



# A generalized bias against disconfirmatory evidence in schizophrenia

Steffen Moritz <sup>a,\*</sup>, Todd S. Woodward <sup>b,c</sup>

<sup>a</sup> *University of Hamburg, University Hospital of Psychiatry, Hamburg, Germany*

<sup>b</sup> *Department of Research, Riverview Hospital, 2601 Lougheed Highway, Coquitlam, B.C., Canada*

<sup>c</sup> *Department of Psychology, Simon Fraser University, 8888 University Drive, Burnaby, B.C., Canada*

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## Abstract

Fixation onto false/unrealistic beliefs is a core feature of schizophrenic delusions. A recent study conducted by our research group has provided evidence for the presence of a bias against disconfirmatory evidence (BADE) in patients with schizophrenia. Importantly, this bias was found with delusion-neutral material. To further validate a BADE as an underlying component of schizophrenic delusions, we recruited 34 presently deluded and non-deluded patients with schizophrenia, along with 26 healthy and 46 mixed psychiatric control participants. Participants were administered a closure task. On each trial, a common object (e.g., elephant) was increasingly disambiguated (i.e., shown in decreasing degrees of fragmentation). The participants were required to assess the plausibility of different interpretations at each of the up to eight stages in each trial. In line with the main hypothesis, patients with schizophrenia downgraded the ratings for incorrect interpretations significantly less over the course of task completion than did healthy and psychiatric controls. In contrast, the gradual upgrading of correct interpretations was similar across all groups, suggesting that the pattern of results obtained for incorrect interpretations reflects a BADE and not a mere repetition of prior responses or a lack of attention to the task at hand. The present study suggests that a BADE is a core feature of schizophrenia, and that this style of thinking is not confined to delusion-congruent scenarios.

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## 1. Introduction

Fixation on false/unrealistic beliefs is a hallmark feature of schizophrenic delusions (Jaspers, 1913/1973). Delusional patients are not amenable to evidence challenging their beliefs. Frequently, proofs against delusional beliefs are not only discarded by the deluded patient but are incorporated in the belief

\* Corresponding author. Universitätsklinikum Hamburg-Eppendorf, Klinik für Psychiatrie und Psychotherapie, Martinistraße 52, D-20246 Hamburg, Germany. Tel.: +49 40 42803 6565; fax: +49 40 42803 2999.

E-mail address: [moritz@uke.uni-hamburg.de](mailto:moritz@uke.uni-hamburg.de) (S. Moritz).

system, for example, by accusing “non-believers” of being part of a conspiracy against the patient. In clinical practice, such “safety behaviours” (Freeman et al., 2002) have led to recommendations against aggressively disputing an overt delusional belief system (Watts et al., 1973) in the absence of a strong therapeutic relationship (Fowler et al., 1995, p. 78–79).

Little effort has been devoted to the question of whether belief fixation in schizophrenia is a feature solely observed for emotionally charged or delusion-specific themes, or whether patients are less able to revise false convictions *in general*. Put differently, it is unclear whether delusional fixation is merely the most dramatic feature of a general inclination not to withdraw from firmly held beliefs. Moreover, it remains to be clarified whether incorrigibility occurs in delusional patients only, or if it is also present in currently non-deluded patients with schizophrenia — perhaps to a lesser but still pathological extent.

In a first study on this issue (Woodward et al., 2004; Woodward et al., *in press*), we successively presented three pictures, which increasingly resolved an ambiguous plot (e.g., the first picture shows a man who is bending over a fence and is watching a barking dog; on the following pictures, it becomes evident that the man has just escaped from the dog by jumping over the fence). After each picture, participants were asked to rate the plausibility of each of the four interpretations. One of the four interpretations appeared implausible on presentation of the first picture, but it eventually proved to be *true* (in the example above: “The man has just escaped from the barking dog”). Two of the other interpretations appeared plausible on presentation of the first pictured scene, but they eventually proved to be false (lures; e.g. “The man has just built a fence for his dog”). The lure interpretations were disproved after either the second or the third (final) picture had been presented. For all trials, one interpretation was *absurd/implausible* at all stages (e.g., “The man is shopping for guard dogs”).

