

The causes of low back pain: a network analysis

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Abstract

Beliefs regarding the cause of low back pain differ between individual sufferers and health care professionals. One consequence of this is the potential acquisition of maladaptive attitudes and behaviour in relation to pain, and increases in the utilisation of primary care services (Health Expect.3(3) (2000) 161). Methods that have been used to elicit the causal interpretation of social phenomena are varied yet they are unable to categorically demonstrate the different weightings or levels of importance that individuals may assign. The diagram method of network analysis allows individuals to spontaneously consider pathways they believe to be critical to a target event and to determine the strength of those pathways.

Seventy-one completed diagrams indicating the causes that sufferers perceived to be related to low back pain were analysed. The mean number of direct causal paths was 5.61 (SD=3.25) and mean number of indirect causal links was 1.16 (SD=2.34). A significant correlation between path frequency and path strength was also found ($r=0.76$, $p=0.001$). Sufferers do not have an overtly complex view of the causative factors of low back pain but were able to define four core contributory causes (disc, sciatica, lifting, and injury) and one indirect pathway between lifting and injury. There was a clear delineation between external (biomedical) and internal (person-related) factors that were attributed to low back pain acquisition. By determining these causal attributions it is proposed that treatment packages could be tailored to address biases in thinking. This may be particularly useful for those individuals who attribute their pain as a consequence of external (or biomedical) causes.

Author Keywords: Low back pain; Network analysis; Causal attributions; UK

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Introduction

Low back pain is a pervasive health care problem. In 1993, the prevalence of back pain involved sixteen and a half million people in the United Kingdom; the annual costs of back pain to the National Health Service were £480 million, whilst the costs of Department of Social Security benefits were estimated at £1400 million ([Clinical Standards Advisory Group, 1994](#)). Since these data were reported further economic analysis has revealed that the incidence is rising, and with it the associated costs. [Maniadakis and Gray \(2000\)](#) proposed that in 1998, in the United Kingdom, the direct health care cost of back pain was £1632 million and the cost of informal care and related production losses totalled £10668 million. The prevalence appears to be increasing and this may be associated with cultural changes that have influenced people's awareness of more minor back symptoms and their willingness to report them ([Palmer, Walsh, Bendall, Cooper, & Coggan, 2000](#)) and the attitudes and beliefs held by referring physicians ([Buchbinder, Jolley & Wyatt, 2001](#)).

Chronic low back pain is defined as pain within the lumbosacral region, buttocks and thighs that is 'mechanical' in nature: it varies with physical activity and varies with time ([Waddell, 1998](#)). Additionally, it is seen to be pain that persists beyond twelve weeks ([Clinical Standards Advisory Group, 1994](#)) or that lasts beyond the expected period of healing ([Andersson, 1999](#)). However, as highlighted by [Evans and Richards \(1996\)](#) there is no standard or agreed definition of chronic low back pain and there remains continuing uncertainty and disagreement within the medical professions related to its cause, nature and treatment. This is problematic, not least because a failure to reach consensus regarding the cause and nature of it can lead to inconsistencies in the advice and subsequent treatment that is given to individuals suffering with low back pain. Such inconsistencies may also shape the beliefs held by the sufferers in relation to their pain. Furthermore, aside from the physical sensation of pain, chronic low back pain is characterised by the impact that it has on the individual's life. Associated anxiety, depression and stress are frequently cited as having an additional major impact on the lives of those who live with persistent low back pain ([Croft et al., 1995](#); [Main, 1983](#); [Sullivan, Reesor, Mikail, & Fisher, 1992](#); [Averill, Novy, Nelson, & Berry, 1996](#)). The presence of pain is seen as a symptom that requires the help of a physician and may lead to the search for a cure as long as the pain continues ([McCracken, 1998](#)). Patients who are more obdurate in their attempts to avoid or rid themselves of pain, and consequently view pain as an absolute barrier to a better life suffer more ill consequences. Those individuals who are unaccepting of their pain appear to be facing the distress that comes from attempting to control an unchangeable aversive experience ([Thompson, 1981](#)).

