

Shorter communication

# An experimental investigation of the impact of biological and psychological causal explanations on anxious and depressed patients' perception of a person with panic disorder

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## Abstract

It is often suggested that biological accounts of the cause of mental health problems are beneficial in health education initiatives. However, an alternative view is that the idea of a diseased brain may result in stigma and therapeutic pessimism in sufferers, professionals and the public with implications for the perception of unpredictability and risk. Anxious and depressed patients ( $n = 49$ ) were randomly allocated to three experimental conditions. Prior to watching a video of a person suffering from panic disorder, participants were told either that research indicated that panic was caused by biological factors, by psychological factors or the cause was unclear (control condition). Those in the biological condition were significantly more pessimistic about the patient's prospects for recovery and rated risks as higher compared to those in the psychological condition. The results call into question the widely accepted practice of promoting biological/disease explanations of mental health problems.

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The grip of biological psychiatry on the mental health field was consolidated by the so-called “decade of the brain”. Presented both as health/scientific education and an attempt to reduce stigma, mental health professionals, the general public and people with mental health problems were encouraged to focus on the biological basis of mental health problems (<http://www.loc.gov/loc/brain/home.html>). This programme:

drew on the expertise of specialists and reported on progress in treating some of the most common brain disorders: “Depressive Illnesses”; “Schizophrenia”; “Developmental/Learning Disorders”; “Alzheimer's Disease”; “Anxiety Disorders.”

Those advocating this position argue that biological disease models are de-stigmatising and empowering relative to psychological models. Promoting the view that mental health problems have biological causes has become an established part of many programmes with the declared intention of reducing stigma, such as that

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of the National Alliance for the Mentally Ill (1996). Note, however, that the promotion of biological factors is not necessarily in the best interest of those with mental health problems and their families. For example, genetic factors may be promoted (or over-promoted) as causal with little regard for the potential impact that such ideas may have (Rimes & Salkovskis, 1998). Biological accounts may have the unwanted effect of inducing pessimism regarding likely treatment outcome in patients, their carers and the general public. Attributing mental health problems to brain disease additionally affect the extent to which sufferers are regarded as unpredictable, potentially antisocial or even dangerous by and to themselves and others. The brain is commonly understood to be the organ of the mind and the “seat of the self”; a diseased brain could mean a diseased mind and self.

Some previous research has suggested that the biological view of mental health problems may not be as beneficial as it has hoped for in modifying public attitudes (Read & Law, 1999; Phelan 2002; Hinshaw & Cicchetti, 2000). In a previous experimental study, we have shown that, in a community sample, the suggestion of “psychological” but not “biological” labels resulted in more positive views of mental health problems (Lam, Salkovskis & Warwick, 2005). Mental health professionals are probably also susceptible to biological labelling effects, although this is more difficult to assess in experimental studies because professionals tend to have already formed strong views in this respect once they qualify; these attitudes may therefore be harder to modify. The importance of clinician’s attitudes to causal factors in mental health problems lies both in their own expectations of patients’ response to treatment (Lam & Salkovskis, submitted) and in the extent to which their causal beliefs are transmitted to patients. The way in which patients understand and accept such causal explanations may in turn affect not only their expectation of change but also their engagement and response in treatment. Understanding how causal labelling might affect patients with mental health problems is thus an important next step in research terms.

There is little experimental research which investigates the extent to which causal accounts (biological and psychological) influence patients’ perceptions of mental health problems. However, ethical considerations make it difficult to justify manipulating patients’ perceptions of their *own problems* as this may have an adverse impact on their subsequent treatment. The present study therefore examined the reaction of non-panic anxious patients to an experimental manipulation of causal labelling applied to a video recording of a panic patient (who is seen describing her experience of panic and anxiety as part of assessment for treatment). Prior to watching the video the patients were randomly allocated to one of three sets of “authoritative” information (explicitly linked to research findings) about the causes of panic, with ratings made after the video being the dependent variables. It was hypothesised that participants given a biological causal attribution for panic would have a more pessimistic view of the prospects for treatment of the person seen on video relative to both controls and those given a psychological account. A weaker prediction was the psychological attribution would result in a more positive view than the control account. In a previous study of mental health professionals, labelling a patient seen on video as suffering from “borderline personality disorder” (Lam and Salkovskis, submitted), affected their clinical judgement not only in terms of their ratings of likely outcome but also on ratings of likely therapeutic process (motivation to change). In the present study, it was considered relatively unlikely that patients would identify process factors of this type, and it was therefore predicted that labelling effects would be confined to outcome expectancies.