Two main findings emerged from this study. First, in line with our a priori hypothesis, subjects with schizophrenia were biased against reducing their plausibility ratings for lure interpretations over the course of the picture sequences when compared with controls, and this was particularly pronounced for currently delusional patients. We refer to this as a bias against disconfirmatory evidence (BADE). Second, in

line with the liberal acceptance account of schizophrenia (Moritz and Woodward, 2004), which holds that patients are willing to accept implausible scenarios, absurd/implausible interpretations were rated as significantly more plausible by schizophrenia patients, irrespective of their current delusional status, than by healthy controls.

In the present study, we attempted to replicate the core finding of the initial study using a different experimental procedure. This time, sequences of picture fragments were used instead of complex scenarios. Over a sequence of up to eight pictures (in the following referred to as stages), a common object (e.g., an elephant) was made increasingly visible (i.e., more features were added to the picture), and it was thus disambiguated. At each stage, interpretations of what the picture might depict were rated on a 5-point scale (dismissed, unlikely, possible, likely, positive decision). As soon as a decision was made, the trial ended. It was expected that patients with schizophrenia would display a BADE, such that compared with controls, patients would display a bias against downrating previously high ratings on the basis of disconfirmatory evidence, whereas controls would strongly revise previously high ratings as counter-evidence accumulated. In contrast, we expected no group differences on incorporation of confirmatory evidence. The inclusion of a psychiatric control group allowed us to test whether a BADE is specific to schizophrenia or is found in other psychiatric illnesses as well.

Furthermore, we tested whether a BADE for delusion-neutral pictures would be more pronounced in currently deluded patients with schizophrenia. The demonstration of an effect specific to delusional patients would strengthen the claim that a BADE contributes to the emergence and/or maintenance of delusions.

## 2. Methods

### 2.1. Subjects

Thirty-four schizophrenia inpatients, recruited from the psychiatric University Hospital of Hamburg, took part in the experiment. Twenty-six inpatients with post-traumatic stress disorder (PTSD) and 20

inpatients with obsessive–compulsive disorder (OCD) were taken from the same clinical environment. PTSD and OCD form a particularly interesting control group in schizophrenia research because those patients on the one hand display symptoms that are reminiscent of positive schizophrenia symptomatology (e.g., intrusions of unwanted thoughts and images), but on the other hand show illness insight (e.g., acknowledgment that these thoughts and images arise from their own mind).

All psychiatric patients had been carefully screened by trained clinicians for the validity of the suspected diagnosis. Diagnoses were verified with the neuropsychiatric interview (MINI, Sheehan et al., 1998) and the Structured Clinical Interview for DSM-IV (SCID-I) sections for PTSD and schizophrenia for the corresponding patient groups. The severity of the OCD symptomatology was determined with the Yale–Brown Obsessive–Compulsive Scale (Y–BOCS, Goodman et al., 1989; German version by Hand and Büttner-Westphal, 1991; Jacobsen et al., 2003). Twenty-six healthy participants had been contacted through advertisement, word-of-mouth or were obtained from an established subject pool. Healthy participants were screened with the MINI. None of the healthy control participants displayed any psychiatric disorder, reported drug and/or alcohol dependence, or suffered severe brain damage. Patients were approached as part of a routine assessment at baseline. Few patients refused to participate and were later approached for participation.

OCD patients with lack of insight or over-valued ideas, as assessed with the Y–BOCS item 11, were

excluded. The severity of the OCD symptomatology in the 20 OCD patients was rated a priori according to the Y–BOCS total score ( $M=23.25$ ;  $SD=5.69$ ). Premorbid intelligence was determined with a vocabulary task (Lehrl, 1995).

The Positive and Negative Syndrome Scale (PANSS, Kay et al., 1989) was administered to examine schizophrenic psychopathology. The PANSS was complemented with six additional ratings from the Positive and Negative and Disorganized Syndrome Scale (PANADSS, Moritz et al., 2001) tapping inappropriate affect, flat affect, associative loosening, thought blocking, auditory hallucinations and other hallucinations. These symptoms were added as the PANSS does not provide (unambiguous) ratings for these symptoms (e.g., flat and inappropriate affects are not differentiated in the PANSS).

Three psychopathological syndromes were derived from the PANSS/PANADSS ratings closely following recent factor-analytic solutions (Mass et al., 2000; Moritz et al., 2001): the positive syndrome (delusions, hallucinations, suspiciousness/ideas of persecution, unusual thought content), the negative syndrome (flat affect, emotional withdrawal, lack of relationship, passive social withdrawal, lack of spontaneity) and disorganization (associative loosening, inappropriate affect, problems with abstract thinking, attention, disorientation). Eighteen of the patients with schizophrenia displayed current symptoms of delusions as assessed with the PANSS symptom for suspiciousness (PANSS positive symptom 6,  $\geq 3$ ).

