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Circumstances of pain onset, blame, and adjustment in chronic pain

By

Heather Margaret Rawle

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Contents

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Thesis Abstract

Research has found that chronic pain patients who perceive their pain to have originally resulted from a specific event are more poorly adjusted than patients who perceive their pain to have arisen for no apparent reason. The first paper of this thesis explores these relationships and notes the lack of investigation into the possible reasons for such findings. It shows that research deriving mainly from the non-chronic pain literature indicates that blame for negative events may play a role in explaining the relationship between circumstances of pain onset and adjustment. Blaming others is more consistently related to poor adjustment. Therefore, the likelihood of other-blame in patients whose pain arises from a specific event, and the association between other-blame and poor adjustment in chronic pain patients is explored in more detail. Implications for research and clinical practice are considered.

The second paper of the thesis describes a study which aimed to explore the role of other-blame as a mediator between circumstances of pain onset and adjustment. It also aimed to explore the relationship between different types of blame and adjustment given the lack of previous research into such relationships in a chronic pain population. The mediating role of other-blame was not supported. However, other-blame for initial pain onset was much more common in the event-related pain onset group and was associated with post-traumatic stress symptoms. In addition, self-blame was associated with anxiety and the use of maladaptive coping strategies.

Literature Review Paper:

Circumstances of Pain Onset and Adjustment in Chronic Pain Patients: The Role of Blame

Heather M. Rawle

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Circumstances of pain onset and adjustment in chronic pain patients: the role of blame.

Heather Margaret Rawle

University of Southampton

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Address for correspondence:

H. Rawle, Training Course in Clinical Psychology, Psychology Department, University of Southampton, Southampton, SO17 1BJ, United Kingdom.

Circumstances of pain onset and adjustment in chronic pain patients: the role of blame

Abstract

Circumstances of initial pain onset are associated with varying adjustment in chronic pain patients. Patients who perceive their pain to have originally resulted from a specific event tend to be more poorly adjusted than patients who perceive their pain to have arisen for no apparent reason. Possible explanations for these associations have received little attention in the chronic pain literature yet further understanding would have implications for both clinical practice and research. Research in non-chronic pain populations has demonstrated various associations between blame for negative events and adjustment. This paper reviews such research and relates the findings to the chronic pain experience. Blaming other people for negative events is the most consistent type of blame to be related to poorer adjustment. Therefore, the discussion becomes focused upon the role of other-blame as a possible mediator between circumstances of pain onset and adjustment.

Circumstances of pain onset and adjustment in chronic pain patient: the role of blame

Chronic pain is a complex problem that influences and is influenced by a variety of factors. Research has shown that psychological factors play an important role in the chronic pain experience. Circumstances of pain onset are also shown to relate to varying adjustment (i.e., emotional distress, pain report, disability and treatment outcome) in chronic pain patients, but very little is written about possible explanations for this. In this review, it is proposed that blame for pain onset may affect adjustment in chronic pain patients. Much of the psychological literature on chronic pain discusses the role of cognition in the chronic pain experience but very little specifically considers the role that blame for initial pain onset may play. Therefore, the aim of this review is to consider the role of blame as a mediator in the relationship between circumstances of initial pain onset and adjustment.

The review is divided into three broad areas. Firstly, the area of chronic pain is introduced with particular reference to the impact of chronic pain on patients, the relationship between chronic pain and emotional distress, and the role of psychological factors in the multifactorial model of chronic pain. It is suggested that identifying factors that distinguish between chronic pain patients in terms of adjustment can indicate targets for psychological intervention. One such distinguishing factor – circumstances of pain onset – is discussed. It is shown that patients whose pain arises from a specific event may be more poorly adjusted compared to patients whose pain begins for no apparent reason. Blame for initial pain onset is suggested as one possible reason for this. The literature on blame and

adjustment is then introduced, noting the need for people to make attributions when threatening events occur. The term 'blame' is explored, followed by an overview of research findings regarding the relationship between different types of blame and adjustment. Blaming others is identified to be most consistently related to poorer adjustment. There follows therefore, an exploration of how other-blame may be more prevalent in patients whose pain arises from a specific event. Suggestions are made regarding how this may result in poorer adjustment in these patients. Finally, implications for research and clinical practice are considered.

Chronic Pain

This section introduces the problem of chronic pain, demonstrating the impact it can have on patients and its relationship with emotional distress. Psychological factors are considered to play an important role in the emergence and maintenance of the chronic pain experience and this is demonstrated by describing the fear-avoidance model of chronic pain.

Definition

Pain has been defined as 'an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (The International Association for the Study of Pain (IASP), 1986, p. S217). The IASP defines chronic pain as pain that continues beyond normal tissue healing time which is usually taken to be three months. Chronic pain can be triggered by injury or illness but frequently an organic basis to the pain cannot be found. Furthermore, chronic pain is often unsuccessfully treated by medical interventions. Acute pain can become

chronic as a result of factors other than the cause of the pain and these are discussed below.

The impact of chronic pain

Chronic pain is an important area to research given the financial and personal costs that result. It is a major health problem in Western Societies and carries a high economic cost in terms of health care, informal care, and production losses (Maniadakis & Gray, 2000). In terms of personal costs, chronic pain negatively affects the quality of life of patients and their families and can severely impair the patient's social, vocational and psychological well-being (IASP, 1986). For instance, roles at work, in the family, and in social settings may be affected, lifestyles may be altered, physical and social activity may be limited, social isolation may occur as patients withdraw from others, time off work and unemployment may continue, and financial insecurity result. Attempts to relieve the pain by the patient him/herself or by medical professionals often meet with failure and the legitimacy of the patient's ongoing complaints of pain may be questioned by others (Osborn & Smith, 1998). Therefore, it is easy to see how emotional and behavioural disturbances can result from the chronic pain experience. The types of emotional distress that have been associated with chronic pain include depression, anxiety, fear, anger, guilt and frustration (Conant, 1998; Craig, 1994; Gaskin, Greene, Robinson & Geisser, 1992; Kuch, Evans & Mueller-Busch, 1993; Okifuji, Turk & Curran, 1999).

The relationship between chronic pain and emotional distress

There has been much debate about the nature of the relationship between emotional distress and chronic pain (see Gamsa, 1994, for review). Longitudinal studies have demonstrated support for the hypothesis that emotional distress is more likely to result from chronic pain than precipitate it (Gamsa, 1990; Radanov, Begre, Sturzenegger & Augustiny, 1996). In addition, psychological disturbance has been found to be as great in patients with an organic basis to their pain as among patients who have no organic basis. This provides evidence against psychoanalytic (psychogenic) theories of pain which view pain as an expression of psychological difficulties (Benjamin, Barnes, Berger, Clarke & Jeacock, 1988). Others have found evidence for a 'shared-mechanisms' explanation (i.e., same biological aetiology for both chronic pain and depression) from longitudinal research (e.g., Breslau, Davis, Schultz, Edward & Peterson, 1994). Therefore, it seems that emotional distress can be related to chronic pain in a variety of ways. It can be a component, cause, consequence, and correlate of chronic pain and therefore, mulitfactorial models are necessary to explain the chronic pain experience. Some of the ways in which emotional distress and pain are related are explained by the fear-avoidance model, described below.

Multifactorial models of chronic pain

Linear causal models of pain that considered pain in terms of either physical or psychological explanations have given way to multicausal explanations that include both physical and psychological influences. Melzack & Wall's (1965) gate-control

theory of pain integrated physiological and psychological factors to explain the pain experience. In its simplest form, the theory proposes that a series of gates exist in the spinal cord through which pain messages pass from all over the body to the brain. If the gates are more open, then more pain messages pass through to the brain and the person experiences more pain. Various factors are thought to be involved in the opening and the closing of the gates, influencing the pain experience. For instance, psychological factors that are proposed to open the gates include stress and focusing on the pain. Psychological research has now made a significant contribution to the understanding and treatment of pain. Chronic pain is now considered to be a complex, multidimensional experience including sensory, motivational, cognitive, attentional, and emotional elements.

Various psychological theories have been suggested to explain the chronic pain experience and provide a focus for intervention, for example, behavioural theories (Fordyce, 1976) and psychodynamic theories (e.g., Engel, 1959). Cognitive approaches are well researched in the pain literature. These consider the influence of factors such as attributions, beliefs, self-efficacy, expectations, attention, control, and coping strategies, on the pain experience and related problems (see Weisenberg, 1994, for review). One model drawing from both cognitive and behavioural theory is the fear-avoidance model.

The fear-avoidance model explains how pain-related fear can lead to increased pain, distress and disability. It is elaborated upon now as it clearly explains the emergence and maintenance of problems related to the chronic pain experience, and has been well supported by research (see Vlaeyen & Linton, 2000). First, pain-related

fear is thought to arise from interpretations of the pain as threatening (i.e., catastrophising). The fear leads to escape and avoidance behaviours so that everyday activities (expected to produce pain) are avoided, leading to increased disability. Persistent avoidance and physical inactivity may further worsen the pain problem by detrimentally affecting the musculoskeletal and cardiovascular systems (Bortz, 1984). Avoidance of activities also may lead to distress such as depression, frustration and irritability as a result of a lack of reinforcement opportunities. Depression and reduced activity have been related to decreased pain tolerance (Romano & Turner, 1985) which may worsen the pain experience. Also, pain-related fear, as with other types of fear, can interfere with cognitive functioning. Patients may be hypervigilant to threatening stimuli and less able to divert attention from pain-related information (Asmundson, Kuperos & Norton, 1997). This would reduce the use of more adaptive coping strategies (McCracken & Gross, 1993). The disability, lower activity, and distress would maintain the cycle, increasing fear and avoidance.

Research into the relationship between chronic pain and adjustment can be criticised for focussing too much on simply describing associations rather than considering mediating or moderating factors. Recent research has shown promise in identifying mediators of the relationship between pain intensity and distress (usually depression) such as 'subjective future' (Hellstrom, Jansson & Carlsson, 1999) and self-efficacy (Arnstein, Caudill, Mandle, Norris & Beasley, 1999). Research should also ask whether there are certain characteristics of chronic pain patients or their circumstances that help to explain differences in their presentation. Identifying factors that distinguish patients who adjust well to their pain compared to patients who do not

can indicate areas for psychological intervention. One area that has demonstrated such differences is distinguishing patients according to the circumstances that led to initial pain onset.

Circumstances of Pain Onset and Adjustment

Types of onset

Patients' descriptions of the circumstances of their pain onset generally fall within one of two broad categories – event and non-event. Specific events that may lead to pain include accidents (at home, at work, or on the road), illness or surgery. Alternatively patients may be unable to identify a specific event and report that their pain arose for no apparent reason.

Onset type and adjustment

Despite the obvious distinctions apparent in circumstances of pain onset, only in recent years has chronic pain research considered how they relate to adjustment. Overall, adjustment appears to be worse for patients whose pain initially arose following a specific event compared to patients whose pain began for no apparent reason (Greenfield, Fitzcharles & Esdaile, 1992; Toomey, Seville, Finkel, Mann, Abashian & Klocek, 1997; Tsushima & Stoddard, 1990; Turk & Okifuji, 1996; Turk, Okifuji, Starz & Sinclair, 1996). However, results are not entirely consistent. Of those who considered pain severity, it was found to be elevated in event-related pain onset groups in the studies by Turk & Okifuji (1996) and Turk *et al.* (1996) but not by Toomey *et al.* (1997). When elevated emotional distress has been found in event-related pain onset groups, measures of generalised distress have tended to be used

(e.g., Toomey *et al.* 1997; Turk & Okifuji, 1996; Turk *et al.*, 1996). When researchers have considered depression in particular, no differences have been found (Turk & Okifuji, 1996; Turk *et al.*, 1996). Other indicators of adjustment have been considered by some researchers and been found to be worse in the event-related groups. These include the employment of a more diffuse and over inclusive pain reporting style, more pain-related job changes (Toomey *et al.*, 1997), more life interference (Turk *et al.*, 1996), and more frequent pain and a poorer prognosis (Tsushima & Stoddard, 1990). Other studies have failed to support these findings (e.g., Kuch, Evans, Watson & Bubela, 1991; Waylonis & Perkins, 1994). However, Turk *et al.* (1996) point out that careful scrutiny of Waylonis & Perkins' data do reveal specific factors that are uniquely related to the traumatic onset type.

Post-traumatic stress. Event-related pain onset patients may also be more likely to have post-traumatic stress (PTS) symptoms. Much research using non-chronic pain samples has shown that psychological disturbance, particularly PTS symptoms, can occur following the types of events that lead to chronic pain. Such symptoms can persist for years after the event (Mayou, Tyndel & Bryant, 1996). PTS symptoms can occur in response to the stress of a severe illness (Chemtob & Herriott, 1994); a one off life-threatening illness event such as myocardial infarction (Bennett & Brooke, 1999); road traffic accidents (RTAs) (Blaszczynski, Gordon, Silove, Sloane, Hillman & Panasetis, 1998); surgery and other medical accidents (Church & Vincent, 1996); accidental falls and sports injuries (Feinstein & Dolan, 1991); childbirth (Czarnocka & Slade, 2000); and work-related injury (Asmundson, Norton, Allerdings, Norton & Larsen, 1998).

Researchers using chronic pain patients whose pain followed an accident (usually RTAs) have also noted high levels of PTS symptoms and depression (e.g., Blanchard, Hickling, Taylor, Loos & Gerardi, 1994; Chibnall & Duckro, 1994; Hickling, Blanchard, Silverman & Schwarz, 1992; Kuch *et al.,* 1991). Others have found that the level of PTS symptoms in patients whose chronic pain had arisen from accidents helped to explain differences in distress and pain (Geisser, Roth, Bachman & Eckert, 1996). Patients who had high PTS symptoms and whose pain resulted from an accident reported more distress (including depression) and pain intensity than other patients.

Research limitations

The above overview demonstrates that in general, patients whose pain occurs as a result of a particular event have higher levels of psychological distress (including PTS symptoms), pain, and disability, than patients whose pain 'just begins'. Such results are apparent even when a more inclusive definition of event-related pain onset is employed to categorise patients (i.e., including illness and surgery as well as accidents) (Toomey *et al.*, 1997). However there are some limitations to the research.

The generalisability of conclusions to the general population of people with chronic pain is limited as research tends to use only new patients drawn from pain clinic samples (see Crombie & Davies, 1998, for limitations of pain clinic samples), and homogeneous samples (e.g., all fibromyalgia by Turk *et al.,* 1996; all headache by Tsushima & Stoddard, 1990). Generalisability is also limited by some researchers excluding certain types of pain onset such as illness and surgery (Turk *et al.,* 1996).

Also, different researchers have categorised circumstances of pain onset in different ways. This makes comparisons of studies difficult and may explain inconsistent findings. To complicate matters, studies may use the same labels to describe a broad type of onset, but these labels may have different meanings. For example, three studies have used the term 'post-traumatic onset' to define pain following accidents, illness and surgery (Toomey *et al.*, 1997), accidents and surgery (Turk & Okifuji, 1996), and accidents only (Turk *et al.*, 1996). Furthermore, not all studies included all types of chronic pain onset and it is unclear in some studies which types of onset were included, and in which category they were included (Geisser *et al.*, 1996; Kuch *et al.*, 1991; Turk & Okifuji, 1996). Only Toomey *et al.* (1997) included all types of onset in their broad categories and made it clear which category included which onset type.