## Method

### *Design*

Forty-nine participants diagnosed as suffering from anxiety and depressive disorders were randomly allocated to one of the three experimental conditions prior to watching a video tape of a woman being interviewed about her panic attacks. All participants received the same background clinical details of the person’s experience of panic, but *different* descriptions of the status of research about the presumed underlying cause of panic disorder. In the control condition the causes were described as not yet entirely clear; in the two other conditions, biological or psychological causes were invoked. Participants were asked to rate their impressions and expectations based *exclusively* on the section of video that they had watched.

## Participants

Participants for this study were recruited from a clinic to which they were referred for psychological treatments. Forty-nine out of 60 participants approached agreed to take part in the study prior to treatment starting, giving the response rate of 82%. Their ages ranged from 21 to 70 (mean = 41.63). Twenty were male and 29 were female. Their diagnoses were obsessive-compulsive disorder ( $n = 7$ ), post-traumatic stress disorder ( $n = 5$ ), health anxiety ( $n = 7$ ), depression ( $n = 27$ ), and general anxiety disorder ( $n = 3$ ). No patients with panic disorder or agoraphobia were included. Table 1 shows the characteristics of the experimental groups.

## Procedure

The aim of the study was explained in a letter and information sheet sent prior to consent being obtained. The precise purpose of the study was not given in order to minimise demand factors. Participants were told that the aim of the investigation was to examine how people viewed patients with panic disorder and their expectations of the treatment outcome. They were given an option of completing the initial questionnaire either at home or in the clinic. Once consent had been obtained, participants were randomly assigned (using sampling without replacement) to one of the three experimental background information conditions about a patient in a video. Randomisation gave rise to 16, 17, and 16 in control, psychological and biological conditions, respectively. Participants were asked to read the background information immediately before watching the video. The video was a 10-min 30 s extract from a first assessment interview of an actual patient with panic disorder with agoraphobia. Participants then completed the patient assessment questionnaire. Finally, participants were asked to describe what they thought the experiment was about, in order to assess whether they had an awareness of the experimental manipulation (none did).

## Experimental manipulation

The experimental manipulation was embedded in the background information given prior to the video. Three variants of background information relating to the patient's experiences of panic attacks were introduced in the written preamble to the video.

Table 1  
Means and (standard deviations) for the three groups

Group	Psychological	Biological	Control
Sample size	17	16	16
Mean age	40	43.25	42.75
Gender ratio (male/female)	6:11	5:11	9:7
Ratio of educational level (above degree/degree/below degree)	1:4:12	3:6:7	0:10:6
Prognosis following treatment (comp)	80 (16.2)	60.94 (24.9)	60.38 (22.7)
Expect duration of treatment (single)	19.05 (6.23)	36.81 (14.67)	23.56 (7.92)
Need for help (comp)	58.53 (10.27)	61.87 (15.37)	65.62 (14.59)
Risk of harming self or others (comp)	10.59 (8.05)	21.04 (20.49)	11.46 (11.29)
Disabling (single)	87.06 (11.6)	83.75 (12.58)	86.87 (10.78)
Motivation (single)	64.71 (26.1)	51.87 (24.82)	48.75 (24.46)
Dependency (single)	64.7 (26.0)	51.87 (24.82)	48.75 (24.46)

In the *control condition*, participants were provided with an accurate description of the patient's experiences of panic attacks, including a recent panic attack and general information about possible causes of the panic disorder.

Susan, aged 37, has been suffering from panic attacks for years; this has considerably restricted her social life. She is reluctant to go out alone and would often stay at home most of the time because of frequent panic attacks. Prior to most attacks, she experiences intense fear and anxiety. Her thoughts at that moment are that she is going to faint and pass out. During attacks, she has very unpleasant bodily sensations. Some of the sensations are: breathing very fast; feeling short of breath, as if she cannot get enough air; heart beating very fast; chest pain; shaking and trembling; and feeling faint and dizzy.

Neither drug therapy nor psychotherapy was previously effective. Because of her recurrent panic attacks, her General Practitioner is concerned with the nature of her condition and referred her for further investigation.

Her latest panic attack happened in a supermarket last Sunday. She felt dizzy and was having difficulty breathing. When the bodily sensations became intense, she grasped a chair to sit down. She felt relieved to be able to sit down just in time, believed that her action had just prevented her from fainting and passing out. Even with the support and company of a friend or a relative, she still experiences a high level of anxiety whenever she is in places such as supermarkets, parks and restaurants, etc. The condition has affected her life and daily functioning to the extent that she is now effectively disabled by her problem. *Research suggests that the causes of panic disorder are not yet entirely clear (emphasis not in original)*

In the *psychological condition* the same information as that in the control condition was used, but different information about possible causes of panic disorder was given:

There is now evidence to suggest that the recurrent panic attacks may be the result of some psychological processes relating to certain bodily sensations. Research indicates that panic disorder is probably a psychological based problem, involving patients thinking in a vicious circle that bodily sensations of anxiety are more dangerous than they really are and which make them more anxious. This further increases the bodily sensations and so on.