Table 1 lists sociodemographic and psychopathological characteristics of the sample. After explaining

Table 1  
Sociodemographic and psychopathological variables

Variables	Healthy (H; $n=26$ )	Schizophrenia (S; $n=34$ )	Psychiatric (P; $n=46$ )	Statistics; LSD post-hoc
<i>Sociodemographic variables</i>				
Age	31.31 (9.46)	33.88 (9.93)	33.61 (9.05)	$F(2,103)=0.65$ , $P>0.5$ , NS
Years of formal school education	11.77 (1.48)	11.72 (1.71)	11.13 (1.60)	$F(2,103)=1.89$ , $P>0.1$ , NS
Gender (male/female)	12/14	23/11	26/20	$\chi^2(1)=2.82$ , $P>0.2$ , NS
Premorbid intelligence (IQ)	112.00 (12.49)	113.78 (15.88)	109.00 (15.14)	$F(2,94)=0.99$ , $P>0.3$ , NS
<i>Psychopathological variables</i>				
Number of hospitalizations (including present)	–	4.74 (4.66)	1.32 (1.22)	$t(76)=4.17$ , $P<0.001$
PANSS total score	–	67.03 (17.11)	–	–
HDRS total score	0.87 (2.70)	–	14.87 (6.84)	$t(67)=12.11$ , $P<0.001$
Chlorpromazine equivalents in mg	–	631.55 (832.31)	–	–

the procedure of the study to the subjects, written informed consent was obtained.

## 2.2. Procedures

Participants were given six experimental trials following two practice trials. All trials consisted of a sequence of eight stages, each showing a common object in decreasing degrees of fragmentation: new object features were added to each new picture until the entire object was displayed at the final stage. For example, at the first stage of a practice trial, which would eventually show a frog, the presented object strongly resembled a lemon, as only the contour of the frog was displayed initially. An illustrative example is shown in the Appendix. The objects were depicted as post-edited simple black-and-white drawings (experimental trials: float/raft, elephant, guitar, mill, mermaid, castle). The pictures were then gradually decomposed into puzzles. Instructions and trials were presented on an Apple® Computer. The trials were run in a fixed order with half of the trials (1, 3 and 5) being accompanied by cue interpretations (depending on the trial: 6–9 interpretations), which were assessed for plausibility until a decision was reached. Plausibility ratings had to be given on a five-point Likert scale (0 = dismissed, 1 = unlikely, 2 = possible, 3 = likely, 4 = positive decision). Once a decision (i.e., rating=4) was reached, the next trial (i.e., first fragment of new picture) was initiated. No feedback was provided about incorrect judgments. Only one of the interpretations eventually proved to be correct. The cue interpretations were derived from a pilot study in which common associations were gathered.

In the remaining trials (trials 2, 4 and 6), no cue interpretations were provided, and the participants were instructed to create their own interpretations at each stage, which were subsequently rated for plausibility. If an interpretation was dismissed at any given stage (i.e., rating=0), it still had to be re-evaluated at all remaining stages. Again, the next trial was initiated once a decision was reached.

## 2.3. Strategy of data analysis

Several indices were calculated. The central index for the BADE was the difference score between mean plausibility ratings for incorrect interpretations at the

first stage relative to the mean plausibility ratings for incorrect interpretations at later stages. The signature of a BADE effect would be a minimal reduction of plausibility ratings for incorrect interpretations across stages for patients. We also calculated a bias against confirmatory evidence (BACE) index (i.e., mean plausibility ratings for correct interpretations at the first stage relative to the mean plausibility ratings for correct interpretations at later stages) to compare the evaluation patterns of facing either confirmatory or disconfirmatory evidence. Both BACE and BADE could only be computed for trials in which at least one response was made. Simple mean comparisons were used to determine group differences for the BADE and BACE parameters at the different stages. Apart from these main parameters (difference score initial vs. later rating for incorrect and correct responses), we also explored the mean plausibility rating for each stage. Since some subjects decided before stage 6 on all tasks, BADE parameters for the entire group could only be obtained until stage 5 (see below). Draws to decision were the average number of stages needed to make a decision (i.e., rating=4). Therefore, the number of stages until a decision gives a measure of early decision making, or a jumping to conclusions (JTC) bias.