Despite this emerging body of predominantly biomedical evidence that attempts to explain or account for the continuance of pain, an equally valid perspective is proposed that challenges the 'dominant medico-scientific' approach ([Williams & Bendelow, 1998, p. 169](#)) to the study of pain. This different point of view questions the validity of the dominant dualist explanations provided to construe pain, whereby 'pain and meaning' ([Morris, 1991, p. 44](#)) are viewed separately, when in fact they should be seen as interdependent. Moreover, [Morris \(1991\)](#) suggested that when we begin to explore the multiple meanings that can be assigned to the pain experience these often prove to be 'at least as important as the medicines we consume' (p.44). There is a clear need therefore, for the meanings that sufferers ascribe for their pain and beliefs relating to its cause to be explored.

Beliefs are assumptions about reality that serve as a perceptual lens or a 'set' through which events are interpreted ([Lazarus & Folkman, 1984](#)). In relation to health and illness evidence attests to the differing beliefs that are held between the lay populace and the medical profession to account for the causes of wellness and disease ([Herzlich, 1973](#); [Pill & Stott, 1982](#); [Blaxter, 1982](#); [Helman, 1984](#); [Davison, Davey Smith, & Frankel, 1991](#)). Early explanations to account for these differences centred on the social psychological models of help-seeking behaviour and decision-making ([Uehara, 2001](#)) and the prominence of the mind-body dualism entrenched within the traditional biomedical culture (see [Ferguson & Marras, 1997](#)). However, [Williams \(1984\)](#) suggests that lay theories of aetiology serve to 'reconstruct some sense of order from the fragmentation produced by illness' (p. 177) and in so doing common sense explanations of illness can be seen to develop to provide a vehicle to enable this reconstruction to occur. [Peters, Stanley, Rose, and Salmon \(1998\)](#) nevertheless suggest that lay and medical beliefs are not separate systems and echo [Shorter's \(1992\)](#) view that medical accounts of common physical symptoms continually diffuse into lay discourse about biology and illness. Low back pain is one such common physical complaint where this diffusion is evident. However, it must be noted that there are conceptual differences between lay knowledge (where lay people may have particular insights into the phenomenon of low back pain) and the phenomenon specific knowledge that patients or sufferers of low back pain have as a direct consequence of their pain experience. Whilst both are equally valid it is the latter that places patients and sufferers in a unique position because of this insider knowledge ([Arskey, 1994](#)).

Beliefs regarding the cause of low back pain differ between individual sufferers and health care professionals ([Cedraschi, Reust, Roux, & Vischer, 1992](#); [Skelton, Murphy, Murphy, & O'Dowd, 1995](#); [Hermoni et al., 2000](#)). [Borkan, Reis, Hermoni, and Biderman \(1995\)](#) found that individual sufferers articulated several factors to account for their pain and these formed the basis for the beliefs that they held. The diversity of these factors ranged from explanations that the presence of pain was due to: an injury; degeneration of the spine; as a punishment; stress and the weather. Additionally, factors such as 'work' 'ageing' and 'childhood injuries' (p.983) were seen to constitute the primary causes of low back pain. More recent findings suggest that perceptions about the cause of low back pain being due to 'a slipped disc' or 'a trapped nerve' are factually incorrect and may account for increased utilisation of primary care services ([Moffett, Newbronner, Waddell, Croucher, & Spear, 2000](#)). This demonstrates the point highlighted by [Shorter \(1992\)](#) whereby medical terminology becomes ingrained into everyday lay language; one consequence of

which is that a poor understanding of the pain symptoms may lead to the acquisition of maladaptive attitudes and behaviour in relation to the pain ([Geisser & Roth, 1998](#)).

Methods that have been used to elicit the causal interpretation of social phenomena are varied and include semi-structured interviews ([Antaki, 1985](#); [Litton & Potter, 1985](#)) and spontaneous discourse ([Campbell & Muncer, 1987](#)). Explication of beliefs and meaning regarding illness perception has previously occurred via an exploration of lived experience. Utilising patient narrative accounts [Kleinman \(1988\)](#) explored and mapped an explanatory lay model to elucidate how illness conditions were conceptualised and how these conceptualisations impacted on treatment choice, compliance and outcome. Other research has shown how individuals perceive hypertension ([Greenfield, Borkan, & Yodfat, 1987](#)) comprehend hip fracture ([Borkan, Quirk, & Sullivan, 1991](#)) and make sense of everyday pain ([Aldrich & Eccleston, 2000](#)). Through direct questioning [Underwood \(1997\)](#) was able to determine patients' beliefs as to the cause of their low back pain. Whilst research evidence provides insight to the extent of lay knowledge, [Furnham \(1994\)](#) argues that the structure and determinants of lay beliefs about health and illness are not considered. He contends that scrutiny is needed to understand those factors that are pivotal to the construction of the meanings given. In support of this position [Aldrich and Eccleston \(2000\)](#) highlight the paucity of detailed work that describes how meaning is created. One mechanism by which this can be achieved is through the exploration of causal attributions. [Roesch and Weiner \(2001\)](#) stated that attributions were constructs worthy of consideration because they allowed an understanding of the world as it had occurred in the past (both recent and distant) but they also served to guide future behaviour. One caveat to this approach is offered by [Peterson and Seligman \(1984\)](#) and [Forsterling \(1986\)](#) whom have highlighted the role that personal biases may play in the (mis)interpretation of events. [Russell \(1998\)](#) however, has argued that the most fair and ethical approach to a consideration of health outcome is to use the perceptions of the service users as it places delivered care in the context of people's expectations. It is within this context that condition-specific attributions should be explored.

