

## Self and group protection concerns influence attributions but they are not determinants of counterfactual mutation focus

Nyla R. Branscombe\*, Ahogni N'gbala and Diane Kobrynowicz

*Department of Psychology, University of Kansas, Lawrence, KS 66045, USA*

Daniel L. Wann

*Murray State University*

The relative impact of differential motivation and knowledge for both counterfactual mutation focus and attributional processes were examined. Functional views of counterfactual thinking predict that what feature of an event is focused on during mutation is influenced by the perceiver's motivation, and that what is mutated is then perceived as causal of the outcome. Other research, however, has indicated that mutation and attribution are based on distinct processes and that the two are not necessarily correlated. In three experiments we investigated the relationship between target mutation and blame assignment following a negative outcome. As expected, both when a group that one is highly identified with and when the self is involved in a negative event, attributions were biased in favour of the self or the in-group. Mutation did not, however, show either a self- or group-serving bias. These findings support the view that mutation focus stems from a relatively automatic orientation towards features that may be differentially available to perceivers with varying degrees of knowledge about a domain, whereas the attribution of blame results from conscious processes that permit motivational influences to operate.

Kahneman & Miller's (1986) norm theory is aimed at explaining when events will seem surprising and when they will seem as if they could not have turned out otherwise. Abnormal events are those for which alternative outcomes come easily to mind. Events for which no such alternatives are available are perceived as normal, making them unlikely to be mutated by perceivers. For example, a person who is shot in a store not previously visited is more likely to be mutated than is a person shot in a store routinely visited (Miller & McFarland, 1986). Similarly, people who engage in contextually unexpected behaviours are perceived as more abnormal and, as a consequence, are more mutable than those whose actions are perceived as more appropriate for that context (Branscombe, Crosby & Weir, 1993; Branscombe & Weir, 1992; Turley, Sanna & Reiter, 1995). Immoral and controllable actions are particularly likely to be mutated, with the outcomes they produce being perceived as they 'shouldn't have been that way' (Alicke, 1992; Miller &

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Turnbull, 1990; N'gbala & Branscombe, 1995; Roesse & Olson, 1995a). In such cases, a more normal event outcome is highly available (see also Kahneman & Tversky, 1982).

### Determinants of counterfactual construction

According to the theory, norms for an event are recruited *post hoc*, after the event itself has been encoded. Norm theory draws on exemplar memory models (Medin, 1989; Smith & Medin, 1981) to suggest that features of the stimulus itself determine the alternative that is constructed. Kahneman & Miller (1986) theorize that norms 'are constructed on the fly in a backward process that is guided by the characteristics of the evoking stimulus and by the momentary context' (p. 150). Thus, this theoretical view portrays the construction of an alternative to what is known to have occurred as a fairly *automatic or spontaneous cognitive process*.

While communicators can and do direct perceivers' attention towards particular features or targets in an event so that they are more likely to be mutated compared to other features not made salient (Branscombe, Owen, Garstka & Coleman, 1996; Macrae & Milne, 1992; Nario-Redmond & Branscombe, 1996), spontaneous mutation is a function of availability which could differ across persons. Thus, different features of an event may be more salient, and more available for mutation, for people who possess more knowledge about a domain compared to those who lack such familiarity. In the current research we tested this possibility—that what is focused on and mutated is dependent on the knowledge possessed about a domain—as well as another, more motivated or strategic view of mutation. Furthermore, we examined the relationship between mutation and attribution, with the expectation that the two will not be consistently correlated. If, as hypothesized by Kahneman & Miller (1986), mutation focus is due to the relatively automatic detection of abnormal features, and the attribution of blame for an outcome involves more extensive consciously controlled processing (see Shaver, 1985, for the goal-directed nature and the time-dependent ordering of the components of attributional judgments), then mutation and attribution might be unrelated much of the time.

In Kahneman & Miller's (1986) theory, there is *no* hint whatsoever that the counterfactual construction process could be motivationally driven. What is targeted for mutation is hypothesized to be 'rapid, automatic and essentially immune to voluntary control' (Kahneman & Miller, 1986, p. 148). Recently, however, several researchers have suggested a more strategic or motivated view of counterfactual construction (Markman, Gavanski, Sherman & McMullen, 1993; Roesse, 1994; Roesse & Olson, 1995b; Sherman & McConnell, 1995). According to Markman *et al.* (1993), 'a full understanding of counterfactual thinking processes also requires consideration of how they might serve people's motives and goal states' (p. 88). Given that the event can be undone, the *direction* of the counterfactual favoured is expected to be a function of the perceiver's motives—to improve future performance on a task or to protect self-esteem. However, the *same antecedent feature* might be focused on as a means of generating either a better or worse counterfactual alternative. An individual could focus relatively automatically on antecedent 'X' because of that stimulus' properties (see Bargh, 1984), yet consciously choose to entertain either a better or worse outcome with that feature as the focus. Thus, mutation focus and the direction of the counterfactual ultimately constructed are conceptually distinct components that could be affected by different variables.

In their expansion of the motivated view of counterfactual generation, Sherman & McConnell (1995) have argued that *which target is specifically selected for mutation* is motivationally driven. In particular, they speculate that people are motivated to focus on and mutate their own actions (e.g. when other plausible targets besides the self are available), in order to maintain the belief that they control their own outcomes. Because Sherman & McConnell (1995) assume that what people mutate is perceived as causal, they argue that 'the mutation of events that are not actually causal can be dysfunctional' (p. 203). Although this hypothesis—that mutating the self necessarily increases self-blame—was not empirically tested, it is predicated on the assumption that 'the counterfactual mutation of a feature implicates that feature as a primary cause of the outcome' (Sherman & McConnell, 1995, p. 204).