## 3. Results

### 3.1. Sociodemographic variables

The samples did not differ on any major sociodemographic background variables (age, gender, formal school education, premorbid intelligence). Post-hoc group comparisons were greater than  $P=0.1$  for all parameters, even before correction for multiple comparisons.

### 3.2. Bias against disconfirmatory evidence (BADE)

All subsequent analyses collapsed ratings from fixed and self-generated trials, as Trial Type (fixed, self-generated) did not yield any significant interaction with Group (schizophrenia, psychiatric controls, healthy controls) when entered as an additional within-subject factor (Fig. 1). While all three samples displayed comparable plausibility ratings at the initial stage (see Table 2), samples differed significantly across time in their acceptance of eventually incorrect

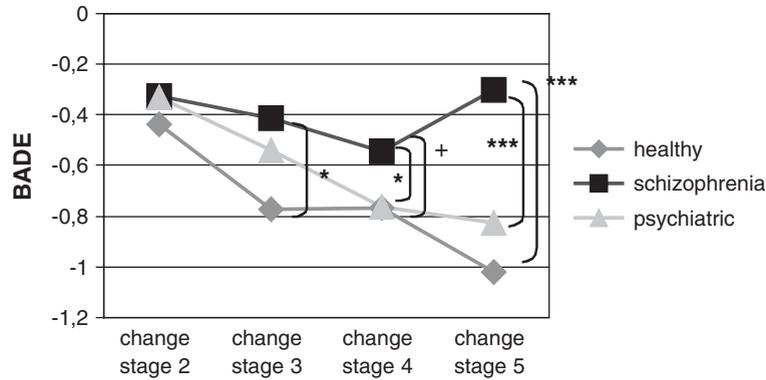


Fig. 1. Bias Against Disconfirmatory Evidence (BADE) — change scores (initial stage to subsequent stages) averaged over self-generated and cued trials. Patients with schizophrenia decreased their plausibility scores significantly less than controls across time. Group differences were strongest for stages 4,  $F(2,104)=3.15, P<0.05$ , and 5,  $F(2,104)=9.80, P<0.001$ . No statistics are provided for later stages due to a large number of missing values in all groups (i.e., 17 participants made decisions before stage 6). For absolute plausibility scores, group differences also yielded significance for stage 5,  $F(2, 104)=6.34, P<0.005$  (S>P, H; both  $P<0.005$ ) and stage 3,  $F(2, 104)=3.15, P<.05$  (S>H;  $P=0.01$ ). + $P\leq 0.1$ ; \*  $P\leq 0.05$ , \*\*\*  $P\leq 0.001$  (post-hoc comparison).

hypotheses. Whereas all participant groups downrated plausibility ratings over the course of the stages, patients with schizophrenia showed the least tendency to do so, which, at stage 5, yielded significance relative to both healthy and psychiatric controls for both change and absolute scores (i.e., mean plausibility for incorrect interpretations at stage 5). At stage 5, 63% and 58% of the ratings given for incorrect interpretations made by healthy and psychiatric control participants were (correct) rejections (rating=0). The corresponding rate in schizophrenia patients was only 47%, which yielded significance to both control groups (overall  $P=0.003$ , post-hoc: S<P, H, at least  $P<0.01$ ). To a lesser degree, group differences also emerged for incorrect interpretations at the third and fourth stage.

### 3.3. Bias against confirmatory evidence (BACE)

Few differences occurred for responses regarding correct interpretations. However, patients with schizo-

phrenia elevated their plausibility scores significantly more than psychiatric controls from stage 1 to 2. However, no differences occurred for the subsequent stage (see Fig. 2). Finally, no group differences yielded significance for absolute plausibility scores in stage 1, 2 or 3.

### 3.4. Secondary parameters

For the trials in which participants had to generate their own interpretations (trials 2, 4 and 6), healthy subjects offered more interpretations than schizophrenia and non-schizophrenia psychiatric patients (see Table 2).