Whilst useful, these methods of inquiry are unable to categorically demonstrate different weightings or levels of importance that individuals may assign to any given set of condition-specific causative determinants. Consequently certain factors may be deemed as less important or irrelevant and this may not be elicited through the analyses chosen. More recently however, network analysis has been utilised to overcome some of these issues.

Early network studies employed a grid method (see [Fig. 1](#)) to determine whether respondents perceived there to be a link between causal factors in relation to one target variable ([Litton & Potter \(1985\)](#) and [Heaven \(1988\)](#); [Heaven, 1994](#)).

	Disc	Stress	Sitting	Lifting	Injury
Disc	0	1	0	1	1
Stress	1	0	0	1	1
Sitting	1	0	0	0	0
Lifting	1	1	0	0	1
Injury	0	1	0	0	0

(5K)

Fig. 1. Example of grid method.

Participants are presented with a grid in which the proposed causes of a social phenomenon (such as employment, loneliness or poverty) are presented vertically and horizontally along the top and sides of a grid. Individual respondents are asked to determine whether there is a link between each of the factors. Agreement was originally indicated by either a binary scale in which individuals enter a '1' if they perceive a link or a '0' if they do not (see [Lunt, 1988](#); [Campbell & Muncer, 1990](#); [Lunt & Livingstone, 1991](#); [Muncer, Epro, Sidorowicz, & Campbell, 1992](#)).

More recent studies have tended to replace the binary form with a Likert scale in which participants rated the strength of a link on a scale of 1–5 (see [Lunt, 1991](#); [Heaven, 1994](#)). Individual grids are then aggregated and networks constructed hierarchically with the strongest link entered first. Network construction stops according to a cause-to-link ratio calculation in which new links are added to the network provided that this does not entail adding a large number of other causal links from causes that already appear on the network.

Criticisms of this method centre on the fact that the resulting networks are not the production of any one individual but instead represent an aggregated response format ([Muncer & Gillen, 1997](#)). They are often, therefore, not representative of what participants perceive as the causal links and generally produce larger networks with more causal links than are perceived by any one participant ([Muncer & Gillen, 1997](#)). Additionally, the grid method requires respondents to consider possible links between causes that may account for the target variable (e.g., loneliness) rather than direct links between the causes and the target variable. It has subsequently been found on many occasions that the direct links between the causes and the target are often considered more important than the links between causes ([Heffernan, Green, McManus, & Muncer, 1998](#); [Muncer, Taylor, & Ling, 2001b](#)). Furthermore, a target factor may have reciprocal causal links with the nominated causes. In a recent network study of the causes of health and illness, participants' representations suggested that stress was an important cause of illness and that illness was an important cause of stress ([Muncer et al., 2001b](#)). Preventing individuals from representing direct paths (as in the earlier grid method) may yield an over complex representation on the one hand (see, for instance, the representation elicited in [Lunt, 1991](#)), as individuals seek to encode their perceptions within the constraints of proposed causes, and, on the other hand, it may limit the subtlety of the representation by precluding the representation of connections to the target factor that are both direct and indirect from a given causal factor. Likewise, there is no place within the grid method for free choice by respondents to include factors that they may perceive as relevant but are not included within the framework of the grid. The diagram method (see [Fig. 2](#)) proposed by [Green and McManus \(1995\)](#) circumvents these issues.