In contrast, we argue that focusing on and mutating the self could be considered dysfunctional, and avoiding mutating the self would be important for well-being, only if there were a one-to-one relationship between mutation and attribution. If, however, mutation is not strongly or even consistently linked with attribution, then what is mutated would not necessarily have implications for the assignment of blame for a negative outcome. We argue that what controls mutation is often different from what determines attributional responses. In fact, we have previously provided evidence that the two processes are answering distinct questions for the perceiver (see N'gbala & Branscombe, 1995). In the case of counterfactual thinking, participants are answering the question 'How could the outcome have been different?' but when it comes to attribution, participants are answering the question 'What specifically brought about this outcome?'

### Determinants of attribution

Wells & Gavanski (1989) were the first to propose that mutating a particular feature of an event influences subsequent attributions. In support of their hypothesis, they found that when it was easy for uninvolved observers to mutate a target and mentally simulate a better outcome, then more causality was assigned to that target compared to when the same mutation did not result in an improved outcome. However, N'gbala & Branscombe (1995), drawing on Copi's (1982) theoretical discussion of the nature of causes, argued and found that what is mutated is not *generally* what people perceive as most causal, responsible or blameworthy. Necessary causes or enabling conditions are typically targeted for mutation, while factors that are sufficient to produce the outcome are overwhelmingly perceived as the cause of the event in terms of attributional responses (see also McGill & Klein, 1993).

For counterfactual thinking to be a general determinant of attributions (see Roese & Olson, 1995b; Sherman & McConnell, 1995; Wells & Gavanski, 1989 for such reasoning), it would indeed have to be affected by motivational factors—attribution is notoriously self-serving (Miller, 1976; Miller, Norman & Wright, 1978; Reiss, Rosenfeld, Melburg & Tedeschi, 1981; Ross, 1977; Taylor & Brown, 1988). In the only empirical investigation thus far to assess whether what is actually mutated might be motivationally driven, little support for the idea was obtained (Burris & Branscombe, 1993). Using a modified version of Wells & Gavanski's (1989) cab driver vignette—where the driver's blame is increased if changing his behaviour would prevent a tragic outcome from happening to a couple whom he refused to transport—adding a motivation to blame the driver did not

alter people's tendency to mutate him rather than other factors. Thus, when high or low racist participants were presented with this vignette, and the race of the cab driver was varied (Black vs. White), level of racism had no effect on mutation focus. Frequency of targeting the driver for mutation was determined by availability (i.e. whether it was easy or difficult to undo the outcome by mutating the driver), regardless of the driver's race. Blame assignment, in contrast, was dependent upon the perceiver's level of racism, with high racists assigning more blame to the Black than the White driver, regardless of what was mutated.

### Overview of the current research

We set out to carefully assess whether mutation focus and attribution are dependent on the same factors and whether they are correlated with each other or not. While it can be difficult to distinguish between motivational and cognitive explanations (Tetlock & Levi, 1982), because knowledge and motivation are frequently correlated, as they are in the domain employed in the current research (see Wann & Branscombe, 1995a), some mutation patterns, if obtained, will be more consistent with one view than another.

In our first study, a motivated view of what is focused on during counterfactual mutation would predict that those high in group identification should be especially likely to avoid mutating the in-group as a whole, when imagining how a bad outcome for the in-group could have been avoided. Instead, when the in-group is a target of mutation, this perspective would predict a greater preference for mutating a specific in-group target, as a means of preserving the overall value of the in-group. Much research from a social identity theory perspective (Branscombe, Wann, Noel & Coleman, 1993; Hewstone, 1989; Marques, Yzerbyt & Leyens, 1988; Tajfel & Turner, 1986) has found that people make group identity supporting evaluations and attributions, even if that means rejecting an undesirable in-group member, and this is particularly the case for highly identified group members. Thus, a pattern of maximal out-group mutations, followed by high levels of specific in-group target mutations would be expected if, as Sherman & McConnell (1995) hypothesize, the target in a sporting event who is mutated most will be also blamed most for the loss. For highly identified fans of a team to avoid negatively evaluating their own group, *and if what is mutated predicts attributions*, they should then be motivated to mutate the out-group more than the in-group and specific in-group targets more than the in-group as a whole.

If, however, mutation focus is primarily dependent on the knowledge possessed about a domain, then the highly identified participants, who do indeed know more about specific in-group members and who think more complexly about the in-group (Wann & Branscombe, 1995a,b), will be capable of mutating specific in-group targets as well as the in-group as a whole. Those who are low in identification cannot easily mutate specific in-group targets because of their lack of knowledge about them (see Wann & Branscombe, 1995a). But, because those who are low in identification also lack a concern with the group, high levels of in-group mutations could be therefore interpreted as due to their lack of motivation to protect the group compared to the highly identified. As a consequence, the mutation pattern of those who are low in identification is less diagnostic of possible motivational versus knowledge influences on mutation focus. Because those who are high in identification *can* do either—mutate the group as a whole or specific individuals—their choices are particularly relevant to the motivational possibility.

Furthermore, predictions derivable from the motivational and knowledge hypotheses differ for the relationship between mutation focus and attribution that can be expected. According to the knowledge-driven hypothesis, mutation and attribution will not be correlated for those high in identification; different factors will determine their responses in the two tasks. Mutation may be related to attribution among those low in identification, however, for these participants will focus on the same features when responding to the two tasks. From a motivated viewpoint, mutation of the out-group and specific in-group targets is a means of preserving the overall positive image of the in-group so such mutations should be especially likely to be correlated with attributions among the highly identified. Such correlations would not be expected among the low identified for they would not be motivated to blame, or avoid blaming, any particular target.