Why are circumstances of pain onset associated with adjustment?

The aforementioned research findings are not easily predicted. For instance, it may be expected that people who are unable to identify a precipitant to their pain onset would report more, not less, psychological distress. Such patients may face skepticism and hostility from others if doctors are unable to attribute symptoms to a physical origin. The Pain Beliefs and Perceptions Inventory (PBPI) (Williams & Thorn, 1989) assesses various aspects of patients' beliefs about their chronic pain and includes a 'mystery' scale which is particularly relevant to patients whose pain does not follow from a specific event. This scale taps into the patient's belief that their pain is an unexplained mystery and differs from the culturally shared belief that pain serves a useful warning function. High scores on the mystery factor have been associated

with higher distress scores (anxiety and depression), lower self-esteem, higher levels of somatisation, and poorer treatment outcome (Williams & Keefe, 1991; Williams *et al.*, 1994; Williams & Thorn, 1989). The researchers explain these results by suggesting that people who believe their pain to be an unexplained mystery are not achieving the adaptive goal of finding a reason for their misfortune or they may have lower perceived control over their pain.

Proponents of the psychogenic model of chronic pain would also expect patients with pain of a spontaneous origin to have greater psychopathology (e.g., Sivik & Delimar, 1994). However, there are difficulties with studies supporting psychogenic models as they have frequently used measures that are confounded with physical disability, therefore inflating scores (Watson, 1982).

More severe physical pathology. It could be proposed that differences between groups occur as the event-related pain onset patients may have more severe physical pathology. However, research has shown that these groups do not differ in terms of extent of physical pathology (Turk & Okifuji, 1996; Turk *et al.*, 1996) and that in addition, physical pathology does not correlate well with pain severity and disability (e.g., Waddell, 1987).

Compensation. Perhaps one reason for poorer adjustment in patients whose pain follows an event is due to this group being more likely to be involved in receiving or seeking compensation. These factors have long been considered to be associated with poor adjustment (e.g., Miller, 1961). However, again, research has found that differences between types of onset groups remain despite controlling for the possible confounding variable of compensation (Turk & Okifuji, 1996).

Fear-avoidance. The fear-avoidance model helps to explain poorer adjustment in patients whose pain results from a specific event as research suggests that fear-avoidance beliefs are particularly salient for these patients (Crombez, Vlaeyen, Heuts & Lysens, 1999). It may be that patients fear re-injury during activities that remind them of the event that initially triggered pain onset. Such fears are symptoms of PTS (i.e., manifested as avoidance of stimuli that reminds the person of the traumatic event) which have been noted to occur in patients whose pain follows a particular event. Furthermore, event-related pain onset patients are likely to have experienced sudden onset of pain. This may accelerate the fear-avoidance cycle as a result of abrupt decreases in patients' activity. The experience of a traumatic injury may also change how patients evaluate sensory information. Therefore, such patients may be even more hypervigilant to bodily symptoms and pre-occupied with physical sensations. They may also be more likely to expect pain given that they can identify an injurious event and therefore be more likely to perceive bodily sensations as pain (Turk & Okifuji, 1996).

Blame. Cognitive-behavioural models such as the fear-avoidance model suggest that patients' beliefs about their pain can affect their pain experience and influence adjustment. Patient's beliefs about their illnesses have long been suggested to account for differences in adaptation to an illness (Moos, 1977). More generally, research considering attributions has often shown them to be an important influence on the impact of various life stresses (e.g., Alloy, Just & Panzarella, 1997). Chronic pain research has tended to investigate the role of cognitions regarding the ongoing pain experience (e.g., pain locus of control, pain attributions, beliefs about treatment)

(e.g., Jensen, Turner, Romano & Lawler, 1994). However, in the light of research findings regarding the influence of circumstances of pain onset on adjustment, it also seems important to consider the role of beliefs about the aetiology of the pain.

A small number of researchers have speculated whether patients whose pain arises following a specific event have a target to blame and that it is this blame that interferes with adaptation to symptoms (Turk *et al.*, 1996). Indeed, DeGood & Kiernan, 1996) have specifically investigated the perception of fault for pain and found evidence to suggest it may be a possible mediator of adjustment. Church & Vincent (1996) also found that blaming others for injuries resulting from medical accidents (e.g., surgery, diagnosis failure or incorrect treatment) was very common and associated with greater psychological distress. Although this study did not use a chronic pain sample, the nature of events causing injury is relevant for many pain patients.

Summary

Psychological factors play a major role in the emergence and maintenance of chronic pain and chronic pain can have a significant emotional and behavioural impact on patients. Circumstances of initial pain onset, i.e., event or non-event related, appear to have a differential impact on adjustment. However, previous literature has paid little attention to the possible explanations for this finding. One possible explanation that is worthy of further exploration is that circumstances of pain onset may be related to certain types of blame for pain onset. Perhaps blame plays a role in accounting for some of the differences in adjustment.

Blame

In this section, the role of blame attributions in adjustment is considered in more detail. On the whole, blaming others for negative events has been related to poorer adjustment. Most research focuses on blaming oneself for negative events but this has been related less consistently with adjustment, sometimes showing worse adjustment and sometimes better adjustment. Inconsistent findings have also arisen from the small amount of research that has considered other types of blame such as blaming chance or the environment. Only one study could be found in a literature search that specifically considers the relationship between blaming <u>others</u> for chronic pain and adjustment (DeGood & Kiernan, 1996). This literature search was conducted using three computer abstracting services: Psychlit, Social Sciences Citation Index (SSCI), and Medline. The search strategy contained the word 'pain' and five relevant words ('blame', 'responsibility', 'cause', 'fault', or 'attributions'). Reference lists and bibliographies were also searched from all retrieved articles.

When blame is mentioned elsewhere in the chronic pain literature, it usually refers to 'self-blame' as a coping strategy for ongoing pain, rather than beliefs about pain onset, and the association with adjustment is usually mentioned with little discussion.

Research into blame and adjustment, and research limitations, are considered in more detail later. Firstly, the terms 'attribution' and 'blame' are introduced.

Attributions

It may be asked why people end up blaming something when they experience a negative event. Attribution theory is concerned with the process whereby people explain their behaviour and the behaviour of others (Heider, 1958; Kelley, 1967). The term 'attribution' refers to inferences that people make about the causes of events or states of being (Michela & Wood, 1986) and attribution research is often concerned with the resulting behaviours and affects. It is this line of investigation which is the focus of the present review.

The main theoretical views and original research regarding attributions come from Weiner (1974) who found causal attributions to be typically classified along the dimensions of locus (whether the cause is internal or external to the person), stability (whether or not the cause is changeable) and controllability (whether or not the cause is under the person's control). One of the assumptions of attribution theory is that people are motivated to seek causal explanations in order to understand, predict and control their environment i.e., the process of making causal attributions is adaptive (Wong & Weiner, 1981). It has been suggested that blaming others or oneself are particular examples of making attributions about external or internal causes (Sensky, 1997) and therefore it seems reasonable to suggest that conclusions regarding blame arise as part of the causal search.

Research suggests that attributional search is most likely when events are unexpected, negative, and highly salient to the individual (Weiner, 1985). For all chronic pain patients, their pain onset is likely to meet all of these criteria. Indeed, recent qualitative research has highlighted the importance of searching for an

explanation for chronic pain by both patients and health professionals, in order to provide the experience with some meaning (Eccleston, Williams & Stainton-Rogers, 1997; Osborn & Smith, 1998). Extensive research into people's interpretations of general illness symptoms has found that people commonly hold beliefs about the causes of their illness and such beliefs have implications for treatment (Lau & Hartman, 1983; Leventhal, Nerenz & Straus, 1980).

What is blame?

There is some debate in the literature regarding the definition of blame. Shaver (1985) devised a theory of blame assignment of which a major assumption is that there are conceptual distinctions between 'cause', 'responsibility' and 'blame'. Shaver asserts that blame of another person only occurs when cause and responsibility have been established, and the justification or excuse of the offending person is not accepted because the outcome was believed to be intentional. Some of Shaver's ideas are based on empirical studies but these are limited. The number of studies is small and most of them are experimental using hypothetical scenarios, forced choice measures, and with samples consisting of students.

According to Shaver, much of the research into the relationship between 'blame' and adjustment does not measure blame at all (see Shaver & Drown, 1986). For instance, Shaver's theory suggests that one can only assign blame to humans, blame cannot be assigned following 'accidents', and asking participants about 'causality' or 'responsibility' does not enable conclusions to be drawn about 'blame'.

Shaver's emphasis on conceptual distinctions are shared by some researchers (e.g., Brems & Wagner, 1994; Harvey & Rule, 1978; Lussier, Sabourin & Wright, 1993; Rich, Smith & Christensen, 1999). However, other researchers have found evidence that in reality, people do not tend to distinguish between the concepts (e.g., Nikcevic, Tunkel, Kuczmierczyk & Nicolaides, 1999; Sholomskas, Steil & Plummer, 1990). However, the latter researchers used less sophisticated measures of blame and responsibility and this may account for the discrepant findings.

With regards to Shaver's assertions about the circumstances when blame can be assigned, it is important to remember that the theory is of how blame should ideally be assigned and not how people assign blame in real life. Even if the theory was correct, in real life people may make errors when assigning blame. For instance, using semi-structured interviews, Church & Vincent (1996) found that half of their sample of people with injuries following medical accidents blamed the hospital. It therefore seems important for research relevant to chronic pain to allow the investigation of 'real-life' blame despite the existence of theories that question the validity of such 'blame'. Shaver & Drown (1986) themselves remark that 'the manner in which blame is inaccurately applied by victims may prove valuable in understanding their emotional adjustment' (p. 701).

Given the proposed conceptual distinctions, it is important to note that the use of the word 'blame' in the current review relates to the construct that studies stated they were investigating even though their measures may suggest otherwise (e.g., when measures refer to the 'cause' of the negative event).

The role of blame in adjustment

Research samples, adjustment, and blame types. The relationship between blame and adjustment has been investigated in a variety of medical populations such as spinal cord injury (Brown, Bell, Maynard, Richardson & Wagner, 1999), cancer (Malcarne, Compas, Epping-Jordan & Howell, 1995), miscarriages (Nikcevic *et al.*, 1999), and renal failure (Rich *et al.*, 1999). Blame has been investigated in relation to specific experienced events (e.g., spinal cord injury, Brown *et al.*, 1999), ongoing difficulties (e.g., illness problems related to end-stage renal disease, Rich *et al.*, 1999) and hypothetical situations (e.g., Stewart, 1999). In the current paper, the research reviewed mainly focuses on that which investigates the relationship between adjustment and blame for real events that happened to the person being questioned.

Measures of 'adjustment' have included those of global and specific emotional distress (e.g., depression, anxiety, anger), various measures of coping, illness specific outcomes, and treatment outcomes. A few studies have also investigated the role of blame in PTS symptoms. The types of blame attributions that have been the main target of investigation in such research have been mostly self-blame, and to a lesser extent, blame of other people. Research has also mentioned other sources of blame such as the environment, chance/luck/fate, and God.

Other-blame and adjustment. Most research into the relationship between blame and adjustment has focussed on self-blame. Blaming others is usually either ignored altogether in these studies or mentioned as an incidental finding. Therefore relatively little is known about factors that influence the relationships between other-blame and adjustment. Such a neglect of other-blame is surprising given the frequent

(but usually incidental) finding that it is associated with poorer adjustment (e.g., Bulman & Wortman, 1977; Church & Vincent, 1996; Czarnocka & Slade, 2000; DeGood & Kiernan, 1996; Derry & McLachlan, 1995; Sholomskas *et al.*, 1990; Taylor, Lichtman & Wood, 1984; Timko & Janoff-Bulman, 1985).

In a review of the literature on the relationship between blaming others for threatening events and adjustment, Tennen & Affleck (1990) considered studies that measured people's responses to a wide range of stressful events, many of them related to medical events. They concluded that blaming others is consistently related to poorer emotional well-being and physical health. Such relationships were found in over three quarters of the 22 studies. The findings were relatively consistent despite the fact that the studies included different stressful events, varied in the timing between events and questioning, and varied in the outcomes and the techniques of measuring the outcomes and blame attributions. The authors recommended a shift in research focus from self-blame to other-blame. Such a shift does not appear to have occurred in the decade following this recommendation despite the continuing conflicting findings related to self-blame.

The most relevant study in the current discussion regarding blame in chronic pain patients was that undertaken by DeGood & Kiernan (1996). The study found that patients who perceived another person to be at fault for their pain reported more global and specific concurrent emotional distress, more behavioural disturbance, poorer response to past treatments, and fewer expectations of future benefits, than patients who faulted 'no-one'. There were no differences between groups in terms of pain severity or activity limitation. A limitation of this study is that it did not include

blame of other factors such as the self, environment, or chance. The study also used the word 'fault' when questioning patients and assumed this meant blame when reporting the results, and did not determine the psychometric properties of the 'fault' measure. The focus of the questioning was on 'fault for pain' rather than specifically for 'pain onset' and as yet, it is unclear whether patients distinguish between these and the impact this may have on responses. Also, the use of a forced choice method did not allow for the possibility that patients may attribute blame to more than one source or consider varying degrees of blame (as has been found by studies employing alternative methods, e.g., Reidy & Caplan, 1994; Sholomskas *et al.*, 1990).

Since Tennen & Affleck's (1990) review, not all research has demonstrated a relationship between other-blame and poorer adjustment (Brown *et al.*, 1999; Reidy & Caplan, 1994). However, none can be found that suggests other-blame is related to better adjustment.

Self-blame and adjustment. Many investigations have specifically aimed to explore the relationship between self-blame and adjustment, but findings are inconsistent. The increased interest in self-blame research can be attributed to a study by Bulman & Wortman (1977) who found self-blame (for spinal cord injury) to be associated with better coping. Many researchers since then have found self-blame to be related to better adjustment in a variety of samples (e.g., Brewin, 1984; Derry & McLachlan, 1995; Timko & Janoff-Bulman, 1985). However, conversely, specific attempts to replicate Bulman & Wortman's study (using similar samples and measures) have found self-blame to be associated with poorer coping (e.g., Nielson & MacDonald, 1988) and found no associations with coping (e.g., Sholomskas *et al.*,

1990). Other studies employing different samples and different assessment methods have also found self-blame to be associated with poor adjustment (Berckman & Austin, 1993; Houldin, Jacobsen & Lowery, 1996; Kiecolt-Glaser & Williams, 1987; Reidy & Caplan, 1994) and to be unrelated to adjustment (Miller & Porter, 1983; Witenberg, Blanchard, Suls, Tennen, McCoy & McGoldrick, 1983). Further, self-blame has been found to be related to both better and poorer coping in the same sample, depending on the point in time that participants were investigated (Taylor *et al.*, 1984).

