The contribution of psychosocial factors to the development of chronic pain: The key to better outcomes for patients?

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1. Introduction

There is growing evidence that aspects of the psychosocial environment are associated with a higher likelihood of reporting pain, particularly chronic pain (Hoogendoorn et al., 2000; Bongers et al., 2002), including some evidence from longitudinal studies that these aspects are potential risk factors for future development of musculoskeletal pain (Nahit et al., 2003; Hartvigsen et al., 2004). By contrast, results from psychosocial intervention studies for pain have, overall, delivered modest outcomes in community settings (Karjalainen et al., 2001; Hay et al., 2005; Jellema et al., 2005; Jones et al., 2006). Chronic pain is a significant public health problem and we need effective interventions that are deliverable in community settings, so we need to understand the reasons (and they are likely to be multiple) why this has occurred.

The disappointing findings may relate to the quality of the interventions or the skills of those delivering them. Several interventions for pain which have shown null results have provided brief training to those delivering it, e.g., brief training of physiotherapists to deliver behavioural therapy (Jones et al., 2006), which may not equip them with sufficient skills. However, an over-arching reason for disappointing findings from interventions that is shared across studies may be fuzzy thinking about psychosocial factors.

There has been a tendency to refer to psychosocial factors without articulating what they are, and rarely have studies of psychosocial factors included specific consideration of the level at which social factors are operating. Despite considerable data showing that “psychosocial factors” are associated with pain and may predict its onset, there is little consistency on the precise factor(s) which may be imparting risk, and often little clarity about what we are measuring. Consequently, interventions have targeted psychosocial factors in an unsystematic manner, often without regard for the appropriate level at which the intervention should occur. Perhaps significantly, few “psychosocial” interventions seem to have addressed the “social” (or environmental) domains, apart from some community-based studies (e.g. Buchbinder et al., 2001).

2. The biopsychosocial model and psychosocial factors

The biopsychosocial model of pain posits that pain experience and its impact on the individual is a function of interacting combinations of somatic input (e.g. nociception), psychological processes (e.g. beliefs, coping repertoire and mood), and environmental contingencies (i.e. social context associated with significant others, community or cultural rules and expectations) (Turk and Okifuji, 2002). Most working in the pain field is familiar with the various iterations of the biopsychosocial model.
(Loeser, 1982; Waddell et al., 1984; Flor and Hermann, 2004), and many studies have examined some of the connections between its layers. For example, the relationship between psychological factors and biological pathways involved in pain processing has been investigated in research on placebo responses (e.g. Finniss and Benedetti, 2005), expectation (e.g. Keltner et al., 2006), and attention (e.g. Valet et al., 2004). Similarly, the relationships between cognitions (thoughts, beliefs), mood, disability and pain experience have been explored (e.g. Turk and Okifuji, 2002; Keefe et al., 2004a). However, less attention has been given to understanding how connections between the psychological/behavioural and social domains may operate.

In a comprehensive overview of the role of psychological factors in chronic pain, Turk and Okifuji (2002) focus only on the relationship between physical and psychological parameters. In their review of biopsychosocial models of pain, Flor and Hermann (2004) primarily address what they term a “psychobiological perspective”. Yet studies of the interactions between pain behaviour and the environment were fundamental to the pioneering clinical work of the behavioural psychologist Fordyce (1976), and have been explored in studies of the impact of environmental feedback on pain report (Flor et al., 2002), and the impact of broader social contingencies on pain and illness behaviour (Harris et al., 2005). Despite this, the contribution of social-environmental factors seems to receive little attention in reviews of the biopsychosocial model. Similarly, with some notable exceptions (e.g. Keefe et al., 2004b, who evaluated the effect of training spouses in assisting patients with osteoarthritic knee pain), these social-environmental aspects have received relatively little attention in interventions described as “psychosocial” (Karjalainen et al., 2001; Jellema et al., 2005).

3. Why has the psychosocial relationship been overlooked?

One explanation lies in definitions. Even within disciplines that traditionally research the relationship between social factors and health, psychosocial factors have been poorly or variously defined. Martikainen et al. (2002) took as their starting point the Oxford English Dictionary definition of psychosocial as “pertaining to the influence of social factors on an individual’s mind or behaviour, and to the interrelation of behavioural and social factors” (page 1091). Perhaps the most important words in that definition are influence and interrelation, as they should motivate inquiry into how social factors influence individual behaviour.

When researchers focus on the individual level, contextual parameters (e.g. social environment) tend to slip into the background, acknowledged but not addressed. This problem has been highlighted recently for psychosocial factors in musculoskeletal pain and work disability, with recommendations that workplace or system-related psychosocial factors should be assessed with appropriate measures, and included as intervention targets where indicated (Sullivan et al., 2005). Similar neglect of psychosocial factors can also happen in the clinical setting, where the immediate demands created by individual-level factors usually dominate. In this context, there is arguably more collective expertise and familiarity in using individual-level risk factor measures and interventions than population-level ones (e.g., those involving the workplace or the general population). These considerations mean we need a clearer specification of the biopsychosocial model of pain and how it might operate at different levels.