In our last two experiments we operationalize motivation in a rather different way. Here the self and an unknown other are presented as the two potential agents who could have brought about a negative outcome—an auto accident. The motivational hypothesis would predict that an unknown other would be mutated significantly more often than would the self, when both perform the same actions that result in a bad outcome. Certainly self-mutations should be avoided, if self-blame is to be avoided and, if the two processes are linked. Conversely, if knowledge influences what is mutated, the self should be at least as likely to be targeted for mutation as an unknown other. Because we hypothesize that the two processes—mutation and attribution—are based on different mechanisms, regardless of what is mutated, we predict that attributions for the outcome will be biased in a self-protective direction. Participants will not assign comparable blame to the self and the other for the accident, even when they mutate themselves. Only when they mutate the other driver, and there is therefore no motivational inconsistency with self-blame, will mutation and attribution be correlated.

Our question in this work most generally centres around the issue of whether what is mutated predicts or mediates attributional responses. While some research supports such an analysis (Wells & Gavanski, 1989), other studies have found that the two processes—mutation and attribution—are quite distinct (Burris & Branscombe, 1993; N'gbala & Branscombe, 1995). We will argue here that what is mutated is often different from what is perceived as causal of a bad outcome and consequently blamed for it. Specifically, we will provide evidence that mutation focus is *not* especially self-serving, although attributional responses certainly are, and that the two processes are often not correlated, particularly when the mutations generated are not consistent with self or group protective attributional goals.

## EXPERIMENT 1

### Method

#### *Participants and design*

Participants (43 males, 43 females) received partial course credit towards their introductory psychology course requirement. They were pretested several weeks in advance to determine their degree of identification with the University of Kansas men's basketball team, the Jayhawks. Based on scores on this previously validated seven-item measure (Wann & Branscombe, 1993), two groups of participants were contacted to participate in the study. Approximately equal numbers of males and females who were either high ( $N = 43$ ) or

low ( $N = 43$ ) in identification with the team were randomly assigned to read one of two descriptions of a basketball game. The two identification groups selected differed significantly in terms of team identification ( $F(1,84) = 430.52, p < .0001$ ), with the highly identified scoring higher on the measure ( $M = 6.47$ ) than the low identified ( $M = 2.20$ ). The Kansas team was always described as losing the match but the margin was either wide (11 points) or narrow (1 point); theoretically (see Kahneman & Miller, 1986), plausible counterfactuals where the Kansas loss might have been avoided should be more available in the narrow margin case than in the wide margin condition. Thus, the design employed was a 2 (high or low team identification)  $\times$  2 (wide or narrow loss margin) between-subjects factorial design. Participant gender was not significant in any of the analyses; hence, all of the analyses reported here ignore this variable.

### *Procedure and materials*

Participants were first informed that the study concerned how people process information in newspaper articles concerning sporting events. They were told that they would be reading a brief article describing a basketball game and then answering some questions about it. The first section of the article explained that the University of Kansas had lost an important game to a rival team, the Oklahoma Sooners. The remainder of the article consisted of a narrative account of the game highlights and various quotes. Eleven discrete pieces of information about the game were presented. Five pieces of information focused on a Kansas team member's performance: (1) one player's excellent early shooting; (2) the leading scorer's ejection from the game; (3) the benching of the top rebounder; (4) an important scorer's fouling out; and (5) the second leading scorer's poor finish. Three statements concerned an Oklahoma team member's performance: (1) one player's unusually good shooting performance; (2) an injury to one of their best players early in the game; and (3) the addition of two particularly intimidating players late in the game. The three remaining pieces of information in the game commentary concerned both teams or the referees: (1) the replacement of the referee with a novice late in the game; (2) the poor free throw shooting on Kansas' part and the excellent performance on this dimension by Oklahoma; and (3) players on each team believing the fans of the other team had made performing well difficult. The 11 pieces of information were intermixed to create a commentary. At the conclusion of the article, one of two scores was given. In the narrow defeat condition and the wide defeat conditions the final scores were respectively—94-Oklahoma, 93-Kansas and 94-Oklahoma, 83-Kansas.

After reading the game description, participants were asked to 'list the five things that could have been different so that the Kansas loss might have been avoided'. Following this mutation task, respondents were asked to indicate how easy or difficult it was to imagine each of the changes actually occurring, on an eight-point scale (1 = very difficult; 8 = very easy). On the next page, participants were asked to make attributions for the game outcome on 14 items, tapping four dimensions: (1) Oklahoma's good luck caused the outcome (2 items); (2) the Kansas loss was due to some negative feature of Oklahoma—their dirty playing and their fans (4 items); (3) Oklahoma was more skilled than Kansas (5 items); and (4) the Kansas team's poor ability (3 items). All ratings were made on eight-point Likert scales, with strongly disagree (1) and strongly agree (8) as end-points. The final item served as a manipulation check on the wide/narrow defeat margin variable. Participants indicated on a 1 (very small) to 8 (very large) scale 'how large Oklahoma's margin of victory was'.

## **Results**

### *Manipulation check*

A 2 (high or low team identification)  $\times$  2 (wide or narrow loss margin) analysis of variance was performed on the item assessing perceived size of Oklahoma's victory. The margin was seen as very small ( $M = 1.69$ ) in the narrow condition and considerably larger ( $M = 4.41$ ) in the wide defeat condition. This difference was reflected in the significant main effect of the points margin variable ( $F(1,82) = 58.25, p < .0001$ ) and no effect for identification level and no interaction between the two variables.

### *Mutations*

Each of the counterfactuals generated by the participants was coded into one of three

categories by two coders working independently. All mutations that mentioned a specific player or individual on the Kansas team were coded as *in-group individual* counterfactuals (e.g. 'if only Mike Maddox, who is the leading Kansas shooter, hadn't been ejected from the game'). Those counterfactuals that focused on the Kansas team as a whole were classified as *in-group team* counterfactuals (e.g. 'if only Kansas had made their shots from closer to the basket'). The remaining mutations, which involved a focus on the Oklahoma team, were coded as *out-group team* counterfactuals (e.g. 'if only Oklahoma hadn't made all their easy shots'). Interrater agreement was 92 per cent and the few discrepancies that occurred were resolved by discussion.