Inconsistencies in findings regarding the relationship between self-blame and adjustment have been explained in terms of perceived control. Two types of self-blame have been suggested to exist which theoretically may have a differential impact on adjustment (Janoff-Bulman, 1979). Behavioural self-blame (blaming one's behaviour), which is situational and considered to be modifiable, has been suggested to be adaptive. This is because it may be associated with a belief in future avoidability of a negative outcome, and therefore enhance a sense of control over the event's recurrence. Characterological self-blame (blaming one's character), which is considered to be global and unchanging and therefore non-modifiable, is suggested to be maladaptive as such control over a recurrence is not possible. The link between characterological self-blame and poorer adjustment can be predicted by the reformulated learned helplessness model (Abramson, Metalsky & Alloy, 1989) which associates internal attributions with low self-esteem and depression. Also, self-blame is considered to be the core relational theme for guilt in appraisal-emotion theories. Although frequently mentioned in the literature, it is debatable whether people

distinguish between characterological and behavioural self-blame, and even when they do, the utility of the distinction has not always been supported empirically (e.g., Nielson & MacDonald, 1988; Sholomskas *et al.*, 1990).

The chronic pain literature has sometimes mentioned the role of self-blame for the chronic pain experience in adjustment as part of more general studies which use measures of pain beliefs or coping that include a self-blame factor. For instance, the Ways of Coping Checklist (Vitaliano, Russo, Carr, Maiuro & Becker, 1985) includes a self-blame scale which has been associated with increased distress in chronic pain patients (Jensen, Turner, Romano & Karoly, 1991). The Pain Beliefs and Perceptions Inventory includes a self-blame dimension which has been associated with depression symptoms (Kiecolt-Glaser & Williams, 1987; Williams, Robinson & Geisser, 1994; Williams & Thorn, 1989) but also with increased physical functioning. These apparently conflicting results have been explained by suggesting that self-blame may motivate people to be over-active which eventually leads to decreased physical functioning, increased pain, and depression (Williams *et al.*, 1994).

Other types of blame. Besides self- and other-blame, some researchers have considered additional sources of blame such as the environment, chance, and God (e.g., Bulman & Wortman, 1977; Downey, Silver & Wortman, 1990; Reidy & Caplan, 1994; Taylor *et al.*, 1984). Results are generally inconsistent regarding the relationship between adjustment and these types of blame, with many studies finding no relationship at all.

Blaming chance and the environment (and sometimes God) have usually been included in research using spinal cord injured patients but many other studies have

failed to consider these sources of blame. It seems important however to include these types of blame given that research has sometimes found them to be more prevalent than self- and other-blame (Brown *et al.*, 1999; Bulman & Wortman, 1977; Heinemann, Bulka & Smetak, 1988; Reidy & Caplan, 1994).

Research limitations

Explanations regarding inconsistent findings in the literature include differences in: the type of event studied, the outcome measures used, the operationalisation of blame, the measures of blame, and the failure to distinguish between blame for the initial event versus blame for subsequent events. Inconsistencies in conclusions could also be explained by the differences in amount of time elapsed since the event in question. Some longitudinal research has suggested that the influence of blame on adjustment diminishes with time (Brown *et al.*, 1999; Richards, Elliott, Shewchuk & Fine, 1997; Schulz & Decker, 1985). However, others have found evidence against this (Derry & McLachlan, 1995; Reidy & Caplan, 1994). In Tennen & Affleck's (1990) review, studies varied greatly in terms of all of these factors, yet still found the findings related to other-blame to be consistent. It may be that the reasons for inconsistencies apply more to the self-blame research (which has indeed been more inconsistent in findings).

General problems with research in this area include the frequent use of small samples (e.g., around 25) and the employment of correlational/cross-sectional designs which do not allow for conclusions to be drawn regarding causal links between blame and adjustment. Only recently have a few prospective studies emerged that have provided evidence for a temporal link between blame and adjustment (e.g., Czarnocka

& Slade, 2000; Derry & McLachlan, 1995; Rich *et al.*, 1999). There is evidence from experimental studies to support a causal influence of attributions on adjustment (McFarland & Ross, 1982). However, it is possible that physical and psychological health outcomes lead to blame attributions or that blame co-occurs with distress, but plays no causal role (Downey *et al.*, 1990).

Another major problem with such research relates to measures of 'blame'. Most researchers design their own measure that specifically relates to the event being investigated, and they typically use a single item to assess each type of blame. Therefore, various methods have been used including rating scales, open ended questions, and spontaneous reports. However few researchers investigate the measures' psychometric properties. In addition, it is not clear in some research how blame was assessed (e.g., Church & Vincent, 1996). A small number of researchers have designed measures to assess a general tendency to blame and have tested psychometric properties (e.g., Gudjonsson & Singh, 1989; Rich *et al.*, 1999). However, these measures are not suitable in their present state for use in studies that consider blame for a specific event (e.g., initial pain onset).

More important however, is the concern that researchers who claim they are assessing blame may not be doing so at all. Often, researchers ask participants about 'responsibility' (e.g., Downey *et al.*, 1990; Frey, Rogner, Schuler, Korte & Havemann, 1985; Taylor *et al.*, 1984) or 'cause' (e.g., Brown *et al.*, 1999; Derry & McLachlan, 1995) which are then subsequently equated with blame. The lack of attention to construct validity in the measurement of causality, responsibility and blame has been raised (Shaver & Drown, 1986). These authors note that judgements of blame may

carry affective connotations, whereas judgements about responsibility and cause may be made with a degree of detachment. The issue of whether blame is being investigated, rather than the other related but distinct variables, clearly needs addressing. Whilst there is evidence to suggest that people respond in similar manners regardless of whether the word 'blame' was used in the question, it still seems reasonable to conclude that the answers will depend on how the question is asked (McArthur, 1972).

Finally, some researchers have criticised studies for relying on subjective, retrospective assessments of blame (e.g., Trieschman, 1988) and state that an assessment of 'actual' blame is necessary. However, as mentioned by Reidy & Caplan (1994), ruminations about blame are part of an individual's psychological reality (regardless of actual circumstances) and therefore are worthy of investigation in terms of their relationship with adjustment.

Summary

The first part of this review proposed that blame for pain onset might account for some of the differences in adjustment found by research investigating the impact of circumstances of pain onset on adjustment. Then it was demonstrated that empirical findings show that people are motivated to search for explanations following negative events, and that this is a common process for chronic pain patients. Thoughts about blame may form part of this search. There is some debate over the concept of blame but it seems important to investigate 'real-life' blame and to ensure that future research distinguishes between blame and the related concepts of causality and

responsibility. Very little research has specifically investigated the role of blame for pain onset in adjustment in chronic pain patients. Most research outside the chronic pain literature investigates the role of self-blame in adjustment but results are inconsistent. Other-blame is more consistently related to poorer adjustment but has been rarely investigated as the focus of study. Blaming chance and the environment also seem important types of blame to investigate given their prevalence.

Blame and Chronic Pain

The following discussion now returns to an exploration of circumstances of pain onset and their relation to varying adjustment in chronic pain patients (i.e., poorer adjustment in patients whose pain initially arises following a specific event). The above discussion demonstrates that poorer adjustment is more consistently related to other-blame than alternative types of blame. Therefore, it may be proposed that patients whose pain originates from a specific event are more likely to blame others and it is this increased blaming of others that contributes towards the findings of poorer adjustment in these patients.

Other-blame in event-related pain onset patients

The proposal that other-blame may be a particular factor associated with patients whose pain arises from a specific event is supported by a model of the relationship between other-blame and adjustment (see Tennen & Affleck, 1990). Tennen & Affleck (1990) proposed that other-blame is more likely to occur when: the event is a one-off occurrence; someone else is present at the time of the event (and is therefore

available to blame); the person blamed is in a position of authority, or has relevant skills or knowledge (but this person does not necessarily have to be present at the time of the event); the person blamed is not well known to the person attributing blame; and the outcome is severe. These conditions are likely to be present for patients whose pain arises from a specific event, particularly when the event is a medical accident. In contrast, these conditions are unlikely to be present for patients who cannot identify an event that led to their pain.

Why may other-blame be related to poorer adjustment in chronic pain patients?

A number of different factors may be considered to account for a relationship between other-blame and poorer adjustment in chronic pain patients.

Anger. Appraisal-emotion theories (e.g., Smith & Lazarus, 1993) emphasise the importance of cognitions as antecedents to emotions and propose that other-blame is the 'core relational theme' for the emotion of anger. This proposal has been supported empirically (e.g., Smith, Haynes, Lazarus & Pope, 1993) although others have criticised the importance attached by theorists of appraisal factors in causing emotions (Parkinson, 1999).

Researchers who have found a relationship between other-blame and poorer adjustment have suggested that the association may occur due to unresolved anger (Bulman & Wortman, 1977; Solomon & Thompson, 1995). Anger has long been considered to adversely affect physical health (Siegman, 1994) and is a common emotion experienced by chronic pain patients (Okifuji *et al.*, 1999). It is a complex emotion and various aspects of it have been investigated with regard to adjustment in

chronic pain patients. Anger in general, inhibition of anger, and anger towards oneself have been related to increased emotional distress (such as depression) (Duckro, Chibnall & Tomazic, 1995), pain intensity (Okifuji *et al.*, 1999), overt pain behaviours (Kerns, Rosenberg & Jacob, 1994), and is predictive of pain perceptions in people with a variety of chronic pain complaints (Conant, 1998; Gaskin *et al.*, 1992; Wade, Price, Hamer, Schwartz & Hart, 1990). Anger related to other-blame may decrease motivation in pain treatment programmes and weaken therapeutic alliance (DeGood & Kiernan, 1996).

As with other-blame, it may be predicted that anger is more common in event-related pain onset patients. Anger is predicted to be more likely when an event results in severe and unexpected damage, the event was intentional and preventable, and there is a widespread perception of injustice. However, events that are caused by mistakes are still related to increased anger when circumstances are perceived to be uncontrollable (Ben-Zur & Breznitz, 1991; Weiner, 1985). Therefore, it is not surprising that anger appears to be particularly common in chronic pain patients whose pain arose from, for example, work-related injuries (Chapman, 1988) and that health care professionals were among the most common targets of anger in chronic pain patients (Okifuji *et al.*, 1999). Furthermore, anger resulting from other-blame has been suggested to predict PTS symptoms (e.g., Riggs, Dancu, Gershuny, Greenberg & Foa, 1992) which are more prevalent in event-related pain onset patients.

Tennen & Affleck (1990) proposed a model of other-blame (see Figure 1) which includes mediators between other-blame and adjustment. These proposed

mediators appear particularly relevant to chronic pain, i.e., coping, social support, and challenging cherished assumptions, and are therefore included in the discussion below.

Insert Figure 1 about here

Coping. The chronic pain literature generally associates less active coping strategies with poorer adjustment (see Jensen *et al.*, 1990; Snow-Turek, Norris & Tan, 1996). Tennen & Affleck's model proposes that blaming others limits the person's range of possible coping strategies and therefore reduces their ability to implement active coping strategies. For example, blaming others may interfere with planful problem solving and limit the process of positive reappraisal (an important factor in long-term adjustment) (Taylor, 1983). If patients ruminate over blaming others, this may impede their resources to cope and distract them from more positive coping efforts (Church & Vincent, 1990). Blaming others could also be considered to be a type of avoidance coping strategy. In addition, blaming others may limit the person's attempts to identify with other people who are perceived as more powerful (e.g., doctors). Identification with perceived powerful others is thought to be beneficial when people consider themselves to be in an uncontrollable situation as it allows the possibility of vicarious control. An important aim of attempts to adapt to threatening events is to regain a sense of control (Taylor, 1983), a factor which is considered in more detail below.

Perceived Control. Perceived control over day-to-day pain, general locus of control, and self-efficacy are among the beliefs that are thought to influence

adjustment in chronic pain patients (see DeGood & Shutty, 1992 for review). Lack of perceived control over oneself, one's environment, or outcomes, is a commonly cited mechanism for poorer adjustment (e.g., Langer & Rodin, 1976). Blaming others indicates that the event leading to pain or the pain itself are factors that are less likely to be under personal control. This could reduce perceived self-efficacy and lead to drop-out from pain management programmes (Coughlan, Ridout, Williams & Richardson, 1995). However, some research has failed to demonstrate that perceived control is either associated with adjustment to illness (e.g., Malcarne *et al.*, 1995; Sholomskas *et al.*, 1990) or is a mediator between blame and adjustment (see Tennen & Affleck, 1990).

Lack of acceptance of responsibility. Other-blame indicates a lack of acceptance of personal responsibility, a factor that has been related to a lack of a sense of control (Tennen & Affleck, 1990). Other-blame may therefore be detrimental given that health psychology research has shown that people who do not take personal responsibility for their health are less likely to adhere to prescribed medication and are generally less healthy in physical and psychological terms (Synder, 1989). Therefore, people who blame others for their pain onset may feel less responsible for the management of their pain, reducing motivation for treatment programmes that emphasise a management approach.

Blaming others for pain onset involves identifying an external reason for pain such as an injurious event. These factors may encourage the patient to maintain an acute model of pain whereby they believe that their pain will continue unless the underlying problem is corrected. This may result in a longer search for a 'cure' from

health professionals and again, reduced motivation in pain management programmes which require a level of personal responsibility (Turk & Okifuji, 1996).

The literature suggests that the role of perceived control in adjustment for events such as chronic pain is not clear-cut. Some have noted that for chronic illness, making attributions about the initial cause of the illness may be of little importance compared to finding ways to control the course of the illness (e.g., Sensky, 1997). Their rationale is that there is little relevance in feeling that one can prevent the initial event happening again as the illness remains regardless, and more effort should be applied to targeting factors that are currently controllable (Dirksen, 1995; Taylor *et al.*, 1984). However, others indicate that blame for initial pain onset is important in relation to perceived control due to the threat of re-injury (e.g., Derry & McLachlan, 1995). This relates to predictions from the fear-avoidance model of pain which highlights the role of fear of re-injury in maintaining chronic pain.

Social support. In Tennen & Affleck's model, other-blame is proposed to interfere with perceived and actual social support, a widely recognised mediator of physical and psychological adjustment (Connell & Augelli, 1990). If the target of blame is also a possible source of support, this may limit the availability of support and the acceptance of it. Blaming others may also deter people (who are not the target of blame) from offering support. Indeed, research into events that may lead to chronic pain has highlighted that adjustment is likely to be particularly difficult for patients who blame health professionals for their initial pain but with whom contact is necessary during treatment (e.g., Czarnocka & Slade, 2000; Church & Vincent, 1990).

Self-regulation. The literature on illness representations applies the logic of control theory to explain the process people go through when they detect illness symptoms (Carver & Scheier, 1982; Leventhal, Benyamini, Brownlee, Diefenbach, Leventhal, Patrick-Miller & Robitaille, 1997). It is suggested that people engage in a process of 'self-regulation' that enables them to take action to reduce the discrepancy between their perceived present state (i.e., in pain) and their perceived normal state (i.e., pre pain onset). It could be proposed that when people explain their pain as a result of their own actions (i.e., related to self-blame) this enables them to understand what they should do to rectify the problem and could motivate them to take action to manage their pain. However, when people blame others for their initial pain onset, they are less able to self-regulate. The person may feel unable to reduce the discrepancy between their present and perceived normal state, therefore interfering with motivation to manage their pain and resulting in distress (Hill, personal communication).

