

Back pain; what is happening when it becomes chronic?

Field J DC PgD FCC

Introduction

Definition of Chronicity: Characterised by long duration. The state of being chronic [1]. Chronic low back pain (CLBP) is frequently taken as pain lasting longer than six weeks [2].

In the absence of frank tissue damage and other than where nerve injury is involved, it has proven difficult to subdivide much low back pain (LBP). This has given rise to the heterogeneous diagnostic grouping often referred to as non-specific lower back pain. Research has shown that where this pain is of shorter duration it responds more quickly and to different treatments than when it is longer lasting. Thus it is increasingly treated as two different if linked conditions and guidelines for their management drawn up separately.

It is believed that chronic non-specific low back pain (CLBP) is not linked to any persisting tissue damage [2]. Precipitating injuries if any, having repaired naturally within the first six weeks of the pains onset.

The processes involved in CLBP can be viewed as being mechanical, neurological, psychological, social and genetic.

Mechanical

Motor control is a dynamic strategy that refers to the generation and co-ordination of an appropriate sequence of movements. It is a controlling system that determines the requirements for movement and stability and produces appropriate strategies to move the trunk and limbs in a balanced, efficient and co-ordinated fashion. In many with CLBP there is good evidence of altered motor control and some anatomic changes such as wasting of the multifidus muscle [3].

Functional changes that appear common across many people with longstanding LBP include:

- Loss of normal 'variability' of gait, with the motion being less smooth, shorter steps, wider feet placement and reduced arm swinging. This is thought to be because the higher centres are taking over responsibility for some normally 'automatic' functions from the cerebellum, possibly through fear of pain or as a consequence of a reweighing of sensory input due to a deterioration of its reliability [4]. This is important because a reduction in the number of available motor patterns makes the system much less adaptable should unexpected stresses be encountered, such as slipping, sudden loading or rapid changes of direction [5]. Lamoth showed that induced pain, and fear of pain in healthy subjects has effects on erector spinae EMG activity during walking, but that this spared other muscles, leaving global trunk kinematics unaffected [6]. The suggestion being that the altered gait observed in CLBP patients is probably an evolved consequence of a lasting pain related to cognition, rather than a simple immediate effect.

Likely to be related to this loss of variability in gait, are the findings Bruno et al have reported into the firing patterns of muscles (erector spinae, gluteus maximus and hamstrings) during a prone hip extension test. They showed that whilst there was no fixed pattern that could differentiate LBP and non-LBP subjects, LBP patients have greater consistency of the pattern compared to non-LBP subjects [7].

- Poor postural control, with a changed strategy involving increasing use of large muscles at the expense of the inter-segmental ones. In CLBP patients these longer muscles show delayed onset but with increases in intensity and duration of contraction [3, 8, 9]. This is probably due to a combination of an imprecise internal estimate of body position due to reduced accuracy in the sensory integration process, and a perception of threat to the back causing tighter evaluative control over the postural strategy [4, 10, 11].
- Deficits in proprioception including a reduced ability to reposition joints to a target position (joint position sense) and reduced ability to detect movement (kinesthesia) [12, 13].
- Reduced activity of the multifidus muscle with resultant atrophy of the muscles close to the site of pain. This is because of changes in corticospinal nerve signalling rather than local factors such as tissue damage, or reflex arcs within the spinal chord [14]. In patients presenting with a unilateral pain distribution the pattern of atrophy is specific to both vertebral level and side [15].

- Generalised disturbance of higher level information processing in the motor control systems. This causes alterations in co-ordination of motions across the whole musculo-skeletal system. An example being decreased 'preparatory and triggered' reactions in the biceps brachii muscles prior to the upper limb accepting a load in patients with lower back pain [16].
- Impaired function of joints may occur through local adhesions or changed muscular activity. This may, particularly if spinal motor units are involved, directly contribute to the perpetuation of pain signalling [17, 18].

Psychological & Behavioural

Until the 1980's back pain was considered to be a largely physical and functional problem but a growing body of published work began to illustrate the influence of non-physical factors on the outcome of treatment. This was crystallised in the Volvo award winning paper by Waddell (1987) 'A new clinical model for the treatment of lower back pain' [19]. In this he called for psychological and social factors to be considered when developing the management plan for individuals with LBP.