4. Levels of influence

Clearly, social factors can operate at different levels of organisation: the macro-level (e.g. socioeconomic and legal structures), the meso-level (e.g. workplaces, schools), and the micro-level (e.g. personal relationships, access to informal help) (Martikainen et al., 2002). There are studies describing some of the relationships between macro-level social factors and pain – e.g., the inverse relationship between socioeconomic status and chronic pain prevalence (Elliott et al., 1999). More clinically focussed studies have demonstrated the relationship between micro-level social factors and pain behaviour – e.g., the importance of spouse and family (Martire et al., 2006) and informal social networks (Peat et al., 2004). Perhaps the largest body of published studies (and the most confusion) relates to psychological factors and the meso-level of social organisation, particularly workplace psychosocial factors. Psychosocial factors operating at different levels are included in the hierarchy of risk factor “flags” (notably in the Yellow, Blue and Black Flags) used in the assessment of work-related pain (Main, 2002). Studies such as the meta-analysis by Harris et al. (2005), showing that workers compensation status has a clinically important, consistent association with surgery outcomes irrespective of the operation type or where it was performed, suggest that workplace group-level factors are an important influence on health.

5. Implications

What are the practical implications of this? We have a broad model, but it may be too broad and in need of better articulation and conceptual development. A causal theory should lead to testable hypotheses. It should pay explicit attention to defining and explaining the sequence of causal factors that result in an outcome, and explaining how multiple causes act jointly to cause disease. This theory should also define the levels at which risk factors operate, and collect data relevant to
that level. For example, a study of factors determining underage alcohol use might include variables on individual-level attributes (attitude to under age drinking, ability to resist peer-group pressure), attributes of their families (parental views on under age drinking), as well as attributes of the community (number of alcohol outlets, price of alcohol, legislation regarding under age drinking). The time dimension is also critical – identifying which factors are important for initiation, clinical onset, continuation and consequences of disease (Von Korff et al., 1992). Some risk factors might be specific for transition between stages. Another aspect of time matters – risk factor patterns can and do change over time. For example, in the Whitehall Study of British civil servants, the relationship between psychological distress and occupational grade changed from a significant positive gradient to a significant inverse gradient over eleven years (Ferrie et al., 2002).

With an improved conceptual model, specific hypotheses can be generated and then the elements of appropriate analytical methods are easier to identify. Current analytic methods rarely evaluate connections between individuals, or patterns of exposure to risk factors at a group level. This is particularly a problem when social factors operate at a group level. Some of the concepts developed in infectious disease epidemiology are helpful here, as they focus on the importance of patterns of connections between individuals. This is a necessary consideration for factors like social support, and the transmission of behavioural norms and knowledge (Koopman and Lynch, 1999). While the agenda is large, effective approaches will be those where broad questions are sensibly broken down into sub-questions that can be researched separately and incrementally (Mackenbach, 1998).

The second practical implication is that we must pay more attention to how we measure psychosocial risk factors. It is important to remember that much of our current knowledge is based on methods using the individual as the unit of analysis, assuming that outcomes in one individual are independent of outcomes in others. These are unsuitable for analysing data where more than one level of analysis is needed (when individual-level and group-level risk factors operate together). If these group-level factors are omitted the models used to generate risk estimates will be mis-specified (Sheppard, 2003). Important risk factors will go undetected, while estimates of individual-level risk factors may be distorted (Mackenbach, 1998). Some behavioural variables that are measured at an individual level may need to be measured at a group level as well (e.g. work and social relationships). This has implications for levels at which interventions are targeted. For example, in evaluating the stress level of a job there can be a workplace measure of this: the average level of stress reported by all people who do that job (a group-level variable). There can also be an individual worker’s assessment of job stress. The difference between the individual and the group assessment is the individual-specific assessment of job stress and, by definition, it is uncorrelated with the group measure.

Like all models, the biopsychosocial model of pain should be expected to provide a range of hypotheses for clinicians and researchers to test. This suggests that in instances where many factors are operating it is unlikely that any monotherapy (e.g. medication, radiofrequency lesioning, relaxation training), or repeated trials of different monotherapies, would achieve meaningful results. From a broader perspective, it might help patients, their doctor, family and employer if their communities had a reasonable understanding of pain and its best management as this could help to set appropriate expectations for treatment and outcomes. There is some evidence for the effectiveness of interventions at different levels, but they often seem to be conducted separately rather than together. What would be the effect of combining a community-level public education campaign to change community attitudes to managing work-related back pain with individual-level interventions that teach injured workers with low back pain more effective pain self-management strategies? While theoretical models can seem overly academic at times, due attention to them can have important implications for research and clinical practice. While a more explicit model does not guarantee better outcomes are achieved, it provides a better theoretical underpinning for designing interventions where potentially modifiable risk factors are identified.

To conclude, more attention is needed in clarifying what it is we are trying to treat when psychosocial factors are involved, what the psychosocial intervention should include, which level(s) interventions should target, and in predicting the results we can realistically expect. To achieve this we require clearer aetiological models to test, and agreement and consistency about what we measure when we consider “psychosocial” factors. We also need to critically evaluate available instruments that measure these factors at different levels; new instruments may be needed to do this. The net result should be greater clarity about the pathways involved in developing chronic and disabling pain, and better formulated interventions, used in a coherent fashion in a workplace, community or country.

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