The mean number of each of the three types of mutations was examined by identification level and the points margin. The three-way interaction was not significant, nor were any of the effects involving the points margin variable. The interaction between level of identification and mutation type was significant ( $F(2,164) = 3.85, p < .02$ ). The highly identified participants were significantly more likely to mutate specific Kansas players ( $M = 1.77$ ) than were those low in identification ( $M = 1.14, F(1,85) = 9.15, p < .003$ ). Conversely, the low identified respondents were more likely to mutate the Kansas team as a whole ( $M = 2.30$ ) than were those high in identification ( $M = 1.77, F(1,85) = 3.47, p < .06$ ). However, the highly identified participants were equally likely to mutate either the Kansas team as a whole ( $M = 1.77$ ) or specific Kansas players ( $M = 1.77, F < 1$ ). The low identified participants were unlikely to mutate a specific Kansas player ( $M = 1.14$ ); as a consequence, their mutations were primarily of the team as a whole ( $M = 2.30, F(1,42) = 12.86, p < .001$ ). The frequency of generating in-group counterfactuals overall (individual and group together) did not differ among the low identified ( $M = 3.44$ ) and the high identified ( $M = 3.54$ ) participants ( $F < 1$ ). Counterfactuals that focused on the out-group team were considerably less frequent and they did not differ for the low ( $M = 1.46$ ) and high identified ( $M = 1.45$ ) groups ( $F < 1$ ).

When the proportion of each of the three types of mutations generated were examined, the same two-way interaction between level of identification and mutation target emerged ( $F(2,81) = 4.43, p < .02$ ). Those high in identification did not differ in terms of the proportions of the three types of mutations generated (35 per cent in-group team, 35 per cent in-group individuals and 30 per cent out-group team,  $F < 1$ ). Those low in identification did generate proportionally more in-group team mutations (47 per cent) than out-group team mutations (30 per cent) and especially compared to mutations involving specific in-group individuals (23 per cent,  $F(2,84) = 8.30, p < .001$ ). Thus, those individuals with a motivation to avoid perceiving the in-group negatively did *not* show a preference for mutations that were protective of the in-group. Only those who lacked the specific knowledge to mutate individual members of the in-group team generated the different types of mutations to differing degrees.

The mean plausibility of the three types of counterfactuals constructed was subjected to a 2 (level of team identification)  $\times$  2 (points margin)  $\times$  3 (counterfactual type) mixed analysis of variance, with repeated measures on the last variable. As expected, the points margin influenced the overall believability of all types of counterfactuals ( $F(1,44) = 3.89, p < .05$ ). In the narrow margin case, mutations were rated as easier to imagine ( $M = 5.69$ ) than in the wide margin case ( $M = 4.93$ ). The significant main effect of mutation type ( $F(2,88) = 4.50, p < .02$ ) indicated that out-group counterfactuals were least believable ( $M = 4.78$ ) compared to the in-group individual mutations ( $M = 5.22$ ) and especially

compared to the in-group team mutations ( $M = 5.76$ ). Hence, the mutations made most frequently by the low identified participants were deemed more plausible than those they generated less frequently (i.e. specific in-group targets). However, for those high in group identification, mutations that they deemed more plausible (i.e. in-group team as a whole) and those they rated less plausible (i.e. specific in-group targets) occurred with equal frequency.

### *Attributions*

Four attributional indices were created assessing: the Oklahoma team's luck as a source of the outcome ( $\alpha = .80$ ), Oklahoma's greater ability as a team as an explanation ( $\alpha = .84$ ), Oklahoma team members' or fans' inappropriate actions caused the Kansas loss ( $\alpha = .73$ ), or the Kansas team's low ability caused the loss ( $\alpha = .62$ ).

Mean responses to the four types of attributional measures were subjected to a 2 (level of identification)  $\times$  2 (points margin) analysis of variance. In all cases, participants were highly self-serving in their attributions for the outcome and this was reflected in the main effect of level of identification. The highly identified were significantly more likely to attribute the Kansas loss to Oklahoma's good luck ( $M = 3.57$ ), which could be seen as temporary, compared to those low in identification ( $M = 2.70$ ,  $F(1,82) = 5.65$ ,  $p < .02$ ). The low identified were more willing to perceive the Oklahoma team as having greater ability than the Kansas team ( $M = 4.44$ ) compared to the highly identified ( $M = 3.53$ ,  $F(1,82) = 8.86$ ,  $p < .004$ ). The highly identified were inclined to agree that specific inappropriate actions by Oklahoma persons caused the poor Kansas outcome ( $M = 4.33$ ), relative to those low in identification ( $M = 3.72$ ,  $F(1,82) = p < .04$ ). Finally, the highly identified were less inclined to attribute the loss to the Kansas team's low ability ( $M = 3.65$ ) than were persons low in identification ( $M = 4.58$ ,  $F(1,82) = 8.51$ ,  $p < .005$ ). The points margin did not significantly influence any of the attributions.

### *Mutation-attribution relationships*

The four attributional indices were correlated with the frequency that participants made the two types of group mutations as well as with their relative proportions, separately for those who were high or low in identification. As can be seen in Table 1, attributions and mutations (for both the absolute number of mutations and the relative proportions) for both teams were significantly correlated among those low in identification. In other words, mutations of, and attributions about, groups are consistently correlated when there is no motivation that might interfere with that link. The frequency of mutating either team as a whole or the relative proportions of each were not, however, significantly correlated with the group attribution measures among the highly identified. Thus, when a strong motivation to view the in-group team positively was present, as is the case with the highly identified participants, mutations that are inconsistent with that motive have no effect on attributions. Finally, neither the frequencies or the proportion of the total mutations generated involving specific in-group individuals were significantly correlated with any of the group attribution measures, among either the high or low identified participants (all 16 correlations hovered around zero).