Just world. Investigations into other-blame has related it to notions of resentment, unfairness, entitlement and a desire for justice (Bulman & Wortman, 1977; DeGood & Kiernan, 1996). These findings fit with the 'just world hypothesis' which states that we have a need to believe that people receive what they deserve in life and deserve what they receive (Lerner, 1975). When such beliefs are challenged, the assumptions that the world is just and orderly are also shattered, and the process of adjusting to the event is slowed down (Janoff-Bulman & Timko, 1987). Tennen & Affleck (1990) call this 'challenging cherished assumptions' and propose it as a mediator in the other-blame/adjustment association. It is a similar idea to control

theory and self-regulation as they are all based on the idea that people have internal working models of the world and themselves (i.e., perceptions of normal states), and distress is predicted to result when discrepancies occur between these models and real life events.

Research Implications

Further research is needed to determine the possible mechanisms that underlie the differences in presentation of patients with event- and non-event-related pain onset. Blame for pain onset is one area that has received little attention in the chronic pain literature. This is despite the large amount of research that suggests that blame is associated with adjustment. Furthermore, blame is a potentially modifiable predictor of adjustment in chronic pain patients and therefore is an important factor to study.

Future research could investigate the role of blame (particularly other-blame given the more robust finding that it is related to poorer adjustment) as a mediator between circumstances of pain onset and adjustment. Adjustment could be broadly defined and include specific emotions that are hypothesised to be related to blame, global distress, PTS symptoms for the event-related patients, disability, and treatment outcomes. Given the possible conceptual distinctions, it would be imperative that measures of blame actually used the word 'blame'. Also attempts should be made to explore the psychometric properties of measures. It may also be important to distinguish between blame for pain onset versus blame for ongoing pain, and investigate whether they are different and if so, how they are related to adjustment. Circumstances of pain onset would also need to be clearly defined so that comparisons

between studies are possible. Generalisability of findings could be improved by using heterogeneous samples of chronic pain patients and include all types of circumstances leading to pain onset.

Tennen & Affleck's (1990) model of other-blame and adjustment is often not mentioned by the blame research and many of the proposed relationships between variables remain untested. Future research using chronic pain samples could investigate the proposed mechanisms such as the role of coping as a mediator between other-blame and adjustment.

Ideally, longitudinal studies would be conducted in order to investigate the temporal links between blame and adjustment. Prospective studies that follow people before their pain onset would be very difficult. One suggestion would be to assess patients visiting their GP soon after initial pain onset, looking specifically at blame attributions for their pain and screening for psychological distress. Subsequent follow-up would allow the investigation of any causal relationship between blame, chronic pain, and distress.

Clinical Implications

The aforementioned research suggests that understanding more about the relationship between blame and adjustment in chronic pain patients whose pain arises from different circumstances may have useful clinical implications. Thoughts/beliefs about blame contribute towards the range of cognitive constructs that make up a patient's model of their chronic pain. They could therefore, be a target in cognitive-behavioural pain management programmes. Given that the circumstances of pain onset cannot be

altered, if blame attributions were shown to be related to adjustment then targeting the patient's thoughts about blame would be of benefit. Maladaptive blame attributions could be challenged, and by considering the available evidence, the patient could consider alternative and more adaptive blame attributions. Findings to date indicate that simply helping the patient consider alternative attributions can make the preferred attribution less convincing, leading to a reduction in distress (Flett & Hewitt, 1990).

Adjustment is likely to be poorer for event-related pain onset patients, and blaming others for pain following an injurious event could encourage an acute model of pain (therefore impeding rehabilitation attempts). Therefore, targeting the beliefs about aetiology of pain should be a priority for patients whose pain follows a specific event. Furthermore, circumstances of pain onset and thoughts about blame are typically reported spontaneously and with ease by patients in consultations with health professionals (DeGood & Kiernan, 1996). They therefore, may be particularly amenable for discussion outside formal cognitive therapy programmes.

Summary and Conclusions

Research in recent years has demonstrated that the presentation of chronic pain patients in terms of factors such as emotional distress, pain variables, disability, and treatment outcome, can be distinguished according to circumstances of initial pain onset. In general, patients whose pain arises from a specific event appear to be more at risk for poorer adjustment compared to patients whose pain arises spontaneously. Although possible reasons for these associations have been mentioned in the literature, there does not appear to be any research that has specifically investigated these factors

in relation to circumstances of pain onset and adjustment. One factor that has been mentioned is blame. Outside the chronic pain literature, much research has investigated the relationship between blame and adjustment, although most of the research has focussed on self-blame. However, the type of blame that has been shown to be the most consistently related to poorer adjustment is other-blame. Only one study in the available research literature appears to have specifically investigated the relationship between other-blame and adjustment in chronic pain patients (DeGood & Kiernan, 1996) and this study suffers various limitations.

The review explored the idea that other-blame for pain onset is more prevalent in chronic pain patients whose pain arose from a specific event and therefore proposed that other-blame may be a mediator between circumstances of pain onset and adjustment. The various ways in which other-blame may result in poorer adjustment in these patients include the possible increase in anger, poorer coping strategies, less social support, poorer perceived control, reduced self-regulation, and the need to believe in a just world. Most of these factors are proposed by Tennen & Affleck's (1990) model of other-blame/adjustment. Limitations on space prevented an exploration of the possible relationships between other types of blame and adjustment in chronic pain patients.

The review has highlighted the various limitations of research into the circumstances of pain onset and the research into blame. Both research areas suffer from poor definitions of key constructs rendering the comparison of studies difficult. In addition, a major problem is the tendency to use correlational/cross-sectional designs. Therefore, it is important to note that conclusions indicating that

circumstances of pain onset and blame are the causes of poorer adjustment are only one possible interpretation.

In conclusion, blame for initial pain onset may be an important factor in explaining adjustment differences in chronic pain patients. Furthermore, blame for pain onset should be a readily available target for intervention in cognitive therapy pain management programmes. The role of blame for pain onset in adjustment in chronic pain patients can only be clarified by further research, and this should aim to improve upon the measurements of blame and designs employed in previous studies.

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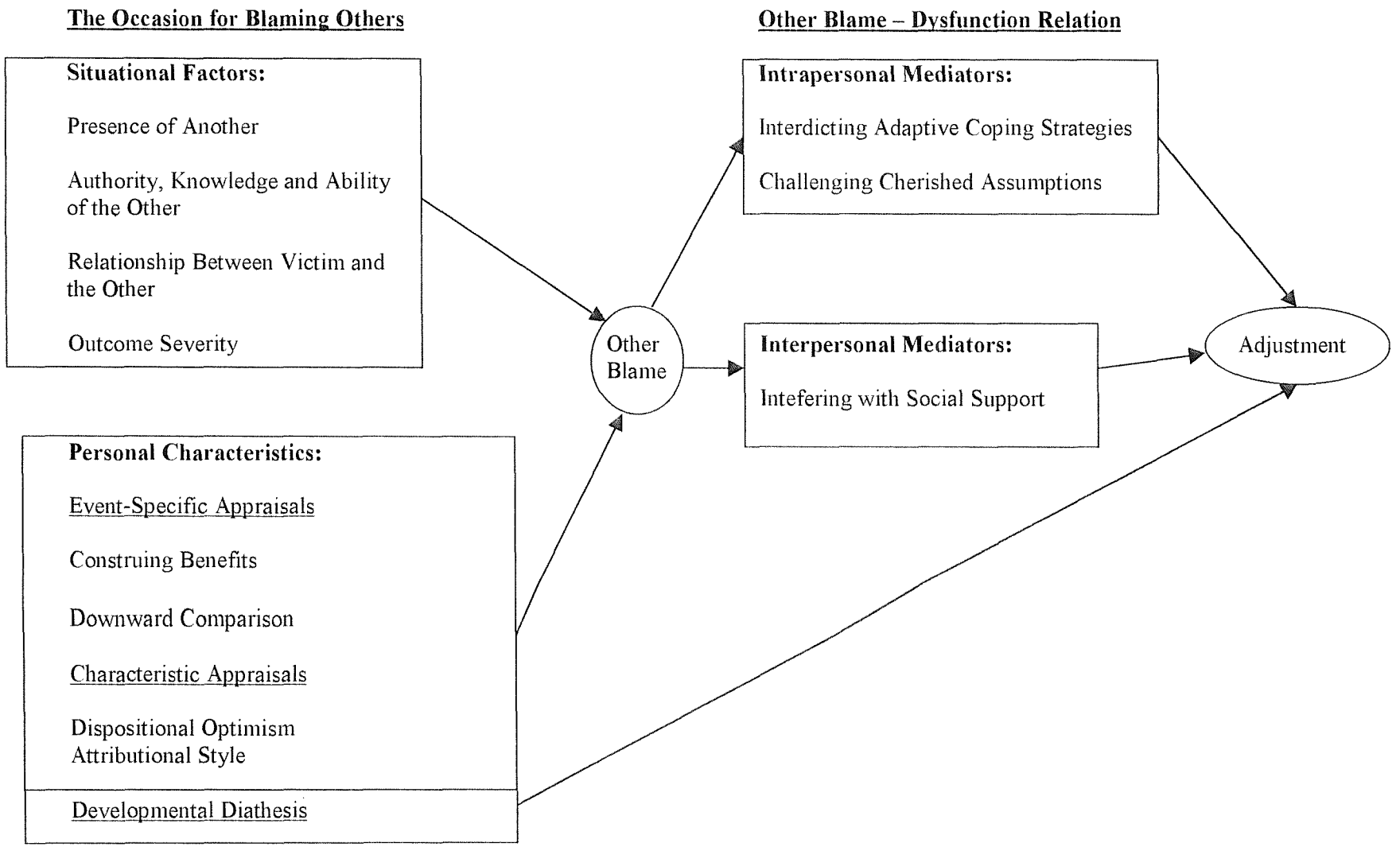
Figure 1. "A model of the factors influencing the incidence of blaming others and its consequences" (p. 220, Tennen & Affleck, 1990).



Empirical Paper:

Circumstances of Pain Onset, Blame, and Adjustment in a Chronic Pain Sample

Heather M. Rawle

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Circumstances of pain onset, blame, and adjustment in a chronic pain sample

Heather M. Rawle

University of Southampton

Running Head: Blame and chronic pain

Address for correspondence:

H. Rawle, Training Course in Clinical Psychology, Psychology Department,

University of Southampton, Southampton, SO17 1BJ, United Kingdom.

Circumstances of pain onset, blame, and adjustment in a chronic pain sample

Abstract

Objectives. Research has shown that chronic pain patients whose pain arises following a specific event are more poorly adjusted compared to patients whose pain arises for no apparent reason. No research to date has specifically considered the role of blame in this pattern of findings. The present study aimed to investigate whether blaming others for initial pain onset mediates the relationship between circumstances of pain onset and adjustment (generalised psychological distress, hostility, and self-reported pain intensity). A further aim was to explore the association between different types of blame and various adjustment variables.

Design. A between and within participants design was employed with patients compared according to the circumstances of their initial pain onset.

Method. An exploratory measure of blame and standardised measures of adjustment variables were completed by the patients.

Results. The event- and non-event-related pain onset groups did not differ significantly in terms of the adjustment variables, and other-blame as a mediator of adjustment could not be supported. However, the event-related pain onset group did blame other people more for their initial pain onset, and this was related to higher post-traumatic stress symptom scores. Self-blame was associated with anxiety and the use of maladaptive coping strategies.

Conclusions. The results suggest areas of further research regarding the role of blame in adjustment of chronic pain patients. Clinical implications regarding the possible detrimental consequences of other- and self-blame are discussed.

Circumstances of pain onset, blame, and adjustment in a chronic pain sample

Chronic pain can arise following a specific injurious event such as an accident, illness or surgery (i.e., 'event-related pain onset'). Alternatively, chronic pain may arise for no apparent reason (i.e., 'non-event-related pain onset'). Research shows that chronic pain patients who perceive their initial pain to arise following a specific event report more generalised psychological distress, more specific psychological distress such as hostility, and increased pain intensity compared to patients whose initial pain onset is perceived as spontaneous (e.g., Thorne, 1998; Toomey, Seville, Finkel, Mann, Abashian & Klocek, 1997; Turk & Okifuji, 1996; Turk, Okifuji, Starz & Sinclair, 1996). In addition, high levels of post-traumatic stress (PTS) symptoms have been reported in patients whose pain follows a specific injurious event (e.g., Chibnall & Duckro, 1994; Geisser, Roth, Bachman & Eckert, 1996). Not all research supports these findings (e.g., Kuch, Evans, Watson & Bubela, 1991; Waylonis & Perkins, 1994) and discrepancies in findings exist between studies that have shown support for some of the adjustment differences. Such inconsistencies may be attributed to methodological differences such as the method of grouping circumstances of pain onset and comparisons of diagnostic-specific patient samples.

An understanding of the reasons why circumstances of pain onset have different influences on adjustment would inform psychological intervention. However, there has been little research that specifically aims to investigate factors that may mediate the relationship between type of pain onset and adjustment. The present study aims to address this research gap by considering the role of blame for pain onset (see Rawle, 2000, for literature review). Blame for negative events has often been

shown to be related to adjustment in non-chronic pain samples. The type of blame to be most consistently related to poorer adjustment is blaming others (Derry & McLachlan, 1995; Sholomskas, Steil & Plummer, 1990; see Tennen & Affleck, 1990, for review). Oneself, the environment and chance are common targets of blame but are less consistently related to adjustment (Brown, Bell, Maynard, Richardson & Wagner, 1999; Michela & Wood, 1986; Reidy & Caplan, 1994; Rich, Smith & Christensen, 1999; Taylor, Lichtman & Wood, 1984). The PTS literature has revealed associations between blame and PTS. Blaming others has been shown to be a major predictor of PTS symptoms (Czarnocka & Slade, 2000). More specifically, intrusive symptoms of PTS have been related to both self- and other-blame (Delahanty, Herberman, Craig, Hayward, Fullerton, Ursano & Baum, 1997; Joseph, Yule & Williams, 1993).

A comprehensive search of the chronic pain literature found only one study which aimed to specifically investigate the role of blame for chronic pain in adjustment. DeGood & Kiernan (1996) found that patients who perceived another person to be at fault for their pain reported more global and specific psychological distress, more behavioural disturbance, poorer response to past treatments, and fewer expectations of future benefits, than patients who faulted no-one. The authors concluded that 'attribution of blame may be an under-recognized cognitive correlate of pain behaviour, mood disturbance, and poor response to treatment' (p.153). However, this study did not compare circumstances of pain onset, it was not clear whether patients were being asked about fault for pain onset or ongoing pain, and the questions were forced choice, thus precluding the possibility that patients may attribute blame to

more than one source or blame sources to varying degrees. Further, the study did not include the word 'blame' in its measures despite warnings from the research literature that participants should be questioned specifically about 'blame' in order to maximise construct validity (see Shaver & Drown, 1986).