### 3.5. Correlational analyses

All psychopathological (three syndromes, composite score for delusions, number of hospitalizations) and sociodemographic (age, gender, education)

Table 2  
Secondary experimental parameters

Variables	Healthy (H; n=26)	Schizophrenia (S; n=34)	Psychiatric (P; n=46)	Statistics; LSD post-hoc
Initial Plausibility Rating (i.e., at stage 1)	1.65 (0.27)	1.52 (0.45)	1.55 (0.29)	$F(2,103)=1.10, P>0.3$ ; NS
# Self-Generated Interpretations (trials 2, 4, 6)	16.42 (3.98)	14.03 (3.61)	13.89 (3.95)	$F(2,103)=4.06, P=0.02$ ; P, S<H
Draws to Decision (rating=4)*	4.79 (.55)	4.45 (.87)	4.65 (.60)	$F(2,103)=1.90, P>0.1$ ; NS
Draws to Exclusions (rating=0)*	2.88 (0.54)	2.59 (0.67)	2.69 (0.65)	$F(2,103)=1.55, P>0.2$ ; NS

Notes. \*=no group differences emerged when parameters were split according to correct vs. incorrect ratings.

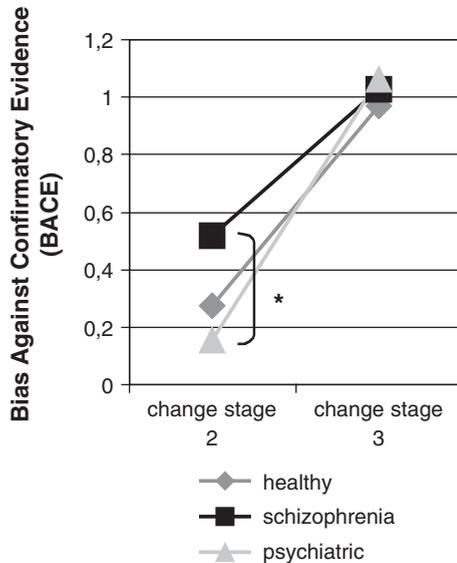


Fig. 2. Bias Against Confirmatory Evidence (BACE) — change scores. At trend level, patients with schizophrenia display a greater change of their plausibility scores from stage 1 to 2,  $F(2,104)=2.85$ ,  $P=0.06$ . For the third stage, no differences occurred. No statistics could be computed for later stages because of excessive missing values. No group differences yielded significance for absolute plausibility scores on stage 1, 2 or 3.  $*P\leq 0.05$  (post-hoc comparison).

indices were correlated with the main experimental parameters. The level of significance was set at 0.01, as the exploratory nature of these analyses inflated the chance of false-positive findings. None of the correlations surpassed this threshold. With a more lax criterion ( $P<0.05$ ), positive and specifically delusional symptomatology still did not correlate with the core experimental parameters. A division of patients into deluded ( $n=18$ ) versus non-deluded ( $n=16$ ) confirmed the correlational results. However, schizophrenic disorganization was correlated with the number of self-generated interpretations in trials 2, 4 and 6 ( $r=0.36$ ,  $P=0.04$ ).

#### 4. Discussion

The results confirm a recent finding (Woodward et al., in press, 2004) that a bias against disconfirmatory evidence (BADE) in schizophrenia is not confined to delusional scenarios, but extends to affectively neutral material. Patients with schizophre-

nia decreased their plausibility ratings for incorrect interpretations significantly less over time than controls. The fact that a significant difference occurred not only between patients with schizophrenia and healthy controls, but also with regard to psychiatric controls, supports the postulation that a BADE is specific to the schizophrenic illness, and not a mere epiphenomenon of psychiatric illness. In view of the result that plausibility ratings for correct interpretations were comparable across all groups, this response pattern cannot be attributed to a general response perseverance, or a lack of attention to the task at hand.

In contrast to our previous work (Woodward et al., in press), no differences emerged between delusional and non-delusional patients. Therefore, whether BADE is a state or trait aspect of schizophrenia remains an open question. In the following, we would like to offer some possible reasons for why the predicted difference between delusional and non-delusional patients might not have occurred.

