logically help to transform what is initially simply an assertion into a fact accepted and, hence, learned.) In distinguishing between causal and noncausal arguments, we want to stress that both types of arguments offer valid reasons to believe that a theory is true, but only causal arguments offer reasons why the theory is true (i.e., explanations). Thus, our point is that when choosing from the set of arguments that could be made in favor of believing a particular theory, a set that typically would include both causal and noncausal arguments, our results suggest that it is more effective to select and utilize the causal arguments. Assuming they are relevant, plausible, and understandable, causal arguments constitute stronger arguments because they affect the balance of available explanations and because they are less likely to be discounted.

The form of causal arguments may vary from logical reasoning to causal scenarios (series of causally related events enabling the occurrence of the event to be explained) or may combine features of both, as did the causal arguments used here. For example, the argument that “AIDS cannot be spread on food because the virus is destroyed by stomach acids” has elements of both logical reasoning and a causal scenario.
Applications

Although this study involved a single belief domain within the context of AIDS education, we expect the results to replicate in any arena in which causal thought is relevant and belief perseverance makes audiences resistant to the acceptance of new information, including formal educational settings. The psychology classroom provides a prime example, where students often remain skeptical of research results that differ from their expectations. Our results reinforce an observation that many instructors will have already made—that an explanation of a concept is often more convincing to students than citing 10 replications showing the concept’s validity.

These results may also help to elucidate recent research on the value of the elaborative interrogation technique for helping students acquire information inconsistent with their prior knowledge. Elaborative interrogation requires students to generate their own explanations to support factual statements (Pressley, Symons, McDaniel, Snyder, & Turnure, 1988). This technique enhances learning, even for information that is contrary to prior knowledge (Woloshyn, Paivio, & Pressley, 1994). This fits well with our model because we propose that explanation availability mediates beliefs, regardless of the internal or external origins of the relevant explanations. In fact, our model may point to a critical mechanism by which the elaborative interrogation technique works. Woloshyn et al. saw this technique as working by enhancing connections with prior supportive knowledge. Our model suggests a different (or perhaps additional) mechanism, in which it is the causal nature of the interrogation (answering why questions) that is important. Other techniques could tap into prior supportive knowledge (e.g., “Think of instances where you have observed __”), without necessarily altering the availability of explanations or changing beliefs. For example, social stereotypes may be challenged by the recollection of counterstereotypical instances, but the stereotypes remain unchanged because such instances are often seen as “exceptions to the rule” (Linville, Salovey, & Fischer, 1986). Elaborative interrogation actually does much more than just heighten attention to prior supportive knowledge; it requires the generation of explanations in support of the target information. By doing so, it may tap into the same processes that make causal arguments effective in the current studies. Our experiments also add credence to concerns of several authors who have noted that science textbooks often do not provide explanations adequate for altering strongly held misconceptions about scientific concepts (Alvermann et al., 1985; Eaton, Anderson, & Smith, 1983).

In the classroom, students’ beliefs about themselves may also resist change (e.g., Lepper, Ross, & Lau, 1986). When such beliefs become maladaptive and self-defeating (for a review, see Slusher & Anderson, 1988), causal arguments may also be useful in efforts to alter these beliefs.

Finally, we note the obvious application of our studies in the arena of AIDS education, in which erroneous beliefs continue to flourish (Herek & Capitanio, 1993). Our results suggest value in making causal arguments in response to these erroneous beliefs.

Future Directions

There are many issues regarding the role of explanation availability in belief change that could not be addressed in the current study and should provide fruitful avenues for future research. The concept of explanation availability itself deserves more study. How do people go about assessing availability when considering their beliefs (and do people readily distinguish explanations from the beliefs themselves)? Future research could examine the particular characteristics of causal information that are effective in changing beliefs. Factors that were constant in these experiments (such as the authority of the communicator, the presence of conclusion statements, etc.) could be varied as moderator variables in future research. It would also be useful to test the generalizability of our findings to different target beliefs. Our target belief about AIDS transmission was a social theory involving a null relationship (i.e., AIDS is not transmitted by casual contact). It will be important for future research to test the generalizability of the current findings to beliefs involving positive relationships (e.g., exposure to violent media increases aggressive behavior). Of course, there may be a difference between medical–scientific and social–behavioral domains as well.

One particularly interesting issue for future research involves the boundary conditions on the effectiveness of causal arguments that are suggested by our belief change model. It is implicit in our model that not just any causal argument should be expected to affect beliefs in a given domain. Rather, it is those arguments that affect the relative availability of explanations in that domain that should be effective. Earlier, we alluded to an assumption that effective causal arguments would have to be plausible, understandable, and relevant to the issue at hand. However, what about arguments that clearly do not fit these criteria? How does a person considering AIDS transmission respond to causal arguments that are irrelevant (e.g., explaining how airplanes fly), implausible (“The virus is as big as a bowling ball”), or unintelligible (perhaps due to extensive scientific jargon)? Our model suggests looking at whether such arguments affect the availability of explanations. If they do not, then we would not expect an effect on beliefs. In this way, our model suggests a variety of comparisons between arguments that could be made in addition to the comparison of causal and noncausal arguments that we have made in this article. For example, it should be possible to vary the relevance of arguments to look for systematic effects on explanation availability and beliefs. One could even assess the impact of arguments based on analogies, which may not address a given social theory directly, but may still affect explanation availability. Such studies would help to map the boundaries on the effectiveness of causal arguments as well as to refine our model further. However, future research may also need to refine the measurement of explanation availability to accomplish these more detailed analyses.
Production tasks (such as the reasons measure in our Experiment 1) will need to be developed further to allow researchers a better view of the content of available explanations.

Some issues could experimentally be addressed in the future by manipulating variables merely assessed here, such as initial beliefs. Unlike belief perseverance studies (e.g., Anderson et al., 1980), which have manipulated weak initial beliefs, one goal in our research was to utilize strong, established initial beliefs. However, those beliefs undoubtedly correlated with a variety of attitude variables (as they did with attitudes toward gay men). Studies could be designed to assign randomly participants to situations that induce very different, yet fairly strong initial beliefs (e.g., assignment to class sections taught from different perspectives or assignment to living units with different group norms). In these situations, initial beliefs would be less confounded with other variables.

One place in which the correlational approach is problematic is in examining mechanisms underlying the biased evaluation of evidence. In discussing potential cognitive or affective foundations for evaluation bias, Lord et al. (1984) could not entirely dismiss motivational processes in such an affect-laden domain of beliefs as capital punishment, although they found the argument for motivational forces less compelling for belief domains in which affective reactions were less likely to occur. Of course, our finding that causal evidence was less subject to bias is consistent with a cognitive interpretation, and in the broader debate over the value of cognitive and motivational constructs (e.g., Anderson & Slusher, 1986), it is consistent with the position that motivational constructs indirectly influence social judgments through cognitive processes. Research manipulating strong initial beliefs could further clarify this issue.

In summary, the experiments presented in this article afford a better understanding of the processes linking belief perseverance and belief change and give researchers further insight into the relation of current knowledge to the acquisition of new information. Whether a belief persists or changes appears to be largely a function of the causal thinking that is associated with the belief. Causal arguments appear to have the ability to shift the balance of available explanations in favor of a communication's message, making them effective in persuading an audience to accept new information.

References