Since blaming others is consistently related to poorer adjustment, it is possible that 'other-blame' is more closely associated with event-related pain onset than non-event-related pain onset in chronic pain patients, and this may offer an explanation for event-related pain onset patients reporting poorer adjustment. The proposal that other-blame may be more closely associated with event-related pain onset is supported by a model proposed by Tennen & Affleck (1990) which specifies circumstances that are likely to result in other-blame. The ways in which other-blame may result in increased psychological distress and pain can be speculated upon. Other-blame has been related to increased anger and hostility (e.g., Smith & Lazarus, 1993; Vieyra, Tennen, Affleck, Allen & McCann, 1990), and anger and hostility has been related to increased pain (e.g., Burns, 1997; Okifuji, Turk & Curran, 1999). Tennen & Affleck's (1990) model of other-blame suggests other mediators of adjustment that are particularly salient to chronic pain but which remain untested by empirical research. For example, they suggest that other-blame interferes with adaptive coping strategies and this mediates the impact of other-blame on adjustment.

Despite the large amount of research into the role of beliefs in chronic pain adjustment (see Jensen, Turner, Romano & Lawler, 1994), no published research to date has investigated the role of different types of blame for pain onset in chronic pain adjustment. Therefore, the broad aim of the present study was to explore the

relationships between blame for pain onset (i.e., other-blame, self-blame, environment-blame, chance-blame, and no blame) and global psychological distress, specific psychological distress (including PTS symptoms), self-reported pain intensity, and circumstances of pain onset.

A more specific aim of the study was to investigate whether other-blame mediates between circumstances of pain onset (i.e., event- versus non-event-related pain onset) and global psychological distress, hostility, and self-reported pain intensity. Since coping is a well researched variable in chronic pain (see Jensen, Turner, Romano & Karoly, 1991) and a suggested mediator between other-blame and adjustment, the relationships between different types of blame for pain onset and coping were also explored.

The study is considered exploratory due to the lack of availability of a standardised measure of blame for pain onset, the lack of predictions regarding relationships apart from those between other-blame and outcome variables, and the limitations associated with a cross-sectional design. However, the exploratory nature of this research is deemed necessary in order to indicate directions for future research in blame and chronic pain. The study is unique in that participants were specifically asked about 'blame' (regarding initial pain onset), using methodology that allowed participants to blame various sources and to varying degrees. With the exception of Toomey et *al.* (1997), previous research into circumstances of pain onset has used diagnostic-specific or event-specific patient samples. The present study employed a heterogeneous chronic pain patient sample and divided them into two broad categories of circumstances of pain onset depending on their ability to identify an event that

precipitated their pain. The sample therefore includes all types of pain onset, maximising the generalisability of findings. The unambiguous method of categorising patients facilitates comparability of findings with other studies.

Method

Design

The study employed a within and between participants design.

Participants

Data was derived from a convenience sample of 100 chronic pain patients attending a hospital pain clinic who fulfilled the inclusion criteria and agreed to participate in the research. For inclusion in the research, participants had to provide written consent, have a pain duration of greater than three months (to fulfill criteria for the definition of chronic pain), be aged 18 years or over, be English speaking, and not be demonstrating evidence of a cognitive or psychiatric impairment.

The sample consisted of 52 males and 48 females with an average age of 50.9 years (SD = 16.35) (range between 23 and 84 years) and an average pain duration of 6.85 years (SD = 8.77) (range between 4 months and 50 years). The most frequent sites of pain were low back (52 per cent) and multiple sites (27 per cent).

Circumstances of pain onset. Patients were classified as either event-related pain onset patients or non-event-related pain onset patients depending on their response to the statements "my pain first began because of, or following one particular event" (n = 65) and "my pain did not begin after a(ny) particular event" (n = 35)

(Appendix I). In the event-related pain onset group, patients' descriptions of events were: accident at work (n = 18); accident at home (n = 14); road accident (n = 7); following illness (n = 8); following an incident related to their health care (such as surgery, receiving physiotherapy or receiving incorrect medical treatment) (n = 8); and other event (n = 10). Twenty of the non-event-related group (57.1 per cent) reported that they had no idea what led to their pain and two of them mentioned gradual wear and tear. The remainder indicated events that led to their pain elsewhere on the questionnaires: following illness (n = 8); following an incident related to their health care (n = 2); and accident at work (n = 3). However, since it was felt that the patient's perception of whether an event led to their pain was more important than what actually happened, the patients were classified according to their response to the statements outlined above.

Measures

All measures were completed by the participants except for the Impact of Event Scale which was only completed by the event-related pain onset patients.

Blame. An exploratory measure was constructed by the author since there was no measure available regarding blame for pain onset (Appendix II, questions 4 – 9). Blame types were selected as a result of a literature search and from interviews with six chronic pain patients. These suggested five blame types which were not mutually exclusive. Self-report rating scales were selected as these have been found to be superior to forced-choice methods and judges' ratings of participants' responses to open questions in attribution/adjustment research (Taylor *et al.*, 1984; Vieyra *et al.*,

1990). The measure required respondents to rate on an 8-point scale the degree to which they blamed others, themselves, chance, environment, and nothing for their pain onset. Participants rating more than zero on the self-blame item were asked to specify if they blamed their character or their behaviour (Janoff-Bulman, 1979). An additional question asked respondents what they blamed for their pain onset if they circled zero for all the blame types in order to ensure all types of blame were considered. Psychometric properties are discussed below.

Psychological distress. Levels of psychological distress were measured using the Brief Symptom Inventory (BSI; Derogatis, 1993) which is a 53 item short form of the Symptom Checklist (SCL-90-R; Derogatis, 1983). It is divided into nine primary symptom dimensions, and provides a global index of distress. One of the dimensions is called 'hostility' whereby items include 'thoughts, feelings, or actions that are characteristic of the negative affect state of anger' (Derogatis, 1993, p.8). Respondents rated the relevance of each item to their experience in the last 7 days on a 5-point scale. The BSI is suitable for use with chronic pain patients (Geisser, Perna, Kirsch & Bachman, 1998) and the psychometric properties are reported to be good (Derogatis, 1993).

Post-traumatic stress symptoms. Patients who stated that their pain followed a specific event completed the Revised Impact of Event Scale (IES: Horowitz, Wilner & Alvarez, 1979). This has been shown to correlate with PTSD (Zillberg, Weiss & Horowitz, 1982) and is widely used for both clinical and research purposes. The 15 item inventory requires respondents to identify a specific life event (the event that led to pain in the present study) and then rate each item according to the frequency with

which it was experienced in the last 7 days. Items measure intrusion (n = 7) and avoidance (n = 8) symptoms. A total score can be calculated. The psychometric properties are reported to be good (Horowitz *et al.*, 1979).

Pain intensity. The Short Form McGill Pain Questionnaire (SF-MPQ; Melzack, 1987) was used to measure the subjective pain experience (see Appendix III for written permission from the author). The main component of the SF-MPQ is 15 pain descriptors (11 sensory; 4 affective) of which the intensity is rated from 0 to 3. This produces three pain scores – sensory, affective and total. It also includes the Present Pain Intensity index of the long form MPQ (Melzack, 1975) and a visual analogue scale. The SF-MPQ correlates highly with the long form MPQ, which itself is reported to have good psychometric properties (Hunter, Phillips & Rachman, 1979; Reading, 1983).

Coping. Level of use of various coping strategies was assessed using the COPE (Carver, Scheier & Weintraub, 1989). This 60 item scale falls into 15 subscales of conceptually distinct coping strategies. Respondents are asked to rate the frequency with which they use each coping strategy on a scale of 1 to 4. The COPE is reported to be a useful measure in chronic pain research (Skevington, 1995). In the current study, a situational version was administered whereby respondents were asked to complete the inventory with respect to how they coped with their chronic pain. Carver *et al.* (1989) report good psychometric properties for the subscales.

The subscales can be divided into those which are hypothesised to be adaptive in situations where active coping is associated with good outcome ('most adaptive strategies'); those which have a less obvious link with active coping but still should be

adaptive ('adaptive strategies'); and those which are predicted to be maladaptive ('maladaptive strategies') (Carver *et al.*, 1989). As active coping strategies are reported to be more adaptive than passive coping strategies in chronic pain patients (Snow-Turek, Norris & Tan, 1996), these categories were used in the present study. The table below shows the subscales which are hypothesised to fall into these three broad categories.

Insert Table 1 about here

For the purposes of the present study, the 15 subscales were collapsed into the three broader categories ('Most adaptive', 'Adaptive' and 'Maladaptive'). This was achieved by calculating the mean score for each global scale based on the scores derived from the subscales that are proposed to contribute to them.

Personal and pain information. Participants were also asked for personal information (age, gender, work status, living arrangements, experience of significant events in the six months prior to pain onset) and pain related information (pain site, duration, perceived cause, experience of operations, receipt of clear explanation about cause of pain, compensation status and medication) (Appendix II).

Procedure

The research received appropriate university and local ethical approval (Appendix IV). The study was conducted in an outpatient pain clinic. Patients who fulfilled the

inclusion criteria were given information about the study by the researcher (see Appendix V). If in agreement, they were given a consent form to sign (see Appendix VI) and a questionnaire pack to complete during their visit. Participants were instructed to complete the IES only if they perceived their pain to have followed an event. In order to examine test-retest reliability of the blame questions, a subsample were asked to complete the blame questions again two weeks later (n = 18).

Data analysis

Checks for normality indicated that many of the variables were not normally distributed. Therefore, non-parametric tests were used as they are considered to have greater power than parametric tests when assumptions of normal distribution are violated (Blair & Higgins, 1985).

In order to determine whether the event-related and non-event-related pain onset groups differed in terms of personal and pain characteristics, Mann-Whitney U tests (for continuous variables) and chi-square tests (for categorical variables) were used. Psychometric properties of the blame questions were also investigated.

The results were then separated into two sections: those related to specific hypotheses and those that were more exploratory. Hypotheses regarding group differences were tested using Mann-Whitney U tests. For within group analyses, Spearman's rho correlations were used.

Specific hypotheses: One-tailed tests were used as hypotheses were directional. To avoid the problem of inflated error rates because of the number of tests performed,

the Bonferroni adjustment procedure was applied and only results significant at the .006 level are reported.

Other-blame as a mediator. The aim was to explore whether degree of other-blame acted as a mediator between type of pain onset (event versus non-event related) and (a) distress variables (BSI Global Severity Index (GSI) and BSI Hostility), and (b) pain intensity (SF-MPQ PRIT). In order to determine that degree of other-blame had a mediational role, first it had to be established that a relationship between the independent variable (type of pain onset) and the mediator (other-blame) was significant. It then had to be established that the relationship between the independent variable (type of pain onset) and the dependent variables (BSI scores and pain intensity) were also significant. Finally, the relationship between the mediator (other-blame) and the dependent variables (BSI scores and pain intensity) had to be significant. If the aforementioned relationships are established, other-blame is included in the regression equation. The relationship between type of pain onset and distress and pain variables should either be no longer significant or should be weakened if other-blame is a total or partial mediator (Baron & Kenny, 1986).

Exploratory analysis: Two-tailed tests were used to explore the relationships between blame attributions and: IES scores, COPE collapsed scales, type of pain onset, BSI scores, and pain scores. Due to the multiple comparisons increasing the risk of Type I errors, a conservative alpha level of .01 was adopted. The Bonferroni adjustment technique was not employed since the analyses were exploratory and adopting an even more conservative alpha level raises the risk of Type II errors.

Results

Sample characteristics

The pain-onset groups did not differ significantly in terms of age, pain duration, pain site, current involvement in pain-related compensation claims, employment status, experience of previous operations, medication, receipt of a clear explanation about their pain, living arrangements or whether the patient was new to the pain clinic. The pain-onset groups were significantly different in terms of gender ($X^2 = 6.77$, $p = .009$) and experience of life events/changes in the six months prior to pain onset ($X^2 = 10.68$, $p = .001$). There were more males (61.5%) and fewer females (38.5%) than expected in the event-related pain onset group. Also, there were fewer males (34.3%) and more females (65.7%) than expected in the non-event-related pain onset group. Patients in the event-related pain onset group reported significantly more experience of significant events/changes in the six months prior to their pain (35.4%) compared to the non-event-related group (5.7%).

Psychometric properties of blame questions

The blame questions showed very good test-retest reliability.

Insert Table 2 about here

Correlations showed 'nothing-blame' inversely correlated with other-blame ($r = .-414$, $p < .001$) and self-blame ($r = -2.14$, $p < .05$). This suggests that the questions were reasonably successful in enabling participants to distinguish between blaming

nothing and blaming either themselves or another person, hence demonstrating the validity of the questionnaire. It was not predicted that the blame types would or would not be correlated in other ways as it was considered that it was possible for people to blame more than one source. Other-blame was also significantly correlated with BSI Paranoid Ideation ($r = .481$, $p < .001$) which supports the validity of the other-blame question. The questions had good face validity.

Blame attributions

Most participants blamed more than one factor. The most likely blame type to coincide with all the other blame types was chance (see Appendix VIII for cross-tabulations of associations between types of blame). Chance was the most popular type of blame overall (i.e., rated more than 0 the most frequently), followed by blaming nothing, self-blame, other-blame, and blaming environment. In the event-related pain onset group, the most popular type of blame was chance, followed by other-blame, self-blame, nothing-blame and environment-blame. In the non-event-related pain onset group, the most popular type of blame was nothing, followed by chance-blame, self-blame, environment-blame and other-blame (see Table 3). In both groups, of those who blamed themselves, most blamed their behaviour (84% overall). When patients rated more than zero on the blame questions, the most popular degree of blame was total blame (i.e., 7) for most blame types. All participants rated more than zero for at least one blame type.

Insert Table 3 about here.

Specific hypotheses

Relationship between independent variable (pain onset group) and proposed mediator (other-blame). As predicted, the event-related pain onset group had significantly higher other-blame ratings than the non-event-related pain onset group ($U = 660.5$, $p < .001$).

Respondents' descriptions of events preceding their pain were coded according to the presence or absence of factors that are predicted by Tennen & Affleck (1990) to lead to other-blame. These authors predict other-blame to be more likely when someone else is present at the time of the event; the person blamed is in a position of authority, or has relevant skills or knowledge; the person blamed is not well known to the person attributing blame; and the outcome is severe. In the current study, the presence of a predictive factor was coded as 1; its absence was coded as 0; and therefore, the maximum total predictive score was 4. As Tennen & Afflecks' model predicted, the score was significantly correlated with other-blame ratings ($r = .435$, $p < .001$).

Since the event-related pain onset group had significantly more males than expected, it may be considered that the higher other-blame ratings in the event-related group was a gender effect. However, since only two people in the non-event-related group rated more than zero for other-blame, further statistical analysis were not considered necessary to demonstrate that the group differences were found regardless of gender.