The word “psychological” was added (Paragraph 2, after “concern with the”).

In the *biological condition* the same information as those of the control condition was used, but the following possible cause of the panic disorder was given:

There is now evidence to suggest that particular parts of the brain are involved in the triggering of panic attacks. Research indicates that panic disorder is probably a biological condition and may be the result of genetic factors, involving biochemical imbalances. Part of the brain is overactive which causes anxiety to increase and trigger off bodily sensations and feeling of panic.

In addition, the word “biological” was added (Paragraph 2, after “concern with the”).

### Measures

The main measure used was the patient assessment questionnaire (PAQ). The PAQ is a modified version of the clinical assessment questionnaire (CAQ), designed to measure clinicians' clinical judgements (Lam & Salkovskis, submitted). The CAQ has good test–retest reliability ( $r = .86$ ). Modifications were mainly clarifications and simplifications. For example, “*stop coming for treatment*” was provided as an alternative meaning to “drop out”; “*get worse again after she has finished treatment*” to “relapse”; “*not going to situations which might make her anxious*” to “situational avoidance”. “Psychotherapy” was described in a way most consistent with cognitive behaviour therapy.

In the version used here, the PAQ consists of twelve 0–100 visual-analogue scales eliciting impressions about the patient seen on video and her panic disorder, where 0 and 100 indicated an extremely positive or negative view, depending on the nature of the scales. For example, in the measurement of view in the likelihood of the

patient's harming herself, 0 was "not at all likely" and 100 was "extremely likely". On the measurement of how well she would respond to therapy, 0 was "extremely well" and 100 was "not at all well".

The primary measure was a composite of the ratings for the likelihood of the patient being "cured" of panic disorder (3 items). In terms of the intensity of treatment likely to be needed, a second treatment composite included two items: whether hospitalisation and professional help are likely to be required. A secondary measure assessing the extent to which patient was regarded as likely to harm herself or others was also included, composed of likelihood of harming herself, harming others and attempting suicide. The degree of disability caused by the person's problem was also assessed. Treatment "process" items were ratings of the patient's motivation to change and her likelihood of becoming dependent on the therapist.

As this was a new version of the measure, test–retest reliability was done for this version of the scale. Eleven participants completed the questionnaire both immediately after the video and again after an hour had passed. Test–retest reliability was good, with the mean over twelve items being 0.82.

### *Treatment of data*

The questionnaire used in the study assesses the differential effects of biological and psychological explanations of panic disorder on patients' (participants) impressions about a patient being assessed for panic attacks and agoraphobia. Eight items from the questionnaire were categorised into 3 different groups for statistical analysis, based on how closely related the items were to each other. Prior to the main analysis, these composite items were each separately entered into repeated measures ANOVAs, with individual items as the repeats factor, and experimental groups as the grouping variable. These analyses were first used to check for group x item interactions; the clear lack of such interaction in each instance justified the use of the composites for the main (experimental groups) analysis. The composites used were: Prognosis following treatment, Risk of harming self or others and Need for help. Four ratings ("How disabling is her condition"; "how motivated to change is she"; "What do you expect the duration of this treatment to be?" and "Would she be likely to become too dependent on the therapist?") were analysed as single items using a one way ANOVA with experimental group as the between-subjects factor. Where main effects of group were detected, simple main effects and post-hoc Tukey LSD tests were carried out to find out which of the experimental groups differed from each other. The threshold for *post-hoc* tests was set to  $\alpha \leq 0.05$ .

## **Results**

Table 1 shows the means and standard deviations of the experimental condition for all single and composite variables analysed.

### *Prognosis following treatment*

This was the primary variable, involving three items in a composite. These items were: "How likely that she would be able to be free of panic attacks following treatment? How likely that she would be able to be free of situational avoidance (not going to situations which make her anxious) following treatment? How curable is her condition?" One way analysis of variance showed that there was a main experimental effect in this composite variable,  $F_{[2,46]} = 5.36$ ;  $p < 0.01$ . Multiple comparisons using Tukey tests indicated there that participants in the biological cause and control conditions did not differ, but both groups rated the patient as significantly less likely to make progress following treatment compared with those in the psychological cause condition.