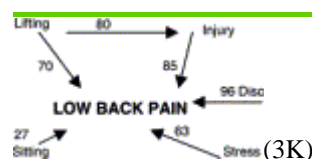


Fig. 2. Example of diagram method.

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[Green and McManus \(1995\)](#) required individuals to draw a network diagram of the risk factors for a target factor, coronary heart disease. Unlike previous network elicitation studies, individuals were not forced to consider each and every connection amongst the factors but were free to sample as they wished. Individuals represented a causal relationship between two factors by drawing a line connecting them and indicated the direction of the causal influence using an arrowhead. Also in contrast to previous studies, the target factor (coronary heart disease) was explicitly represented. In the diagrams created, a possible causal factor may be connected to the target factor in a variety of ways. A causal factor may have a direct path to the target factor, or it may have an indirect path to it via some other factor, or it may have both a direct path and an indirect path to the target factor. The diagram represents what individuals spontaneously consider the critical pathways.

In addition to representing a path, individuals were required to rate the strength of each causal path on a scale from zero to one hundred. A composite diagram was constructed that indicated both the percentage of individuals including each causal path and the mean strength of those causal paths. [Green and McManus \(1995\)](#) showed that the total path strength of a factor (the strength of both direct paths and all indirect paths) predicted participants' ratings of the effectiveness of different actions based on each of the risk factors in reducing the risk of coronary heart disease.

In a subsequent study that examined perceptions of the factors increasing a person's prospects of employment, [Green, McManus, and Derrick \(1998\)](#) confirmed the importance of path strengths in predicting the ratings of the effectiveness of different actions designed to increase a person's employment prospects. [Muncer, Taylor, Green, and McManus \(2001a\)](#) have also used the diagram drawing method to investigate nurses' representations of the causes of work-related stress. In this study they also investigated the reliability and validity of the resulting networks by comparing the network produced with that produced from a previous network study and also with the findings of a large-scale study of stress in the National Health Service ([Haynes, Wall, Boplden, Stride, & Rick, 1999](#)). In the latter case, the pattern of correlation between the nine dimensions of the causes matched many of the features of the network diagram.

Within the diagram method of network construction individuals are presented with a list containing factors that may cause the target variable but additionally there is opportunity to incorporate other factors that the respondent may feel is personally relevant. Individual respondents are required to draw a line to represent the link between the causal factor and the target variable. They are also required to indicate the strength of this cause by providing a value from, for example, 0 to 100 ([Green et al., 1998](#)). Of particular relevance to these network studies is that to date, the majority have utilised samples of participants that are comprised of undergraduate students. As a result, these studies, whilst demonstrating the efficacy of the methodology, may be problematic due to the elicitation of causal attributions to social phenomena from individuals who do not perceive the target variable to be personally relevant at the time of grid construction (e.g., loneliness). The exception to this are the studies which explored factors that were perceived to be contributory to work-based stress in nurses and utilised a sample consistent with the research aim ([Taylor, White, & Muncer,](#)

[1999](#); [Muncer, Epro, Sidorowicz, & Campbell \(1992\)](#) and [Muncer, Taylor, Green, and McManus \(2001a\)](#)). It could be argued therefore, that personally experienced phenomena would hold a different meaning in comparison to those who are asked to consider an abstract or personally meaningless concept and consequently this may impact on the network that is derived.

No studies to date have utilised network analysis to elicit those factors that low back pain sufferers perceive cause low back pain. This research is important for two reasons. Firstly, it is needed to develop our understanding of the complexities inherent in the casual attributions sufferers make to account for their pain. Secondly, it may provide additional evidence to support claims made by Moffet et al. (2000) that perceived causes that are factually incorrect may lead to greater utilisation of health care services.

Method

Participants

Two hundred and thirty four low back pain sufferers comprised the initial sample pool. These patients were recruited as part of a larger study exploring expectations of low back pain treatment. Consecutive patients referred to two North East of England Spinal Assessment Clinics and two North East of England Pain Clinics comprised the study participants. One element of this larger study required individuals to indicate what they believed the cause of their back pain to be. One hundred and eleven participants failed to provide an answer or stated that they didn't know (77 and 34, respectively). The remaining 123 participants provided 18 different reasons to account for the cause of their pain ([Table 1](#)). There were no statistically significant differences in relation to age, gender or duration of pain between those participants who did provide a cause and those who did not.