Relationship between independent variable (pain onset group) and dependent variables (BSI GSI, BSI Hostility, and PRIT). The event-related pain onset group

reported higher mean scores for global distress and BSI Hostility compared with the non-event-related pain onset group. Although this was in the predicted direction, none of these differences were significant (even at an alpha level of .05). There were no significant differences between the event and non-event-related pain groups in terms of total pain intensity (SF-MPQ PRIT).[1]

Insert Table 4 about here

Relationship between proposed mediator (other-blame) and dependent variables. Against predictions, other-blame was not significantly correlated with BSI Hostility (r = .115, p = .128) or pain intensity (r = .150, p = .068). The correlation between other-blame and global distress was not significant at the set level (r = .193, p = .027).

Other-Blame as a mediator. The hypothesis that other-blame is a mediator between type of pain onset and distress and pain intensity can be rejected without further analysis since not all of the pre-requisite relationships were found to be significant.

[1] Since previous research has grouped patients differently to the present study (e.g., including all patients whose pain arose from illness in one group or the other) and found differences in distress and pain, patients were regrouped in this way and these analyses were repeated. No significant differences were found.

Exploratory analysis

Relationship between blame and PTS symptoms. On average, the event-related pain onset group scored within the 'low distress' range (see Horowitz, 1982) for intrusion (mean = 8.33, SD = 9.82) and avoidance (mean 7.09, SD = 9.10). The only type of blame score to be significantly correlated with the Impact of Event Scale scores was other-blame. Other-blame was significantly correlated with the Avoidance subscale ($r = .402$, $p < .01$) and total score ($r = .361$, $p < .01$) but was not significant at the set level for the Intrusion subscale ($r = .295$, $p = .026$).

Relationship between blame and coping. Self-blame scores were significantly correlated with the use of 'maladaptive' coping strategies ($r = .297$, $p < .01$). There was a trend for other-blame to be correlated with the use of 'most adaptive' and 'adaptive' coping strategies but the correlations were not at the set significance level ($r = .230$, $p = .021$; $r = .211$, $p = .035$).

Type of pain onset and blame. The non-event-related pain onset group had significantly higher 'blame nothing' scores than the event-related pain onset group ($U = 541$, $p < .001$). There was a trend towards more self-blame in the event-related pain onset group (mean = 1.80, SD = 2.680) compared to the non-event-related group (mean = .97, SD = 2.01) but this was not significant ($p = .078$).

Relationship between blame and distress. Self-blame was significantly correlated with anxiety ($r = .280$, $p < .01$). There were no significant associations for the other types of blame.

Relationship between blame and pain. None of the blame types were significantly correlated with any of the pain variables.

Discussion

The study aimed to examine whether blaming others for initial pain onset played a mediating role between circumstances of pain onset and generalised psychological distress, hostility, and self-reported pain intensity. As predicted, there was significantly more other-blame for initial pain onset in the event-related pain onset group. Tennen & Afflecks' (1990) model of factors predicting the incidence of other-blame was also supported (i.e. presence of another person at the time of the event; the blamed person being in a position of authority, or having relevant skills or knowledge; the blamed person not being well known to the person attributing blame; and the outcome being severe). However, contrary to previous findings, there were no significant differences between circumstances of pain onset in terms of generalised psychological distress, hostility, and self-reported pain intensity. Therefore, the results provided evidence against the hypothesis that other-blame is a mediator between circumstances of pain onset and psychological distress and pain.

The study also aimed to explore the relationships between blame attributions (other-blame, self-blame, environment-blame, chance-blame, and nothing-blame) for initial pain onset and measures of distress (including PTS symptoms), pain intensity, and coping. Other-blame was significantly related to both total and avoidance PTS symptoms. Self-blame was significantly related to anxiety and the use of maladaptive coping strategies. Blaming chance, the environment, and nothing, were not significantly related to any measure of adjustment.

Responses regarding blame for initial pain onset suggest that people typically blame a variety of factors. Patients were much more likely to blame others if their

pain initially arose following a specific event. Patients whose pain arose spontaneously were more likely to blame nothing. Blaming chance was endorsed by more than half the sample, regardless of circumstances of pain onset. When patients blamed themselves, more than 80 percent blamed their behaviour rather than their character (as found in previous research using different samples, e.g., Sholomskas *et al.*, 1990). These findings suggest that future research into blame for pain onset should provide respondents with the opportunity to identify a variety of targets for blame, including others, self (behaviour) and chance, and also allow for the possibility that nothing may be blamed. The results also call into question the use of forced choice methods such as those used by DeGood & Kiernan (1996).

Contrary to findings from previous research (e.g., Thorne, 1998; Toomey *et al.*, 1997; Turk & Okifuji, 1996), the event-related and non-event-related pain onset groups did not significantly differ in terms of generalised and specific distress, and pain. This discrepancy does not appear to be due to differences in categorising circumstances of pain onset, but may possibly be explained by differences in sample characteristics. Overall, patients in the present study had lower distress scores than those employed in previous research with at least a third scoring in the non-clinical range. Also, in contrast to the present study, most previous studies have employed patients who are new to pain clinics and therefore likely to include patients with shorter pain durations. It may be therefore, that the influence of circumstances of initial pain onset on distress and pain diminishes over time as memory for initial pain onset fades and other factors prove more important in affecting chronic pain adjustment e.g., cognitive, emotional, social, and behavioural factors.

Inconsistent with previous findings, other-blame was not significantly related to hostility or generalised distress. However, consistency between previous and current findings was found in the lack of association between other-blame and pain (DeGood & Kiernan, 1996). There are various possible explanations for the lack of significant findings. Although other-blame was common in the event-related pain onset group, the mean extent of this blame was relatively low. The level of blame may not therefore, have been high enough to trigger anger as predicted by appraisal-emotion theories. Further, longitudinal research has suggested that the relationship between blame and adjustment may diminish over time (Richards, Elliott, Shewchuk & Fine, 1997). This may account for the lack of significant findings as the present sample had a lengthy average pain duration.

Much of the previous research that has associated other-blame with poorer adjustment has purported to measure 'blame' but have employed measures using various wording such as 'responsibility', 'cause' and 'fault'. However, these concepts are not necessarily interchangeable (Shaver & Drown, 1986). The present study specifically asked people about 'blame' and so it may be that poorer adjustment is not associated with blame but is associated with the related concepts e.g., fault.

Some theories emphasise the importance of perceived controllability of negative events in predicting anger (Ben-Zur & Breznitz, 1991). It is possible that the lack of association between other-blame and anger in the current sample could be due to patients being unable to control the events that were perceived to precipitate their initial pain onset (e.g., surgery and road accidents). As Parkinson (1999) concludes, the relationship between other-blame and anger may not be as clear as stated by

appraisal-emotion theories, and this conclusion appears to be supported by the findings of the present study.

Alternatively, the lack of support for the prediction that other-blame would be related to anger may have been due to the employment of a potentially unsuitable measure of anger. The BSI Hostility subscale was selected in order to minimise the number of questionnaires administered to patients. Although hostility and anger are related constructs, and previous research has demonstrated an association between other-blame and hostility using the long form of the BSI (Vieyra *et al.*, 1990), it is also argued by some that the concepts are not interchangeable (Eckhardt, Barbour & Stuart, 1997; Miller, Smith, Turner, Guijarro & Hallet, 1996). Future research could clarify an association between other-blame and anger in chronic pain patients by employing measures that specifically assess anger (e.g., State-Trait Anger Expression Inventory, Spielberger, 1988).

The finding that other-blame was linked to PTS symptoms (total and avoidance) is consistent with some research using non-chronic pain samples (e.g., Czarnocka & Slade, 2000). However, the findings are contrary to studies that have found PTS symptoms to be more related to self-blame than other-blame (Joseph *et al.*, 1993). Anger has been found to predict PTS symptoms (Andrews, Brewin, Rose & Kirk, 2000; Ehlers, Mayou & Bryant, 1998; Feeny, Zoellner & Foa, 2000) and therefore, one explanation for the other-blame/PTS symptoms association may be that other-blame results in anger which in turn leads to PTS symptoms.

The finding that other-blame is related to avoidance PTS symptoms is contrary to previous studies which found other-blame to be more related to intrusive symptoms

(Delahanty *et al.*, 1997). However, the other-blame/avoidance PTS symptoms association in chronic pain patients would be predicted by the fear-avoidance model of pain (see Vlaeyen & Linton, 2000). Fear-avoidance beliefs have been found to be particularly salient for people whose pain results from a traumatic injury (Crombez, Vlaeyen, Heuts & Lysens, 1999). Such beliefs can maintain the chronic pain experience and be symptomatic of PTS as people may avoid reminders of events reminiscent of those which triggered the pain for fear of re-injury. It seems reasonable to propose that blaming others is also a type of avoidance (e.g., avoidance of responsibility). Future research could clarify the relationship between other-blame and avoidance behaviour in chronic pain patients by employing the Ways of Coping Checklist (Folkman & Lazarus, 1980) which includes an 'avoidance coping' factor.

It is possible that other-blame is not directly related to PTS symptoms but that a third factor was influencing both variables. For example, research has shown trauma severity predicts PTS symptoms (Ehlers *et al.*, 1998) and severity of outcomes is a factor predicted to result in other-blame (Tennen & Affleck, 1990). Trauma severity was not assessed in the present study but future research could explore such factors, perhaps using a measure of the participant's own perception of trauma severity (Blanchard, Hickling, Mitnick, Taylor, Loos & Buckley, 1995).

The trend that other-blame was related to more adaptive coping is contrary to predictions from Tennen & Affleck's (1990) model which proposed that other-blame should interfere with adaptive coping strategies. In addition, the finding that self-blame was related to the use of maladaptive coping strategies is contrary to some previous studies (e.g., Bulman & Wortman, 1977) but consistent with others (e.g.,

Nielson & MacDonald, 1988). Self-blame for negative events has been proposed to lead to poorer coping as a result of the individual believing that the event was avoidable which decreases the person's beliefs in their own effectiveness (Brewin, 1984; Nielson & MacDonald, 1988).

The exploratory analysis also found self-blame to be related to anxiety. This is somewhat surprising given that self-blame in chronic pain patients is more often related to depression than anxiety, both when it is measured as part of a coping questionnaire (see Jensen *et al.*, 1991) and when it is measured as part of a questionnaire on pain beliefs (Williams, Robinson & Geisser, 1994). The self-blame and depression link is often explained by noting that characterological self-blame reduces perceived control and therefore is linked to depression as predicted by the reformulated learned helplessness model (Abramson, Metalsky & Alloy, 1989; Janoff-Bulman, 1979). In the studies reported by Jensen *et al.* (1991) and Williams *et al.* (1994), it is not possible to determine whether patients blamed their pain experience on their character or their behaviour. However, in the present study, it is clear that most of the 'self-blamers' blamed their behaviour rather than their character, and therefore, a lack of association with depression is not surprising. Instead, it could be speculated that patients who blame their initial pain onset on their own behaviour may be particularly anxious about re-injury (as predicted by the fear-avoidance model) since they recognise the possible influence they can have on triggering pain.

There are various limitations of the present study which mean results should be considered with caution. The cross-sectional design means that conclusions drawn regarding the nature of causality between variables should be regarded as speculation

only. Although standardised measures were used for psychological distress, pain, PTS symptoms and coping, the measure of blame for pain onset was designed specifically for the study due to the lack of an existing measure. Unlike much previous research, the exploratory nature of the blame measure was emphasised and attempts were made to consider the psychometric properties which showed promise. All data was based on self-report and is retrospective and therefore the accuracy of the information is uncertain. Also, conclusions drawn from the current sample of pain clinic patients can not be generalised to the general population of chronic pain sufferers given the selection biases inherent in clinic samples (see Crombie & Davies, 1998).

Future research could address some of these limitations by employing prospective designs in order to establish direction of causality between variables. Blame for pain onset could be explored using qualitative methods leading to the development of a valid and reliable measure of blame suitable for quantitative research. Generalisability of results could be maximised by questioning patients with chronic pain of a short duration before attendance at a pain clinic. These could be followed up over time to explore any changing relationships between blame and adjustment. Future research should also explore in more detail the relationship between other-blame and PTS symptoms, perhaps considering factors that may influence this relationship (see Blaszczynski, Gordon, Silove, Sloane, Hillman & Panasetis, 1998). Self-blame for initial pain onset may also be an important factor for further investigation given its association with indicators of poorer adjustment.

Although results must be interpreted with caution and require clarification from further research, they do suggest that clinicians working with chronic pain

patients should be vigilant for patients who blame others or themselves for their initial pain onset. Patients whose pain initially arose following a specific event and who blame others for their initial pain onset may be at a higher risk of experiencing PTS symptoms. Self-blame may be related to anxiety and the use of maladaptive coping strategies. Since blame for initial pain onset appears to be a relatively easy attribution to access, cognitive behavioural pain management interventions could explore patients' blame attributions and direct patients towards more neutral blame types (e.g., chance and the environment) and blaming nothing, given the stronger evidence that these types are unrelated to distress and pain.

In conclusion, to the author's knowledge, this was the first study to explore the mediating role of other-blame on the relationship between circumstances of pain onset and psychological distress and pain. It was also the first study to explore the relationship between various types of blame and adjustment in a chronic pain sample. Although results did not support the mediating role of other-blame, some interesting findings did emerge which have implications for further research and clinical practice. Contrary to previous research, the present study found no differences between event-related and non-event related pain onset groups in terms of psychological distress and pain. It may be that the influence of circumstances of pain onset on chronic pain adjustment diminishes over time and therefore may be best addressed soon after pain onset. However, other-blame may be common in event-related pain onset patients and should be targeted in treatment as it is related to PTS symptoms. Self-blame should also be targeted as it appears to be related to elements of poorer adjustment that may interfere with management of chronic pain. Future research could investigate further

the relationship between other-blame for initial pain onset and PTS symptoms, and further explore the role of self-blame for initial pain onset on adjustment to chronic pain. However, it is also important to develop a measure of blame for pain onset that is valid and reliable.

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Table 1 Hypothesised 'adaptive' function of the COPE subscales

Subscale	'Adaptive' function (Carver *et al.*, 1989)
Active coping	
Planning	
Seeking instrumental social support	Considered to be most adaptive
Positive reinterpretation and growth	
Acceptance	
Seeking emotional social support	
Suppression of competing activities	Considered to be adaptive
Restraint coping	
Turning to religion	
Focus on and venting of emotions	
Denial	
Mental disengagement	Considered to be maladaptive
Behavioural disengagement	
Humour	
Alcohol/drug use	

Table 2 Test-retest reliability of blame questions

	Spearman's rho	p value (one tailed)
Blame environment	.997	.000
Blame nothing	.950	.000
Blame other	.895	.000
Blame self	.654	.002
Blame chance	.549	.009

Table 3 Number and percentage of patients who rated more than zero on blame questions

Blame type	All patients	Event-related group		Non-event-related group	
	N	N	Percent	N	Percent
Chance	68	48	73.8	20	57.1
Nothing	49	22	33.8	27	77.1
Self	35	27	41.5	8	22.9
Other	33	31	47.7	2	5.7
Environment	20	13	20.0	7	20.0

Note. Totals add up to more than 100% because people could rate more than one type of blame.