First, the present sample was smaller than the one employed in the former study. Thus, group differences may have failed to emerge due to a lack of power. Second, methodological differences in terms of the experimental set-up between the first and the present study, are likely to account for the apparent discrepancy. Unlike the initial study, in which participants had been lured into a false interpretative direction (see section 1), in the present study the appropriate response in the initial stage was rather ambiguous, in that it did not provide strong lure interpretations. At the initial stage of the fixed trials, the correct interpretation and the alternatives given were equally plausible. Therefore, preliminary decisions were less likely under the present experimental conditions. That is to say, subjects may have experienced a higher degree of uncertainty on the initial trial for the present study compared with our initial study, suggesting that the ability to shift away from *strongly* held (incorrect) positions is a feature that may separate delusional from non-delusional patients.

Congruent with this assumption, in another previous study (Moritz and Woodward, 2005) we found that delusional but not non-delusional patients reacted increasingly incautiously over the course of a probabilistic reasoning task, in which subjects were asked to deduce from which of two jars a sequence of beads

derive: on the first task, a large proportion of patients in both subgroups decided very early in favour of the *correct* one of two jars in response to a sequence of beads (i.e., jumping to conclusions bias). In the second task, participants were shown a sequence of beads that lured them into believing that the new task demands would be as obvious as the preceding: the first 10 beads clearly favoured one of the two jars; the second half, however, made both jars equally plausible. Unlike healthy and non-delusional patients, delusional patients proceeded to respond hastily in the third task, suggesting that they were still adopting the previously successful but now obsolete response strategy of the first task, and did not take into account (or did not comprehend) the cautioning experience of the second task.

As a final consideration, we would like to mention some limitations of the present study and some suggestions for future research. First, to assess the representativeness of the sample, it would have been favourable to determine additional cognitive (e.g., executive functioning, reasoning) and meta-cognitive measures. Moreover, it would have been beneficial to concurrently assess the relationship between measures tapping delusional incorrigibility and the BADE. The co-administration of such a measure (e.g., Brett-Jones et al., 1987) may more directly test the claim that the two constructs are linked. Finally, longitudinal studies are needed to directly confirm the hypothesis that a BADE contributes to

delusion formation/maintenance and is just an expression or correlate of deludedness. Further, the relationship between a BADE and other cognitive biases needs to be elucidated.

The present findings offer potential therapeutic implications. If delusion formation and maintenance are mediated via a general BADE, cognitive therapy in schizophrenia could use tasks such as the ones employed in the previous (Woodward et al., *in press*) and the present study to promote critical hypothesis testing. An improvement of cognitive skills needed for hypothesis testing may encourage schizophrenia patients to cast doubt on strongly held positions. Such an indirect strategy has clear advantages over an overt challenge of the core of the belief system. First, threats to the therapeutic alliance/compliance are reduced. Second, meta-cognitive skills training could help to alleviate acute symptoms, as well as prevent future exacerbations, by changing the cognitive “infrastructure” of the delusion. Against the background of strong evidence that delusions are not only multi-faceted in form and content, but presumably share different causal underpinnings (Blackwood et al., 2001; Garety and Freeman, 1999), we have recently constructed (Moritz et al., 2005) a meta-cognitive training program which, apart from modules targeting a BADE deal with jumping to conclusions assesses attributional style and other reasoning biases that may promote the occurrence of psychosis.

## Appendix A

### Image 1

Pictures were successively presented to subjects on a computer screen

- seal
- person in winter clothing
- flower
- river scenery
- mermaid
- octopus
- harp
- seashell
- fish



exclusion, unlikely, possible, likely, **Decision**

- seal
- person in winter clothing
- flower
- river scenery
- mermaid
- octopus
- harp
- seashell
- fish



exclusion, unlikely, possible, likely, **Decision**

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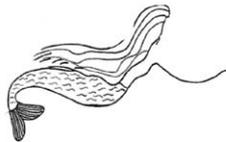
exclusion, unlikely, possible, likely, **Decision**

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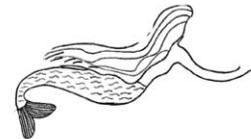
exclusion, unlikely, possible, likely, **Decision**

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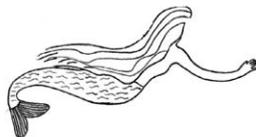
exclusion, unlikely, possible, likely, **Decision**

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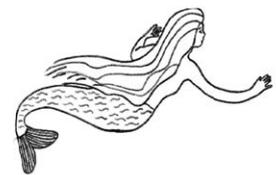
exclusion, unlikely, possible, likely, **Decision**

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exclusion, unlikely, possible, likely, **Decision**

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exclusion, unlikely, possible, likely, **Decision**

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