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Table 1. Eighteen causes of low back pain identified from original sample

Low back pain causes
Wear and tear
Arthritis
Sciatica
Disc problem
Joint problem
Trapped nerve
Weak muscles
As the result of an injury
Lifting and carrying
Sitting for too long
Standing for too long
Walking
Driving
Lying down
Not enough rest
Specific movements whilst working
Stress
No pain medication

The 123 participants were asked to participate further by completing a network diagram. Seventy-one completed diagrams (57.7%) were submitted for analyses. Again, no statistically significant differences were found in relation to age, gender or duration of pain between those participants who provided a diagram and those who did not. Ages of participants ranged from 18 to 83 (mean 45.24) and there were 31 males and 40 females.

Design

All 71 participants constructed a single diagram indicating the causes that they perceived to be related to low back pain.

Procedure

Individuals providing a reason to account for low back pain were asked to participate in the network study. From the original eighteen causes ([Table 1](#)) that were identified fifteen were selected to be included within the instructions to participants. The remaining three causes (wear and tear, not enough rest and specific movements when working) were omitted as we perceived these to be either too general or were specific to participants who were working. It was also felt that as the diagram method enables participants to include any perceived causes of their low back pain within their networks that the omission of the three causes from the list would not impact on the derived networks. Participants were therefore provided with an A4 piece of paper. Placed in the centre of the paper were the words LOW BACK PAIN. Above this were the following instructions:

There are many reasons why people develop low back pain. Possible causes are: sciatica, trapped nerves, arthritis, an injury, lifting and carrying, driving or being in a

car, stress, disc problems, no painkillers, lying down, weak muscles, joint problems, standing, sitting and walking.

Write down the causes from the list that you think cause back pain. You can also add any other causes that you think of. Also give the cause a number from 1 to 100 to show how strong you think that cause is. 1=a weak link, 100=a very strong link.

Participants were also given an example of a previous drawn diagram featuring causes of coronary heart disease. Each participant constructed the diagram individually. After completion, participants were thanked and provided with information detailing how their completed diagram would be collated with those provided by other low back sufferers to produce an overall model of the related causes of low back pain.

Results

A composite diagram was prepared by examining the separate paths between any two factors for each of the 71 participants. Participants produced mainly direct paths between the causes and the target back pain, with only 35.8% of participants including indirect causal paths between causes. The mean number of direct causal paths was 5.61 with a standard deviation of 3.25 and the mean number of indirect causal links was 1.16 with a standard deviation of 2.34. Causal paths that were endorsed by at least 10 per cent of participants ([Green & McManus, 1995](#)) are listed in [Table 2](#).

Table 2. Paths proposed by at least 10% of participants, with mean path strength

Pathway	n	Percentage	Mean strength
Disc→LBP	42	59.15	72.3571
Lift→LBP	40	56.34	71.3500
Sciatica→LBP	37	52.11	80.4054
Injury→LBP	36	50.70	74.8611
Nerves→LBP	34	47.89	70.0000
Arthritis→LBP	32	45.07	70.4688
Muscles→LBP	31	43.66	62.9032
Sit→LBP	26	36.62	54.2308
Stand→LBP	26	36.62	49.6154
Joints→LBP	23	32.39	63.2609
Walk→LBP	20	28.17	49.0000
Stress→LBP	20	28.17	48.2500
Driving→LBP	13	18.31	46.1538
Lying→LBP	10	14.08	34.4000
Painkillers→LBP	8	11.27	64.3750
LIFTING	8	11.27	61.2500

Disc problems, lifting and carrying, sciatica and injury are included on at least 50 per cent of participants' diagrams. *Lifting and carrying to injury* is the only indirect path that meets the 10% criterion.

[Fig. 3](#) shows the composite diagram from all causes. The various proportions of individuals (10–29%, 30–49%, 50–100%) who included a particular path are shown by lines of different types.

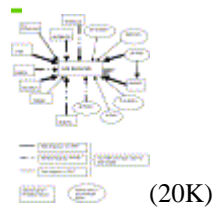


Fig. 3. Composite network diagram of the perceived causal paths of low back pain. Only paths drawn by more than 10 per cent of participants are represented.

We also calculated for each path its mean strength, using the scores given by those who included it. The mean strength of each path is represented by different line widths. Thus we are able to separate path frequency, which is the proportion of participants who included a particular path, and path strength. Participants in this study did not include any reciprocal paths from back pain to any of the causes.