Table 4 Means and standard deviations for the BSI Global Severity Index (GSI), Hostility, and the SF-MPQ Pain Rating Index Total (PRIT) by pain onset group

	Event-related group (N = 65)		Non-event related group (N = 35)			
	Mean	SD	Mean	SD	U	Sig.
Distress						
GSI	0.89	0.77	0.67	0.55	968.5	NS
Hostility	0.80	0.93	0.53	0.65	961.5	NS
Pain						
PRIT	15.66	8.37	15.86	9.09	1114.5	NS

Appendices

Appendix I: Questions determining circumstances of pain onset (cover sheet to Impact of Events Scale).

Appendix II: Questionnaire regarding personal and pain information, including questions regarding blame for pain onset.

Appendix III: Written permission from the author to use the Short-Form McGill Pain Questionnaire.

Appendix IV: Letters of ethical approval:

- University Ethical Approval
- Bath Local Research Ethics Committee
- Royal United Hospital Research and Development Committee

Appendix V: Participant information sheet.

Appendix VI: Consent form.

Appendix VII: Instructions for authors of target journals:

- Clinical Psychology Review
- British Journal of Health Psychology

Appendix VIII: Cross-tabulations of associations between types of blame.

Appendix 1

Questions determining circumstances of pain onset (cover sheet to Impact of Events Scale).

Please tick the box against the statement which best describes how your pain started:

A) My pain first began because of, or following one particular event. ☐

Briefly say what happened .....

.....

.....

.....

.....

B) My pain did not begin after any particular event. ☐

If you ticked (A), please complete the attached 'Impact of Event Scale'. Please think about the event when completing this scale.

If you ticked (B), please ignore the attached 'Impact of Event Scale'.

Appendix II

Questionnaire regarding pain and personal information, including questions regarding blame for pain onset.

Chronic pain and thoughts about its causes

Participant number..... Date.....

Thank you for taking part in this study. Please could you complete the following questions about some personal details and your pain.

Personal Details

1. Age .....
2. Sex Male / Female
3. Are you working at the moment? Yes / No
4. What was your last paid employment? .....
5. Do you live with anyone at home? Yes / No

   If 'Yes', with whom? .....
6. Have you had any significant events or changes in the 6 months before your pain started? Yes / No
7. Is this your first attendance at the pain clinic? Yes / No

About your pain

1. Where is your pain? (e.g. low back pain) .....
2. How long have you had your pain? .....
3. What do you think caused your pain originally? .....
   .....
4. How much do you blame another person/other people for your pain onset? (please circle a number from 0 to 7; 0 = I don't blame others for my pain onset at all; 7 = I completely blame others for my pain onset).

0	1	2	3	4	5	6	7
Not at all							Completely

Please turn over

5a. How much do you blame yourself for your pain onset? (please circle a number from 0 to 7; 0 = I don't blame myself for my pain onset at all; 7 = I completely blame myself for my pain onset).

0	1	2	3	4	5	6	7
Not at all							Completely

If you circled a number **other than** 0 for question 5a., please answer 5b.

5b. Do you blame (a) your character/personality/the kind of person you are for your pain onset ☐

or (b) your behaviour (i.e. something you did or did not do in the past) ☐

(Please tick one box only)

6. How much do you blame chance for your pain onset? (please circle a number from 0 to 7; 0 = I don't blame chance for my pain onset at all; 7 = I completely blame chance for my pain onset).

0	1	2	3	4	5	6	7
Not at all							Completely

7. How much do you blame the environment for your pain onset? (please circle a number from 0 to 7; 0 = I don't blame the environment for my pain onset at all; 7 = I completely blame the environment for my pain onset).

0	1	2	3	4	5	6	7
Not at all							Completely

8. How much do you blame nothing for your pain onset? (please circle a number from 0 to 7; 0 = I completely blame something for my pain onset; 7 = I don't blame anything at all for my pain onset).

0	1	2	3	4	5	6	7
Completely blame something							Don't blame anything at all

9. If you circled 0 for **all** the questions 4 to 8, please write what you blame for your pain onset .....

10. Have you had any operations because of your pain? Yes / No

11. Has anyone given you a clear explanation as to why you have your pain? Yes / No

12. Are you currently involved with any compensation claims with regards to your pain? Yes / No

13. Are you currently taking any medication for your pain? Yes / No

Appendix III

Written permission from the author to use the Short-Form McGill Pain Questionnaire.

Dr Melzack
Department of Psychology
McGill University
1205 Dr. Penfield Avenue
Montreal
Que. H3A 1B1
Canada

R·U·H
Royal United Hospital Bath NHS Trust

Pain Clinic
Friend's Outpatients
Royal United Hospital
~~Combe Park~~
Bath BA1 3NG
England
27th July 1999

Dear Dr Melzack

I am writing to request your permission to use the Short Form McGill Pain Questionnaire in my research which constitutes part of my Doctorate in Clinical Psychology. I would like to use the measure along with other questionnaires to examine the relationship between attribution of blame and chronic pain, post-traumatic stress and other psychological distress.

If you would like any further information about my research, please do not hesitate to contact me. I can be contacted by post at the Pain Clinic (see address above), by fax: ~~England~~ 01225 825776 (please ~~mark~~ document 'Pain Clinic') or by e-mail: hmr1@soton.ac.uk

I look forward to hearing from you.

Yours sincerely

HMRawle

Heather Rawle
Trainee Clinical Psychologist

Aug 2, 1999
Dear Ms. Rawle,
It is a pleasure to give you permission to use the SF-MPQ.
Ronald Melzack

Royal United Hospital
Combe Park, Bath BA1 3NG
Tel: (01225) ~~123331~~ 824331

FA710

Appendix IV

Letters of ethical approval:

- University ethical approval
- Bath Local Research Ethics Committee
- Royal United Hospital Research and Development Committee

University of Southampton

Department of Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5000
Fax +44 (0)23 8059 4597
Email

FAO Heather Rawle
Clinical Psychology Department
University of Southampton
Highfield
Southampton

23rd July 1999

Dear Heather,

I am writing to confirm you that your ethical application titled, "The impact of type of chronic pain onset on psychological distress: the mediating role of blame attributions", has been given approval by the department.

Should you require any further information, please do not hesitate in contacting me on (01703) 593995.

Yours sincerely,

KNSmith

Kathryn Smith
Academic Secretary

BATH LOCAL RESEARCH ETHICS COMMITTEE
Direct tel/fax: 01225 825725. e-mail: *research.ethics@ruh-bath.swest.nhs.uk*

R·U·H
Royal United Hospital Bath NHS Trust

23 July 1999

Ms Heather Rawle
Trainee Clinical Psychologist
Pain Clinic
Friends' Outpatients
RUH
Bath

Dear Ms Rawle

BA54/99-00 *(please quote this reference on all correspondence)*
The impact of type of chronic pain onset on psychological distress: the mediating role of blame attributions

At the meeting held on 22 July 1999, the Committee considered your application for approval of the above study, comprising the following documents:

- Completed South & West application form
- Protocol
- Questionnaires: Brief symptom inventory; Impact of Event Scale; COPE; McGill pain questionnaire; adapted IPSAQ; Chronic pain and thoughts about its causes
- Patient information letter and consent form
- Consultant/Nurses information sheet
- Consent for participation of patients from Dr T Cook, Dr M Baird and Dr A Souter

The Committee was pleased to approve this interesting study, which may proceed. We look forward to knowing the outcome in due course, and wish you success with your research.

Please note that for all research being undertaken within provider trusts, approval must be obtained from the Culyer Lead prior to commencement of the study.

This Committee is organised and operates according to ICH/GCP and the applicable laws and regulations. Any changes or extensions to the protocol, or additional investigators should be notified to the Committee for approval. Serious and unexpected adverse events should also be notified to the meeting. May we remind you of the Data Protection Act 1984 and the need to conduct the trial in accordance with the Good Clinical Practice Guidelines.

The Committee is required to audit progress of research and to produce a yearly report to the Avon Health Authority and Department of Health. You are therefore required to provide a brief yearly report and a short final report.

Yours sincerely

Dr Peter Rudd
Chairman

Royal United Hospital
Combe Park, Bath BA1 3NG
Tel: (01225) 428331

R·U·H
Royal United Hospital Bath NHS Trust

Date: 20/7/99

Dear Ms. Raisle

Re: Research Project Impact of type of chronic pain onset on psychological distress: Mediating role of blame attributions.

I am pleased to confirm that the above project has been given RUH approval and enclose a signed copy for your records.

Yours sincerely

Cathy Flower.

Cathy Flower
For Professor Horrocks
Chair RUH R&D Committee

Royal United Hospital
Combe Park, Bath BA1 3NG
Tel: (01225) 428331

Appendix V

Participant Information Sheet

Re: Research study on chronic pain and thoughts about its causes

This research project invites you to complete some questionnaires. Below is some information to help you decide whether or not to take part. Please take time to read the following information carefully and discuss it with others if you wish. Take time to decide whether or not you wish to take part. Thank you for reading this.

ABOUT TAKING PART IN RESEARCH

1. Information obtained during the course of the study may help us to understand chronic pain better.

2. It is up to you to decide whether to take part or not. Even if you do decide to take part, you are free to withdraw at any time and without giving a reason. This will not affect the standard of care you will receive. Your doctor will not be upset if you decide not to take part.

3. All the information collected about you during the course of the research will be kept strictly confidential. Any published report of the research will not identify you.

Please also read the enclosed information that is specifically about the study.

Royal United Hospital
Combe Park, Bath BA1 3NG
Tel: (01225) 428331

INFORMATION FOR PATIENTS ABOUT THE STUDY

1. **Study Title**
   Research study on chronic pain and thoughts about its causes.

2. **What is the purpose of the study?**
   Past studies have found that the way people think about causes of events can affect how they cope and feel. The study aims to explore what people with chronic pain think about the causes of their pain and how this may affect how they cope with their pain and how they feel.

3. **Why have I been chosen?**
   You have been chosen to take part because you are a patient at the Royal United Hospital's Pain Clinic. Approximately 90 other patients will be studied.

4. **Who is organising the study?**
   The study is being carried out as part of my Doctoral training in Clinical Psychology (University of Southampton).

5. **What will happen to me if I take part?**
   You will be asked to sign a consent form and complete six questionnaires today whilst waiting for your appointment or treatment. The questionnaires take no more than one hour to complete. You may also be asked to (a) complete a seventh questionnaire today, or (b) complete two of the questionnaires again at a later date (these will be sent to you by post with a stamped addressed envelope included).

6. **Are there any disadvantages in taking part in this study?**
   It is possible that after completing the questionnaires, you may find yourself thinking about your pain and how you feel. If you would like to discuss such thoughts or feelings further and are unable to do so at the time with me, you can contact me at the pain clinic (01225 824331).

7. **What are the possible benefits of taking part?**
   It is hoped that information from the study can help our understanding of the experiences of chronic pain patients and suggest ways in which pain can be managed better.

8. **Confidentiality – who will know I am taking part in the study?**
   All information which is collected about you during the course of the research will be kept strictly confidential. Questionnaire packages will be coded. Therefore your name will not appear on any of the questionnaires you complete but will be recorded on a separate sheet with a number. Any information about you which leaves the hospital will be anonymised so that you cannot be recognised from it.

9. **LREC Approval**

This study has been approved by Bath Local Research Ethics Committee.

10. **What will happen to the results of the study?**

The results of the study will be written in a report and submitted as part of the requirements of the University of Southampton's Doctorate of Clinical Psychology. A copy of the full report will be available at University of Southampton's library. A summary of the results can be obtained from the pain clinic from September 2000.

11. **Contact for further information**

Please do not hesitate to contact me should you require further information about this study. I can be contacted at the pain clinic (01225 824331).

If you would like to contact an 'independent person' for advice or information about the study, Mike Osborn, Clinical Psychologist, can be reached on the same number as above.

Thank you for taking the time to consider this research.

Heather Rawle
Trainee Clinical Psychologist, BA, MSc.

Appendix VI

Consent form

R·U·H

Royal United Hospital Bath NHS Trust

CONSENT FORM

Title of Project: **Research study on chronic pain and thoughts about its causes**

Name of Researcher: Heather Rawle, Trainee Clinical Psychologist, BA, MSc.

Please initial box

1. I confirm that I have read and understand the information sheet for the above study. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time without my medical care or legal rights being affected. ☐

3 I agree to take part in the above study. ☐

Name of patient	Date	Signature
Name of person taking consent (if different from researcher)	Date	Signature
Researcher	Date	Signature

Royal United Hospital
Combe Park, Bath BA1 3NG
Tel: (01225) 428331

Appendix VII

Instructions for authors of target journals:

- Clinical Psychology Review
- British Journal of Health Psychology

CLINICAL PSYCHOLOGY REVIEW

INSTRUCTIONS TO AUTHORS

AIMS AND SCOPE: *Clinical Psychology Review* publishes substantive reviews of topics germane to clinical psychology. Its purpose is to help clinical psychologists keep up-to-date on relevant issues outside of their immediate areas of expertise by publishing scholarly but readable reviews. Papers cover diverse issues, including: psychopathology, psychotherapy, behavior therapy, behavioral medicine, community mental health, assessment, and child development.

Reviews on other topics, such as psychophysiology, learning therapy, and social psychology, often appear if they have a clear relationship to research or practice in clinical psychology. Integrative literature reviews and summary reports of innovative ongoing clinical research programs are also sometimes published. Reports on individual research studies are not appropriate.

SUBMISSION REQUIREMENTS: All manuscripts should be submitted to Alan S. Bellack, Department of Psychiatry, The University of Maryland at Baltimore, School of Medicine, 685 West Baltimore Street, Suite 618, Baltimore, MD 21201-1549, USA. Submit three (3) high-quality copies of the entire manuscript; the original is not required. Allow ample margins and type double-space throughout. Papers should not exceed 50 pages (including references). One of the paper's authors should enclose a letter to the Editor, requesting review and possible publication; the letter must also state that the manuscript has not been previously published and has not been submitted elsewhere. One author's address (as well as any upcoming address change), telephone and FAX numbers, and E-mail address (if available) should be included; this individual will receive all correspondence from the Editor and Publisher.

Papers accepted for *Clinical Psychology Review* may not be published elsewhere in any language without written permission from the author(s) and publishers. Upon acceptance for publication, the author(s) must complete a Transfer of Copyright Agreement form.

COMPUTER DISKS: Authors are encouraged to submit a 3.5" HD/DD computer disk to the editorial office. Please observe the following criteria: (1) Send only hard copy when first submitting your paper. (2) When your paper has been refereed, revised if necessary, and accepted, send a disk containing the final version with the final hard copy. If the disk cannot be converted, the hard copy will be used. (3) Specify what software was used, including which release, e.g., WordPerfect 6.0a. (4) Specify what computer was used (IBM compatible PC, Apple Macintosh, etc.). (5) The article file should include all textual material (text, references, tables, figure captions, etc.) and separate illustration files, if available. (6) The file should follow the general instructions on style/arrangement and, in particular, the reference style of this journal as given in the Instructions to Contributors. (7) The file should be single spaced and should use the wrap-around end-of-line feature, i.e., returns at the end of paragraphs only. Place two returns after every element such as title, headings, paragraphs, figure and table call-outs. (8) Keep a back-up disk for reference and safety.