### *What do you expect the duration of this patient's treatment to be?*

This variable is a less direct measure of response to treatment. One-way analysis of variance showed that there was a main effect of group in the measurement of how long would the patient's treatment be,  $F_{[2,46]} = 13.3$ ;  $p < 0.0001$ . Multiple comparisons using the Tukey LSD tests indicated that participants in the biological condition perceived the patient as likely to need a significantly longer period of treatment sessions

than those in either the psychological or control conditions. There was no difference in the perceptions of participants in the psychological and control conditions.

#### *Need for help*

This variable was intended to evaluate the extent to which participants believed the patient to be in need of more extreme treatment, and was a composite of ratings of need for intensive professional help and hospitalisation. No differences between experimental groups were noted; the main effect of experimental group was not significant  $F_{[2,46]} = 1.13, p > 0.3$ .

#### *Risk of harming self and others*

There were three items in this composite variable, which was intended to reflect judgements of disturbance of “self”. These items were: “How likely is she to harm herself? What is the likelihood of her attempting suicide? How likely is it that she would harm others?” One way of analysis of variance showed that there was a significant main experimental effect in this composite variable,  $F_{[2,46]} = 3.43; p < 0.05$ . Multiple comparisons indicated that participants in the biological cause condition regarded the patient as having a higher risk of harming self and others than those in the psychological cause and control conditions, who did not differ from each other (Table 1).

#### *Disability*

The main effect of group for this variable was not significant  $F < 1$ .

#### *Motivation to change and dependency on therapist*

These two variables were intended to reflect easy-to-understand therapy process factors. There was no evidence of main effects of experimental group in either the rating of motivation to change ( $F_{[2,46]} = 1.9, p > 0.1$ ) or likelihood of dependency on therapist ( $F < 1$ ).

### **Discussion**

This study aimed to investigate the effects of biological and psychological explanations of the cause of panic disorder and agoraphobia on anxious and depressed patients' perception of a person suffering from Panic Disorder seen on videotape. As predicted, labelling panic disorder as a “biologically” caused problem adversely affected the patients' ratings of the likelihood of the person responding to treatment, and led to a significantly higher estimate of treatment time likely to be required. For both of these variables this was true only relative to the group allocated to the experimental condition in which a psychological account of causation was indicated. The same pattern of results was found for the “harm” ratings. No experimental effects were found for the rating of the need for more extreme variants of treatment, degree of disability the patient seen in the video was likely to be currently experiencing, and therapy “process” variables (motivation to change and dependency on the therapist).

The results in this sample of participants (clinically identified anxiety and depression) are consistent with previous findings in a healthy sample (Lam et al., 2005). In this previous study, participants were simply given general descriptions of a range of psychiatric/psychological problems attributed to biological, psychological or “unknown” causes. It was found that the biological account resulted in more negative ratings of a range of factors, including prognosis and the likelihood of self-harm. However, this study used only very general descriptions of the problems to be rated, making it possible that the effects of the causal manipulation operated only at an abstract level, and would not have an impact when applied to an identifiable. The use of a video tape of an actual patient in the present study indicates that the previously noted impact of “labelling” is not a purely abstract effect, but can affect the perception of a real person suffering from anxiety. This has implications not only for stigma (e.g. perceptions of dangerousness) but also for help seeking, given the

evidence that the perception of causes and anticipated prognosis of a disorder may influence help seeking (Angermeyer, Matschinger, Riedel-Heller, 1999).

There is already both correlational and experimental evidence suggesting that that biological and genetic causal beliefs are related to negative attitudes in the general public. The most consistent findings concern perceptions that ‘mental patients’ are dangerous, antisocial and unpredictable (Green, McCormick, Walkey, & Taylor, 1987; Riskind and Wahl, 1992; Nunnally, 1961); that patients may take a more passive role with mental health professionals (Hill and Bale, 1981); that such accounts may actually contribute to the continuing problem of stigmatisation of mental health problems (Walker & Read, 2002; Read & Harre, 2001; Crisp, 1999; Sayce, 2000) and that mental health problems may be associated with mental health professionals taking a more paternalistic role in treatment interventions. The present findings, in the context of previous publications, suggest that biological explanations of mental health problems may increase public, professional and patient perception of harm (self-harm and harming others) and result in more negative predictions regarding prognosis, whilst psychological accounts may have the opposite (destigmatising) effect.

Patients suffering from panic disorder were excluded from this study for ethical reasons; we sought to minimise the impact of the experimental manipulation on the participants’ perception of their *own* problems. Having established the effect of a biological account of the cause of a problem which they do *not* suffer from, future experimental studies need to establish the impact of biological explanations of a problem which the person actually suffers from themselves. That is, it is important to understand whether the way people think about the causes of the problems they are presently experiencing may adversely effect how they react to treatment. If it does, it then has to be determined whether these negative effects can be blocked or reversed.

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