**Table 1.** Correlations between the absolute number and proportion of the mutations generated involving the in-group and the out-group teams and the four types of attributions for high and low identified participants

Attribution type: Identification group	Mutation type			
	Kansas—In-group team		Oklahoma—Out-group team	
	Number	Proportion	Number	Proportion
Oklahoma's luck				
Low identification	-.39*	-.41*	.56*	.55*
High identification	-.03	-.03	.15	.16
Oklahoma inappropriate				
Low identification	-.44*	-.50*	.49*	.46*
High identification	-.15	-.15	.27	.27
Oklahoma's more able				
Low identification	.47*	.47*	-.41*	-.41*
High identification	.05	.04	-.05	-.06
Kansas less able				
Low identification	.34*	.31*	-.30*	-.30*
High identification	-.11	-.11	-.03	-.03

*Note.* Correlations with an \* are significant at the  $p < .05$  level or less. High scores on the mutation measures indicate more of that type of mutation. High scores on each of the attribution measures indicates greater agreement with those items as labelled.

## Discussion

Motivation, in the form of high group identification, did not encourage avoidance of in-group team mutations, or more frequent out-group team mutations. Instead, mutation frequencies for the in-group as a whole and specific in-group members were identical for those high in identification, and both were higher than mutations focusing on the out-group (although not in terms of proportions where the three types did not differ). Differential mutation rates were observed only for those low in identification: specific in-group members were less likely to be mutated relative to the in-group team as a whole. We argue these results are inconsistent with a motivational view of mutation focus because the highly identified showed no preference for in-group individual mutations (which would be a means of salvaging the overall value of the group) compared to mutations involving the in-group team as a whole. Furthermore, among the highly identified, when they did perform the group protective mutations—those centred on specific in-group members—there was no link between such mutations and the attributional responses. These mutation results, we argue, are more supportive of the hypothesis that mutation focus is dependent upon familiarity with the groups and complexity of the knowledge possessed about them than with motivation to defend the group. The highly identified have more knowledge of the specific in-group members than do those low in identification and they think more complexly about the in-group (Wann & Branscombe, 1995a,b). Because of their increased familiarity, the highly identified are able to mutate specific player actions; the low identified possess less knowledge about individual team

members so they are unlikely to construct a mutation by focusing on a specific target. By default, then, the low identified are more likely to mutate the team as a whole.

Attributions, on the other hand, were consistently motivationally driven. On all four indices, the highly identified made more group-serving attributions than did those low in identification. What was mutated was correlated with the attributional responses among participants low in identification, replicating some prior work among uninvolved observers (Wells & Gavanski, 1989). Mutation focus was uncorrelated with attribution among those high in identification, also replicating a number of prior studies (Burris & Branscombe, 1993; N'gbala & Branscombe, 1995). The relationship between mutation and attribution appears to depend on the knowledge possessed about a domain. When little knowledge is possessed, as was the case with the low identified fans, what is mutated is correlated with attribution but when participants are highly informed about the domain, what they mutate is not what they blame.

In order to pursue our hypotheses further, an entirely different context was selected; the types of counterfactuals constructed and blame assignment for an auto accident were assessed. While the design of Expts 2 and 3 was identical, the former assessed mutation type for a moderately threatening event and the latter for a highly threatening event. If counterfactual focus is motivationally driven, participants should show an avoidance of mutating the self and a distinct preference for mutating the other potential target in the scenario. As with the prior experiment, however, we expected that mutation would be governed by knowledge and familiarity. Thus, the self should be the focus of mutation, at least as often as an unknown other, because people think about themselves more complexly than they think about others (Linville, 1982). However, when assigning blame, the other driver was expected to be decidedly more to blame than the self for exactly the same actions. When the self is mutated, mutation and blame assignment should be uncorrelated, although mutating the unknown other should be correlated with the blame assigned to that person.

## EXPERIMENT 2

### Method

#### *Participants and design*

Undergraduates (36 males, 42 females) were randomly assigned to read one of two counterbalanced versions of an automobile accident. In one version the west-bound driver and the actions committed by him were assigned to an unknown other 'Andy Fowler', and the south-bound driver's actions were assigned to 'You'. In the second version, assignment of the drivers to actions was reversed—the participant was described as the west-bound driver and the unknown other was presented as the south-bound driver. This was done in order to control for any effects due to the specifics of each driver's behaviour.

#### *Procedure and materials*

After reading the auto accident description involving the self and another, participants were asked to 'imagine and write down the four ways in which the accident might have been avoided IF ONLY . . .'. In this experiment, minor physical injuries for both drivers were reported and the damage to the autos was said to be covered by insurance.

The auto accident description itself contained features that could be easily mutated for either driver (and the self and other were assigned to both sets of driver behaviours equally often). Specifically, for Driver A, it

was said that sun glare made it difficult to see the traffic light. For Driver B, it was said that there was a patch of ice that made it difficult to stop. Both drivers were also described as being preoccupied with some personal news; Driver A was thinking about a grandmother who had been recently hospitalized and Driver B was hurrying to pick up a sick friend at work. Neither driver had consumed alcohol before the accident, which occurred at a busy traffic intersection.

All participants rated on 0 (not at all) to 100 (very much) scales, with five-point increments, the extent to which the other driver and the self caused, was responsible for, and should be blamed for the accident. On the same scales, participants rated the extent to which each driver should be punished for the accident and whether each driver was guilty of criminal negligence. Finally, to assess attributions to other possible causes besides the two drivers, participants rated the extent to which the accident was simply due to fate.