The most frequently included paths in this network also have high path strengths of over 70. The correlation between path frequency and path strength is also significant ($r=0.76$, $p=0.001$). It is noteworthy however, that the indirect path between *lifting and carrying* and *injury* has quite a high path strength (over 60) even though it has low path frequency.

Previous work has already gone some way towards establishing both the reliability and validity of models derived from network analysis ([Green & McManus, 1995](#); [Heffernan et al., 1998](#); [Muncer, Epro, Sidorowicz, & Campbell \(1992\)](#) and [Muncer, Taylor, Green, and McManus \(2001a\)](#)). We can provide a weak check on the reliability by randomly splitting the sample in two and comparing the two groups for path inclusion. In this case two randomly selected groups were not significantly different ($p>0.05$) for frequency of inclusion of any path appearing on the diagram in [Fig. 3](#).

Content analysis

In addition to the elicitation of the main causative pathways, quantitative content analysis was performed on the other causes that individual sufferers perceived also contributed to the cause of their low back pain. Twenty-seven participants provided a total of 33 responses that account for 16 other causes of low back pain. These are shown in [Table 3](#).

Table 3. Additional factors provided to account for low back pain

Cause	n	Percentage ^a
Posture	7	21.2
Gardening	4	12.1
Lack of exercise	4	12.1
As a consequence of a related disease	2	6.1
Hereditary factors	2	6.1
Painful/heavy menstruation	2	6.1
Pregnancy and childbirth	2	6.1
Sport	2	6.1
Back spasm	1	3.0
Being overweight	1	3.0
Caring for a sick relative	1	3.0
Due to hysterectomy	1	3.0
Menopause	1	3.0
Poor self care (of back)	1	3.0
Side-effect of other medication	1	3.0
Unhealthy diet	1	3.0

Similar to the main causes that were extracted via the network analysis, the related perceived causes of low back pain were attributed to either a physical condition or a specific person centred factor. Of these 16 additional causes, 7 are physical/exercise related (posture, gardening, lack of exercise, sport, back spasm, caring for a sick relative and poor self care), 4 are related to the female reproductive system (painful/heavy menstruation, pregnancy and childbirth, due to hysterectomy and menopause) and 2 causes are related to diet (being overweight and unhealthy diet). The remaining three causes (as a consequence of a related disease, hereditary factors, side-effect of other medication) can be seen to be distal to the other thirteen, insofar as they reflect factors outside of the control of the individual.

Discussion

The findings from this study have shown that sufferers did not have an overtly complex view of the causative factors of low back pain but were able to define four core (high consensus $\geq 50\%$ consensus) contributory causes of low back pain (disc, sciatica, lifting, and injury) and one indirect pathway between lifting and injury. Moreover, the total number of pathways that were generated ($n=16$) reflects the inherent difficulties found in clinical practice in providing a concise diagnosis for pain arising in the lumbar region. Furthermore, there was a clear delineation between external (biomedical) and internal (person-related) factors that were attributed to low back pain acquisition. Likewise, content analysis of the additional causes provided by participants produced a further sixteen factors that reflected this dualism.

In the data presented by [Borkan, Reis, Hermoni, and Biderman \(1995\)](#) participant accounts also reflected these distinctions. They cited features of their pain that were perceived to be of neurological origin (e.g., disc problems, muscle weakness) or of a mechanical nature (e.g., due to sitting, standing, lifting and driving) or due to

psychosocial factors (e.g., stress). Female participants also reported that back pain was attributed to pregnancy and childbirth. Such explanatory models, derived via qualitative methodologies, certainly add to our understanding of the lived experience of low back pain, but they do not establish the level of consensus amongst the sufferers in relation to the attribution of the cause of low back pain. The findings from our network study therefore, complement and extend those described by [Borkan et al. \(1995\)](#). The use of the diagram approach employed in this network study has enabled the complexities of the causal attributions made by these low back pain sufferers to be revealed. Participants have assigned their own weighting to the factors that they attributed to be the cause of their pain and as a consequence the strength of the consensus regarding the causes of low back pain have been found. Moreover, this approach has afforded insight into the consensual causal determinants of low back pain that would be difficult to achieve using any of the commonly used methods (see for example [Antaki, 1985](#); [Campbell & Muncer, 1987](#); [Litton & Potter, 1985](#) discussed above) and this needs to be considered for future research where the elicitation of causal attributions in relation to pain and illness is to be determined.