TITLE PAGE: The title page should list (1) the article; (2) the authors' names and affiliations at the time the work was conducted; (3) a concise running title; and (4) an unnumbered footnote giving an address for reprint requests and acknowledgments.

ABSTRACT: An abstract should be submitted that does not exceed 200 words in length. This should be typed on a separate page following the title page.

STYLE AND REFERENCES: Manuscripts should be carefully prepared using the *Publication Manual of the American Psychological Association*, 4th ed., 1994, for style. The reference section must be double spaced, and all works cited must be listed. Avoid abbreviations of journal titles and incomplete information.

Reference Style for Journals:
Raymond, M. J. (1964). The treatment of addiction by aversion conditioning with apomorphine. *Behavior Research and Therapy, 3*, 287–290.

For Books:
Barlow, D. H., Hayes, S. C., & Nelson, R. O. (1984). *The scientist practitioner: Research and accountability in clinical and educational settings*. Elmsford, NY: Pergamon.

TABLES AND FIGURES: Do not send glossy prints, photographs or original artwork until acceptance. Copies of all tables and figures should be included with each copy of the manuscript. Upon acceptance of a manuscript for publication, original, camera-ready photographs and artwork must be submitted, unmounted and on glossy paper. Photocopies, blue ink or pencil are not acceptable. Use black india ink and type figure legends on a separate sheet. Write the article title and figure number lightly in pencil on the back of each.

PAGE PROOFS AND OFFPRINTS: Page proofs of the article will be sent to the corresponding author. These should be carefully proofread. Except for typographical errors, corrections should be minimal, and rewriting the text is not permitted. Corrected page proofs must be returned within 48 hours of receipt. Along with the page proofs, the corresponding author will receive a form for ordering offprints and full copies of the issue in which the article appears. Twenty-five (25) free offprints are provided; orders for additional offprints must be received before printing in order to qualify for lower publication rates. All coauthor offprint requirements should be included on the offprint order form.

NOTES FOR CONTRIBUTORS

1. The aim of the *British Journal of Health Psychology* is to provide a forum for high quality research relating to health and illness. The scope of the Journal includes all areas of health psychology across the life span, ranging from experimental and clinical research on aetiology and the management of acute and chronic illness, responses to ill-health, screening and medical procedures, to research on health behaviour and psychological aspects of prevention. Research carried out at the individual, group and community levels is welcome, and submissions concerning clinical applications and interventions are particularly encouraged.

The following types of paper are invited:

(a) Papers reporting original empirical investigations

(b) Theoretical papers which may be analyses or commentaries on established theories in health psychology, or presentations of theoretical innovations

(c) Review papers, which should aim to provide systematic overviews, evaluations and interpretations of research in a given field of health psychology

(d) Methodological papers dealing with methodological issues of particular relevance to health psychology

2. The Journal is international in its authors and readers. Contributors should bear the international readership in mind, particularly when referring to specific health services.

3. Pressure on Journal space is considerable and brevity is requested. Papers should normally be no more than 5000 words.

4. Supplementary data too extensive for publication may also be deposited with the British Library Document Supply Centre. Such material should be submitted to the Editors together with the article for simultaneous refereeing. Further details of the scheme are given in the *Bulletin of the British Psychological Society*, 1977, **30**, February, p. 58. A copy of that note may be obtained from the Journals Department.

5. This Journal operates a policy of blind peer review. Papers will normally be scrutinized and commented on by at least two independent expert referees as well as by an editor or associate editor. The referees will not be made aware of the identity of the author. All information about authorship including personal acknowledgements and institutional affiliations should be confined to a removable front page and the text should be free of such clues as identifiable self-citations ('In our earlier work...'). The paper's title should be repeated on the first page of text.

6. The editors will reject papers which evidence discriminatory, unethical or unprofessional practices.

7. Submission of a paper implies that it has neither been published elsewhere nor is under consideration by another journal.

8. In preparing material for submission authors should follow these guidelines:

(a) Contributions must be typed in double spacing with wide margins and on only one side of each sheet. Sheets must be numbered. Four good copies of the manuscript should be submitted and a copy should be retained by the author.

(b) Tables should be typed in double spacing, each on a separate sheet of paper. Each should have a self-explanatory title and be comprehensible without reference to the text.

(c) Figures are usually produced direct from authors' originals and should be presented as good black and white images preferably on high contrast glossy paper, carefully labelled in initial capital/lower case lettering with symbols in a form consistent with text use. Unnecessary background patterns or lines and shading should be avoided. Paperclips leave damaging indentations and should be avoided. Any necessary instructions should be written on an accompanying photocopy. Captions should be listed on a separate sheet.

(d) The Editors propose to adopt structured abstracts and all articles should be preceded by a structured abstract of between 100 and 250 words (less in the case of a short paper), giving a concise statement of the intention and results or conclusions of the article. Authors requiring further details on structured abstracts should contact the Journals Department (details on inside front cover).

(e) Bibliographic references in the text should quote the author's name and the date of publication thus: Hunt (1995). Multiple citations should be given alphabetically rather than chronologically: (Blackburn, 1996; Fortheringhame, 1994; Norman, 1995). If a work has two authors, cite both names in the text throughout: Choi and Salmon (1995). In the case of reference to five authors, use all the names on the first mention and *et al.* thereafter except in the reference list. For six or more, use *et al.* throughout.

(f) References cited in the text must appear in the list at the end of the article. The list should be typed double spaced in the following format:

Hunter, M. (1994). *Counselling in obstetrics and gynaecology*. Leicester: The British Psychological Society.

Pruitt, S.D., & Elliott, C.H. (1989). Paediatric procedures. In M. Johnstone & L. Wallace (Eds.), *Stress and medical procedures* (pp. 157–174). Oxford: Oxford University Press.

Ray, C., Phillips, L., & Weir, W.R.C. (1993). Quality of attention in chronic fatigue syndrome: Subjective reports of everyday attention and cognitive difficulty and performance on tasks of focused attention. *British Journal of Clinical Psychology*, *32*, 357–364.

Note that journal titles are cited without abbreviation.

(g) Measurements should be in units of the International System. A guide to these is included in The British Psychological Society's *Style Guide*, available at £3.50 per copy from the Society at St Andrews House, 48 Princess Road East, Leicester LE1 7DR, UK.

(h) If the title of the article is longer than 80 characters, a short title should be provided for use as a running head.

(i) Footnotes are expensive to set and should be avoided.

(9) Proofs are sent to authors for correction of print but not for rewriting or the introduction of new material. Fifty complimentary copies of each paper are supplied to the senior author, but further copies may be ordered on a form supplied with the proofs.

(10) Authors should consult the Journal editor concerning prior publication in any form or in any language of all or part of their article.

(11) To protect authors and journals against unauthorized reproduction of articles, The British Psychological Society requires copyright to be assigned to itself as publisher, on the express condition that authors may use their own material at any time without permission. On acceptance of a paper submitted to the Journal, authors will be requested to sign an appropriate assignment of copyright form.

Appendix VIII

Cross-tabulations of associations between types of blame

Blame was assessed using an 8-point rating scale (0 = not at all to blame; 7 = completely to blame). The cross-tabulations were constructed by collapsing the ratings to indicate no blame (i.e. rated zero) and some blame (i.e. rated above zero).

The tables show that the most likely type of blame to be associated with other types of blame was chance. The least likely combinations of blame were other-blame with nothing-blame, and self-blame with environment-blame.

	Blame Other	
	Rated zero	Rated above zero
Blame chance : Rated zero	25	7
Rated above zero	42	26
Blame environment: zero	60	20
Rated above zero	7	13
Blame nothing: Rated zero	27	24
Rated above zero	40	9
Blame self: Rated zero	46	19
Rated above zero	21	14

	Blame Self	
	Rated zero	Rated above zero
Blame chance: Rated zero	23	9
Rated above zero	42	26
Blame environment: zero	54	26
Rated above zero	11	9
Blame nothing: Rated zero	31	20
Rated above zero	34	15

	Blame Chance	
	Rated zero	Rated above zero
Blame environment: zero	30	50
Rated above zero	2	18
Blame nothing: Rated zero	13	38
Rated above zero	19	30

	Blame Nothing	
	Rated zero	Rated above zero
Blame environment: zero	43	37
Rated above zero	8	12

Critical Review

Overall, the study appeared successful as an initial attempt in exploring the associations between blame and circumstances of pain onset and adjustment. The results suggested areas of further research regarding blame in chronic pain (e.g., other-blame and post-traumatic stress symptoms) and one of the strengths of the study was the relatively large number of participants (n = 100). However, there are several limitations to the study.

The cross-sectional design did not enable the direction of causality between the variables to be determined. Ideally, one would assess blame and adjustment variables soon after the initial pain onset, perhaps when patients first visit their GP. The assessment could then be completed again a few months later if patients visit their GP again and they meet the criteria for chronic pain. This would ensure a prospective link between blame and adjustment. Such a design would also ensure the use of patients who are not yet attending a chronic pain clinic. This is desirable as research has found that using pain clinic patients leads to selection biases (e.g., they tend to be more disturbed) which limits the generalisability of results to the general population of chronic pain patients (see Crombie & Davies, 1998, for review of selection biases in chronic pain research). The current study used a chronic pain clinic sample. The researcher attempted to limit selection biases by initially only requesting participation from new patients who had not yet seen the consultant. However this proved impractical and since it would have severely restricted the sample size, the inclusion criteria were broadened to include all patients.

Since blame was the focus of the study it was important to ensure the use of a method of assessing blame that was valid and reliable. However, a thorough search of the published literature into blame did not produce a suitable measure and the author of the present study had little option but to develop a method of assessing blame. Some psychometric properties were examined but time prevented a detailed investigation and the exploratory nature of the measure was emphasised in the empirical paper. Therefore, it is important to acknowledge the lack of information regarding the validity and reliability of the items.

The only existing measures of blame that explored psychometric properties are those that measure tendency to blame rather than blame for a specific event, e.g., blame when domestic violence occurs or blame when people commit criminal acts. Of course, these were not suitable to adapt for the present study. The one method of assessing blame for pain used in previous research (DeGood & Kiernan, 1996) did not explore any psychometric properties and was not considered suitable anyway due to its own limitations (e.g., using the word 'fault'; not allowing blame to more than one source, or blame of chance; not making it clear if the fault was about the initial pain onset or the ongoing experience of chronic pain).

Although the measure of blame used in the study was developed after considerable thought (interviews with patients and an extensive examination of the blame literature), the measure could be improved if more time were available. First of all, the concept of blame in chronic pain patients could be explored using a qualitative approach whereby semi-structured interviews could be carried out with patients. This could lead to a more detailed questionnaire based on themes arising from interviews,

perhaps with several items relating to each type of blame (i.e., self, other, chance, etc.). It would then be possible to explore psychometric properties such as internal consistency.

Questionnaires using response methods such as rating scales have been shown to be preferred methods of measuring beliefs related to causes of illnesses and to elicit similar responses to those obtained by interviews (see Marteau & Senior, 1997, p.256-7). Attribution research has shown that self-report rating scales have a greater relationship with adjustment than when forced-choice methods are used (Taylor, Lichtman & Wood, 1984). In addition, causal attribution ratings have been found to be higher when made by participants compared to when judges rate participants' responses to open questions (Vieyra, Tennen, Affleck, Allen & McCann, 1990). A measure of blame when illness-related problems occur (for renal disease) was found after the present study had begun (Rich, Smith & Christensen, 1999) which explored psychometric properties. This method could be adapted for future studies by asking people to write a description of their initial pain onset and then answer questions in relation to it in order to determine type and degree of blame. Alternatively, a simple alteration to the present measure would be to include an open question regarding what the person blames for their initial pain onset in order to verify their responses to the other blame items. Interviews could also explore if people's blame for initial pain onset is different to their blame for ongoing pain. If differences were found, this would have implications for the wording of questions in future research into the role of blame in chronic pain patients. Also, it was assumed in the study that people did

search for explanations for their pain but interviews would enable the verification of this by exploring whether blame is mentioned spontaneously.

Other measures used in the current study could be improved upon. The COPE was chosen to assess the use of adaptive and maladaptive coping strategies as it had been recommended for use with chronic pain patients (Skevington, 1995) and it was easily available. This meant that data collection could begin promptly, therefore maximising the chance of a relatively large sample size. However, after beginning the study, other measures were discovered in the chronic pain literature which may have been more appropriate. For instance, the Coping Strategies Questionnaire (Rosensteil & Keefe, 1983) was developed on a chronic pain sample and can be divided into active and passive coping scores. This may have been a better measure to use since studies exploring psychometric properties have used chronic pain samples (e.g., Snow-Turek, Norris & Tan, 1996).

It was important to limit the number of questionnaires given to participants and so the Brief Symptom Inventory (BSI) Hostility subscale was used to indicate associations between other-blame and anger. This seemed an appropriate choice given that previous research has demonstrated associations between other-blame and the hostility subscale of the long form of the BSI (Vieyra et *al.*, 1990) and also a relationship between the BSI Hostility subscale and circumstances of pain onset (Thorne, 1998). However, given the debate about the conceptual distinctions regarding hostility and anger (e.g., Eckhardt, Barbour & Stuart, 1997), it may have been more appropriate to use a measure of anger such as the State-Trait Anger Expression Inventory (Spielberger, 1988).

The space constraints of the literature review resulted in the content focussing on the proposed consequences of blame (in terms of adjustment) and the role of other-blame in chronic pain patients. However, it would also have been interesting to explore how other types of blame could be related to the experience of chronic pain patients and also the predictors of blame besides the circumstances of pain onset. For instance, Tennen & Affleck's (1990) model proposes that attributional style will influence the incidence of other-blame as people who tend to make external causal attributions would be more likely to blame others for misfortunes. Originally, the study aimed to explore this relationship in addition to the other aims. The Attributional Style Questionnaire (ASQ, Peterson, Semmel, Von Baeyer, Abramson, Metalsky & Seligman, 1982) was considered unsuitable since it combines 'other people' and 'circumstances' in the externality dimension. These aspects should be separated in order to investigate blaming other people. Therefore, as advised by Dr Kinderman (personal communication), a 12-item measure was developed by the author of the present study that considered internality on three dimensions (internal, external-personal, and external-situational), globality, stability and controllability. Items and response methods were based on the ASQ, the Interpersonal, Personal and Situational Attributions Questionnaire (IPSAQ, Kinderman & Bentall, 1996), and the Extended ASQ (Metalsky, Halberstadt & Abramson, 1987). This measure was completed by 70 of the patients who participated in the present study. Test-retest reliability and other psychometric properties were investigated based on the methods used by Kinderman & Bentall (1996). However, validity and reliability relating to the dimensions particularly relevant to other-blame (i.e., the internality/externality items)

were poor. Also, responses seemed to be confounded with the chronic pain experience of patients and it became clear that only a prospective study was useful in order to determine a causal relationship between attributional style and blame. For these reasons and given the space constraints of the papers, it was decided to omit this attempt from the write-up.

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