## Results

### *Mutations*

Each of the four mutations generated by the participants were coded by two independent readers according to whether they focused on the self (e.g. 'if only I had been going down the hill at a slower speed'), the other driver (e.g. 'if only the other driver had been going at a slower speed so it would have been easier to brake'), or some environmental condition (e.g. 'if only the weather was different'). Inter-rater reliability was high (97 per cent); the few discrepancies that occurred were resolved by the first author. The relative frequencies of the three types of mutations were examined using a three-level (type of mutation) repeated measures analysis of variance. The frequency of mutating the three targets was significantly different ( $F(2,154) = 5.61, p < .004$ ). The self was targeted for mutation ( $M = 1.62$ ) more often than environmental factors ( $M = .97, F(1,77) = 8.40, p < .005$ ), as was the other driver ( $M = 1.41, F(1,77) = 4.51, p < .04$ ). Although the self tended to be mutated more often than the other driver, this effect was not reliable ( $F(1,77) = 1.80, p < .18$ ). When the proportions of the total mutations generated were examined, the same pattern of results emerged: the self was mutated more frequently (40 per cent) than the other driver (35 per cent) and both were more frequently targeted than other factors (25 per cent). When only the *first* mutation generated was examined, the self (40 per cent) and the other driver (40 per cent) were mutated equally often, and both were mutated more frequently than the environmental conditions (20 per cent,  $\chi^2(1) = 4.79, p < .05$ ).

### *Attributions*

The five measures assessing degree of fault assigned to each of the drivers were combined to create an index for the self ( $\alpha = .87$ ) and the other driver ( $\alpha = .90$ ). Relative blame assignment for the self, the other driver, and fate was examined using a three-level (target) repeated measures analysis of variance. The degree of blame assigned to these three factors varied significantly ( $F(2,154) = 4.96, p < .008$ ). The self was blamed ( $M = 39.33$ ) significantly less than the other driver ( $M = 50.42, F(1,77) = 9.00, p < .004$ ) and the other driver was blamed more than fate ( $M = 38.91, F(1,77) = 6.36, p < .01$ ). The amount of blame assigned to the self and fate did not differ ( $F < 1$ ).

### *Mutation-attribution relationships*

The number of other driver mutations was positively correlated with the amount of blame assigned to that person ( $r(78) = .32, p < .005$ ). Similarly, the number of other driver

mutations was negatively correlated with blame assigned to the self ( $r(78) = -.33$ ,  $p < .003$ ). This is conceptually similar to the prior results with low identified participants. The number of self-mutations did not significantly correlate with the amount of blame assigned to the self ( $r(78) = .14$ , n.s.) or the blame assigned to the other driver ( $r(78) = -.03$ , n.s.), which is the same as what was found previously among highly identified participants. Mutation frequency of environmental conditions was uncorrelated with blame assignment to all three targets.

On the assumption that the first mutation generated might be perceived as the most plausible, and as a consequence could affect blame assignment more strongly than subsequent mutations, blame for each target was correlated separately for those who mutated either the self or the other driver first. Consistent with the analyses involving all of the mutations generated, self-blame was not associated with mutating the self first ( $r(78) = .19$ , n.s.). Again, mutating the other driver first was correlated with the blame assigned to that individual ( $r(78) = .40$ ,  $p < .001$ ).

## Discussion

No evidence that the self is avoided as a means of undoing a negative outcome was observed. In fact, the self tended to be the favoured target for mutation overall. Yet, participants were highly self-serving in their attributions of blame. The other driver was always blamed more than the self. Only when what was mutated (in terms of either the first mutation or the overall number) was consistent with more blame for the other person was there a correlation between mutation and attribution. When, however, what was mutated might imply self-blame, there was no relationship between mutation and attribution.

Although a very different context was employed, the results of this study are quite consistent with those obtained in the first experiment. What is mutated appears to be what is most familiar or known to the perceiver. No evidence that mutation is self-serving or motivationally driven was obtained. If, however, what is mutated is not consistent with prevailing blame avoidance motivation, then there is no correlation between the two measures. Only when mutations (e.g. the other driver is mutated most frequently) are consistent with the attributional goal (e.g. to blame the other more than the self) are mutation and blame correlated.

In order to rule out an alternative explanation that assumes the motivation to avoid self-blame was insufficiently high to also influence mutation, Expt 3 was undertaken. The severity of the outcome, and hence the motivation to avoid self-blame, was increased. Lasting physical trauma was added for both drivers. If motivation does influence mutation focus, then the self should be avoided as a target of mutation significantly more frequently in this experiment compared to the previous one, and the other driver should be favoured more as a means of undoing this very serious outcome. If, however, mutation is guided by familiarity with a target or complexity of knowledge, the self should continue to be targeted for mutation at least as often as the other driver. Again, if the determinants of mutation and attribution are different, no correlation between the two should be observed when their implications are inconsistent with the desire to avoid blaming the self for a negative outcome.

## EXPERIMENT 3

### Method

#### *Participants and design*

Undergraduates (34 males, 30 females) were randomly assigned to read one of the two counterbalanced versions of the auto accident. Again, in one version the south-bound driver was said to be 'You' and the west-bound driver was an unknown other 'Stephan Dunbar'. The driving behaviours assigned to the two targets were reversed in the second version of the event.

#### *Procedure and materials*

The only change in the event description involved a more severe event outcome. In this experiment, both vehicles were completely destroyed and the injuries sustained by the two drivers were said to be extensive and likely to affect their futures; both drivers were permanently disabled. The mutation instructions solicited only one counterfactual by asking participants to 'describe the most striking way that the accident could have been avoided IF ONLY . . . '.