Whilst other previous research (Moffet et al, 2000) has shown that low back pain sufferers do use biomedical terminology when describing their pain problems and when recounting the cause of their problem, our findings have revealed the subtleties within such terminological use. As [Table 2](#) shows, of the six pathways with greatest path strength (≥ 70), four are described using medical terminology. Of these, participants have distinguished between causal attributions that are due to nerves, sciatica and discs. This use of biomedical terminology within the everyday language of these participants suggests that contrary to [Geisser and Roth \(1998\)](#) individuals within our sample appear to demonstrate a good understanding of their pain symptoms and are able to attribute these symptoms to simple diagnostic criterion. Whilst the use of such terminology may be factually incorrect and/or inappropriately used [Stacey \(1988\)](#) suggested that "people's ideas are logical and valid in their own right, although they may not be consonant with biomedical science or with any other organised healing system" (p.142). [Chapple, Campbell, Rogers, and Roland \(2002\)](#) also suggest that lay and medical knowledge are not discrete entities and that there is likely to be a certain degree of overlap between the two. [Arskey \(1994\)](#) proposes that patients are placed in a unique position, as they possess practical experience and insider knowledge of their condition. It can be argued therefore, that this privileged knowledge is then used for the active development of a lexicon that embraces the biomedical terminology to create meaning. This 'socialised processing' ([Hewstone, 1983, p.11](#)) of information is mediated via language-based communications to enable individuals to learn about causes and adopt specific hypotheses about the cause of their pain. Furthermore, as our findings revealed so many direct causal pathways for low back pain, this would appear to support [Weiner \(1985\)](#) who suggested that individuals who actively seek the causes of outcomes might subsequently adapt more positively to that outcome. However, the separation between external (biomedical) and internal (person-related) causal attributions may also reflect one method by which individuals have attempted to make sense of their pain and instead may be indicative of maladaptive coping. This can only be alluded to however, as this was not directly considered within this study.

Research on social action suggests that individuals who believe that events are related to their own behaviours are more likely than individuals trusting fate or powers

beyond their control to take steps to change health behaviours. [Phares \(1976\)](#) proposed that the cognitive and motivational aspects of the internal-external dimension leads those individuals who have an internal orientation to a superior position in exerting power and control over their environment. If this is the case, then such distinct internal-external causal attributions may have a significant impact in relation to health maintenance and health service use. [May, Rose, and Johnstone \(2000\)](#) also suggest that lay models highlight the relationship that sufferers have with their pain and as such they may be predictive of the outcome of treatment. The networks produced in this study and presented in the composite diagram ([Fig. 3](#)) represent such a lay model. Over half of the direct paths were attributed to external/biomedical factors and these were also the pathways that were most heavily endorsed. This may reflect the needs of the individuals within this sample to have a diagnosis for their physical pain—to attach a meaningful label to their symptoms that legitimates their pain. [Glenton \(2003\)](#) has argued that such "achievement of disease" (p. 2250) can release the individual from personal responsibility for the pain and enable the individual to assume a sick role position. However, caution must be applied here as six direct pathways were also attributed to internal or person-centred factors, albeit with lower path strength.

The six internal or person-centred causal attributions all relate to activities that are undertaken as part of everyday living and suggest that participants have an awareness of the role that mechanical loads play in both the genesis and maintenance of low back pain. The fact that the only indirect pathway is between lifting and injury further supports this. Eight individuals endorsed this indirect pathway however forty participants included a direct pathway of lifting within their diagrams. We do not know whether forty individuals have attributed lifting a load leads to an injury that in turn leads to back pain yet have not articulated this middle stage in their diagram, nor do we know whether forty individuals believe that after lifting a load no injury needs to transpire for back pain to occur. This failing of the methodology to enable the exact meaning of participants' diagrams to be elicited is problematic. Whilst the pathways demonstrate the consensus of causal attribution the explicit meaning of what exactly the pathways represent remains elusive.