### Results

#### *Mutations*

The mutation generated by each participant was coded by two individuals in terms of whether it involved a focus on the self (e.g. 'if only I had been paying more attention to the road'), the other driver (e.g. 'if only the other driver would not have entered the intersection'), or some environmental factor (e.g. 'if only Americans weren't so addicted to the automobile and the weather hadn't been so bad'). Inter-rater reliability was 94 per cent; the few discrepancies that occurred were resolved by the first author. The relative frequency of the three types of mutations did not vary ( $\chi^2(2) = 3.88$ , n.s.). Both the self (38 per cent) and the other driver (40 per cent) tended to be mutated more frequently than other environmental factors (22 per cent), although neither comparison attained significance ( $ps < .10$ ).

#### *Attributions*

The five items assessing the fault assigned to each of the drivers were combined to create an index for the self ( $\alpha = .94$ ) and the other driver ( $\alpha = .94$ ). Relative blame assigned to the self, the other driver and fate were examined in a three-level (target) repeated measures analysis of variance. Blame assignment varied significantly among the targets ( $F(2,126) = 3.30$ ,  $p < .04$ ). The self was assigned significantly less blame ( $M = 40.20$ ) than the other driver ( $M = 52.68$ ,  $F(1,63) = 7.82$ ,  $p < .007$ ). The amount of blame assigned to fate ( $M = 46.48$ ) did not differ from that assigned to either the self or the other driver, but fell in between the two.

#### *Mutation-attribution relationships*

Mutating the other driver (versus the self) was positively associated with the blame assigned to that person ( $r(50) = .27$ ,  $p < .05$ ), but there was no relationship between mutating the self (versus the other) and degree of self blame ( $r(50) = .05$ , n.s.). Another way to examine the link between what is mutated and blamed is to compare the blame

assigned by persons who mutated different targets. Accordingly, a 3 (type of mutation)  $\times$  3 (target of blame) mixed analysis of variance was performed, with repeated measures on the latter factor. The two-way interaction between what was mutated and the blame assigned to the targets was not significant; blame was apportioned differentially by target as was revealed in the earlier analysis, but it was not a function of what target was mutated.

### Discussion and comparison of the results obtained in Expts 2 and 3

Experiment 3 involved considerably more serious outcomes than did Expt 2. If motivation were to encourage the avoidance of self-mutations as a means of avoiding self-blame, the self should not be mutated particularly when the outcome was serious. However, the pattern of mutations observed in the two experiments was essentially the same. The percentage of participants who mutated the self first (40 per cent), or as a percentage of their total mutations (40 per cent), did not differ when the outcome was not especially serious (Expt 2) compared to the percentage of self-mutations that were generated (38 per cent), when the outcome was very serious (Expt 3,  $\chi^2 < 1$ ). In neither of the experiments was there a preference to avoid mutating the self. Likewise, the percentage of participants who mutated the other driver first (40 per cent), or as a percentage of their total mutations (35 per cent) did not differ in Expt 2 compared to Expt 3 (where 40 per cent of the mutations were of the other driver,  $\chi^2 < 1$ ). Finally, the percentage of participants who mutated some other environmental factor first did not differ for Expt 2 (20 per cent) versus Expt 3 (22 per cent,  $\chi^2 < 1$ ).

Blame assignment was consistent across the two experiments as well. The other driver was blamed more ( $M = 50.42$  and  $M = 52.68$  for Expts 2 and 3 respectively) than the self ( $M = 39.33$  and  $M = 40.20$  for Expts 2 and 3 respectively), regardless of the severity of the outcome. There was no effect of experiment ( $F < 1$ ), implying that the motivation to avoid self-blame was activated even though a relatively mild negative outcome was employed. Mutation and attribution were positively correlated only when mutation was consistent with more blame for the other driver. Mutating the self was unrelated to attribution.

### GENERAL DISCUSSION

In three studies, where motivation was operationalized in different ways, no evidence was obtained that mutation focus is motivationally driven. Rather, some support for the idea that mutation focus may be a function of the knowledge possessed about a domain was obtained. In Expt 1, the highly identified should have been motivated to avoid mutating their in-group as a whole. However, they focused on the in-group team in their mutations equally as often as they focused on specific in-group targets, and mutations of the in-group team were seen as equally plausible as those involving only a specific in-group member. In Expts 2 and 3, there was no tendency to avoid mutating the self, even as the outcome increased in severity.

Attribution, on the other hand, clearly was both self- and group protective. Regardless of what was mutated, those who were most motivated to perceive the in-group positively (i.e. the highly identified) showed stronger in-group-favouring biases than did those who

were less concerned with the group. Likewise, regardless of what was mutated, participants consistently blamed themselves less for the auto accident outcome compared to the other driver.

Even for highly traumatic events experienced by the self such as rape or the accidental death of a loved one, people often mutate something about themselves rather than the other people involved in the event or environmental conditions (Branscombe, Owen & Allison, 1995; Davis, Lehman, Wortman, Silver & Thompson, 1995; Dunning & Parpal, 1989). Therefore, the results obtained in the current experiments are not simply due to the use of events that did not actually happen to the participants, or that were insufficiently traumatic. However, it is possible that because the participants knew they were not actually involved in the auto accident, but simply imagined themselves in that context, the tendency to focus on the self was not as high as it might be were they not in this 'observer' role, but did in fact find themselves victims of such a negative outcome. In other words, such a 'role-playing' context may have undermined the expected familiarity-based tendency to focus on and mutate the self more so than the other driver. In one study of rape victims (Branscombe *et al.*, 1995), virtually all of the spontaneous counterfactual thoughts reported focused on some aspect of the self's actions or inactions. But, despite high levels of focusing on the self in their mutations, participants consistently blamed the assailant more than themselves for the outcome that did occur.