Another limitation with this method is that the fifteen direct pathways derived from participant diagrams reflect the fifteen examples provided in the instructions to participants. This suggests one of two things. Either the participants were primed in their diagram construction by this list of possible causes resulting in the production of an unrepresentative model, or that the list of causes, derived as it was from participants in the larger study, truly reflect the causal attributions of individuals with low back pain. The latter is more likely as each of the 71 participants who produced network diagrams also provided data from which the original list was derived. Because of this the resulting composite network produced by the participants should be seen as representative of the causal attributions of this sample. Furthermore, as 27 participants did include a total of sixteen other specific causes beyond those included in the list, suggests that individuals were able to construct a diagram utilising other causes that were not included within the instructions. It is interesting to note however, that the three omitted causes (wear and tear, not enough rest and specific movements when working) did not appear in any of the networks produced here. One possible reason to account for this could be that those participants who provided these causes for pain from the original study did not later complete network diagrams.

It must also be acknowledged that the composite network was produced from responses from just over half of the participants who provided a cause for their pain in the initial stages of the study. Despite this, a satisfactory response rate resulted. The sample was purposive, that is, all participants were individuals who suffered with back pain. This is an important point as many of the reported network studies (see [Green & McManus, 1995](#); [Green et al., 1998](#); [French, Marteau, Senior, & Weinman, 2002](#)) utilise samples that include individuals who do not have any personal experience of the phenomenon being studied and consequently the resulting networks tend to be complex. The high number of direct pathways and low number of indirect pathways that were generated in our study suggests that when the sample includes individuals with direct personal experience a more direct and consequently less complex model emerges. This has implications for the management of low back pain. Clearly individuals who attribute their pain to an external cause will require a different treatment approach initially, compared to their counterparts who attribute pain to causes within their own control. High dropout and relapse rates ([Kerns & Haythornthwaite, 1988](#); [Turk & Rudy, 1990, 1991](#); [Richmond & Carmody, 1999](#)) following psychological treatment programmes are disconcerting and [Kerns and Rosenberg \(2000\)](#) suggest that ‘efforts to identify individuals prone to these negative outcomes’ (p. 49) should be pursued. The differential attributions found within this study may provide one method by which such individuals may become known.

Allied to this, there is a need to ascertain whether health care utilisation and treatment outcome differs between individuals premised on these causal attributions. By determining the causal attributions that low back pain sufferers make regarding their pain, treatment packages could be tailored to address inaccuracies or biases in thinking. This may be particularly useful for those individuals who attribute their pain as a consequence of external (or biomedical) causes. Finally, with the prevalence of low back pain increasing and with patient expectations near ungovernable ([Pickering, 1996](#)) there is a clear need to set treatment boundaries and likely outcomes within the frame of reference of the individual sufferer. There is a need therefore to determine whether the causal attributions produced by patients are synonymous with those of the doctors/health practitioners to whom they consult. Elicitation of the causal attributions made by these health professionals is needed for two reasons: primarily, as there is little agreement within the medical professions related to the cause, nature and treatment of chronic low back pain it would be useful to have information concerning consensual causal attributions for low back pain from this group of people and secondly, to determine whether the variations in patient attributions reflect possible differences in the information received from different doctors.

There is an opportunity here to use network analysis methodology to identify consensual patient and practitioner data and this could be achieved beyond the specific study of chronic low back pain. Research that examines the causal attributions made by people who experience pain and illness is unable to demonstrate which of the identified causes are assigned greater importance by sufferers and tend to merely uncover a broad range of potential determinants that provides little insight into those that are deemed most important or of greatest relevance. However, if the diagram method considered here were to be used across a range of illness or pain or poor health states (e.g., breast cancer, arthritis or obesity) then more meaningful and consensual data would be produced. On the basis of such data, personally relevant treatment packages and cause-specific educational material could be produced. If this

were to prove effective this may lead to improved outcomes, improved patient satisfaction with delivered care and potentially an overall reduction in health care utilisation.

Conclusion

This network analysis has revealed the complexities, subtleties and consensus of the causal attributions made by low back pain sufferers. This is the first study, to our knowledge, to utilise this method with individuals who have chronic low back pain and it has shown that the individuals within our sample have a good understanding of their pain symptoms and are able to attribute these to simple diagnostic criterion. Utilisation of the diagram method with a sample that has personal experience of the phenomenon under investigation has shown that direct causes are more readily attributed rather than indirect, and individuals tend to differentiate these causes between external and internal factors. Identification of the causal attributions made by health professionals to account for low back pain, in comparison with those of low back pain sufferers, may highlight potential misconceptions from either party and could lead to tailored treatment packages being developed. In so doing this may provide one possible way to stem the increasing prevalence of low back pain.

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