As N'gbala & Branscombe (1995) have reported previously, what people mutate is not necessarily what they rate as most causal or blameworthy. When there was no investment in a particular attributional conclusion, as was the case with the low identified sports fans, then mutation was correlated with attributions. This is consistent with Wells & Gavanski's (1989) research where the participants had no stake in what attributions were made; they were uninvolved observers evaluating events of no personal importance. When, however, important attributional goals were at stake, as was the case in the current research—both when participants were highly invested in a group and when the self was imagined to be one of the agents involved and was focused on in the mutation—then mutation and attribution were not correlated. Only when the mutation generated was consistent with the individual's attributional goals—to blame the other driver—was there a relationship between what was mutated and blame assignment.

Prior research has suggested that controllable causes are more frequently targeted for mutation than are uncontrollable causes (Giroto, Legrenzi & Rizzo, 1991; Roesse & Olson, 1995a). The results obtained in the current research were generally consistent with this notion; both the self and other in-group members are likely to be seen as more controllable, and should be therefore targeted for mutation more than an out-group or unfamiliar other. Both types of in-group mutations were, in fact, rated as more plausible than those that focused on the out-group. Yet, despite the greater plausibility of in-group mutations, and their potentially higher perceived controllability, they were not linked with attributions among participants who were highly identified with the in-group. Similarly, although in the narrow points margin condition more plausible counterfactuals were available, there was no effect of this variable on attributions. There was, however, support for Kahneman & Miller's (1986) claim regarding spectators at sporting events where, 'the outcome of a contest is more commonly undone by improving the losing performance than by imagining a poorer performance of the winning team' (p. 143). Participants consistently mutated the in-group, or the losing team, more frequently than

the winning team, regardless of their identification level, although this could be also due to differential perceived controllability of the two types of targets.

Familiarity with a domain implies availability of more specific alternatives, allowing more features to appear potentially exceptional which is the critical variable controlling mutation, according to Kahneman & Miller (1986). Similarly, greater complexity of knowledge implies more options for mutating specific features of an event. That is, indeed, what we observed. The highly identified mutated specific in-group targets more than those low in identification and the self was mutated at least as often as an unknown other, although such mutations failed to influence evaluations of the groups in the former case and blame assignment for the accident in the latter experiments. Although the familiarity hypothesis could predict more self than other driver mutations, the lack of difference observed here in these two types of mutations might well be due to the fact that the participants knew the accident did not actually happen to them.

When answering the question, 'How could this outcome have been otherwise?', we speculate that participants generally focus on what their pre-existing detailed knowledge indicates *enabled or was merely necessary* for the outcome to occur. People who are unfamiliar with a domain, the low identified fans, are forced to answer this question almost exclusively by focusing on general features of the event or the in-group as a whole. In contrast, when assigning blame, and answering the question 'What caused or brought this event about?', participants with a stake in the answer point to additional factors that they took for granted or presupposed when performing the mutation task—the other person or the out-group. Those not motivationally involved may accept the feature that they mutated and not look any further when assigning blame. Therefore, only among perceivers without a particular attributional goal, or for those whose mutations are motivationally consistent with their attributional goal (i.e. mutating the other driver is consistent with the goal of blaming the other), would a relationship between mutation and attribution be expected. Blame, then, for the uninvolved judge, can be assessed most easily by assigning more to the most available feature—the factor initially mutated. This would result in the feature that is mutated being the same as the feature attributed the most blame, as we observed among low identified fans, and in the auto accident experiments when the other driver was mutated. However, future research that also manipulates the order of the two tasks engaged in—mutation and attribution—could further assess the validity of this notion.

In conclusion, the direction of the counterfactual that is ultimately constructed—whether the event is undone so that a better or worse alternative is favoured, or deemed to be the most likely alternative to reality—might well be influenced by self-protection motivation (cf. Markman *et al.*, 1993). We did not directly address the preferred direction of counterfactual generation. Our instructions exclusively requested participants to undo the outcome so that a better outcome was imagined. No evidence that mutation focus, which is independent of direction, is influenced by self- or group protection motivation was obtained for such better outcome alternatives. We argue that what was focused on and seemed easiest to change was what was most accessible, given the amount of knowledge the participant possessed about the domain. It is in this sense that, we argue, mutation may be relatively automatic and stimulus driven (see Bargh, 1984). As a consequence of such automatic focusing effects, motivation may be unable to exert an influence on what is mutated. Attribution processes, in contrast, require considerably greater elaboration and conscious attention so that motivation has an opportunity to play a role.



Our results provide support for Kahneman & Miller's (1986) original proposition that mutation is driven by exceptionality, but what stands out and is focused on may depend on the knowledge the perceiver possesses about a domain. No evidence to support Sherman & McConnell's (1995) speculation that mutation focus itself is motivated was obtained. The two self experiments most clearly tested their argument that mutating the self is dysfunctional because of its hypothesized effect of increasing self-blame. Despite high levels of self-mutation, participants consistently avoided blaming themselves for the outcome. Therefore, because mutation is not necessarily linked with attribution, there is no basis for the claim that *mutation focus per se* is either functional or dysfunctional. Mutating the self was not predictive of attributions. Attributions were consistently self- or group protective, regardless of what was mutated.

### Acknowledgements

Preparation of this manuscript was facilitated by a General Research Fund grant from the University of Kansas to the first author. We thank Vincent Yzerbyt and two anonymous reviewers for their insightful comments that helped to improve the manuscript, and Amy Cohen, Jason Coleman and Kris King for their assistance with the data collection and coding. Portions of this research were presented at the Nags Head Conference on Judgment and Decision Making, Highland Beach, Florida, May 1994.

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Received 7 December 1995; revised version received 8 November 1996