The importance of psychological factors in back pain is exemplified by work showing that patients who avoid activity because of fear of causing pain or doing more damage (fear-avoidance) are substantially more likely to have ongoing pain a year later than those who do not. Psychological factors have been shown to be up to seven times more predictive of lasting symptoms than physical tests or other factors drawn from case histories [20, 21].

In 1995 Vlaeyen et al drew together the psycho-social evidence in the form of a fear-avoidance model to explain the progression of an acute back problem into a chronic one [22]. This describes how individuals exposed to a painful back problem, most likely in the form of mild tissue trauma, have two options for coping; In the first they are not overly concerned by their pain and continue largely normal activity even if this increases their discomfort temporarily. Because they are able to perform these activities they are reassured and persist whilst the initiating injury heals leading to recovery.

The second route is for the pain to evoke concern and anxiety often associated with catastrophic thoughts of further damage and lasting disability. This cognition, linked to aberrant beliefs about the natural history of back pain, leads to fear of further harm and results in a change in behaviour tending away from perceived threatening activity. Because avoiding activities prevents (in the patients mind) the realisation of the catastrophically imagined outcome, these beliefs and avoidance behaviours are maintained and the underpinning aberrant beliefs reinforced. This results in further reductions of activity. In the model this leads to persisting pain (chronicity), disuse, depression and increasing disability; all enduring even after the initiating injury has settled.

That avoidance behaviour is always learnt is key to understanding the model in a clinically useful way. This learning may be through direct experience (once bitten afterwards shy), vicariously via information received (i.e. being told, reading, hearing stories) or through observation (possibly watching others pain behaviour) [23]. The development of inappropriately negative beliefs and understanding relating to pain and its possible

consequences is called catastrophisation. This is felt to be a precursor to pain related fear; with fear going on to cause avoidance behaviour, reducing activity and resultant disability [24].

Because it is anticipation of pain which triggers the avoidance behaviour, and not the experience of pain its self, individuals do not test activities so challenging the belief that they will be painful. Therefore there is less chance that incorrect assumptions will be corrected and so unhelpful expectations may be maintained.

Latterly adjustments have been suggested to the fear-avoidance model to separate the effects of anxiety from fear and to include the role of functional self efficacy (belief in ones ability to complete a task) [25, 26]. However whether these are academic or will be shown to be clinically useful amendments has not been demonstrated.

As well as affecting activity, beliefs and understanding influence LBP patient's posture and patterns of motion. This is illustrated by Moseleys' work showing the way that LBP subjects carried themselves was unrelated to the duration or amount of pain but rather was directly related to their perception of their problem [11].

Patients perception of pain and their response to it occurs within the framework of their individual lives. Other psychological factors that may or may not predate the pain will play a significant part in their response. This includes emotional state, the presence and severity of anxiety or depression (distress) and any personality disorders [21]. In particular the degree of distress has been negatively correlated with outcomes of LBP treatment. [27 - 29].

Social

Relationships with others provides strong motivation, with profound influences on activities. In LBP these can be positive or can encourage inappropriate activity avoidance either directly, or as a secondary gain [30]. Concern with returning to any group after a period of absence can be frightening, the more so the longer the absence, this is felt to be a partial explanation of the difficulties in getting the long term sick back into the work place [31].

Neurological

In CLBP neurological hypersensitivity to pain impacts on the system at three levels; peripheral sensitisation, central sensitisation and altered pathways within the higher centres.

Peripheral sensitisation

Most peripheral sensitisation is short lasting and related directly to stimulus or the effects of local inflammation. It may endure where a nerve axon is traumatised (nociceptive pain) where ongoing irritation produces enduring sensitisation of the nerve both in its tissue terminal and centrally.

Central sensitisation

Central sensitisation is an increase in the excitability of neurones within the central nervous system, so that normal inputs begin to produce abnormal or exaggerated responses.

Mechanisms for sensitisation within spinal cord include:

- A constant intensity, yet prolonged or repetitive input from nociceptors resulting in an increasing intensity of signalling up the spinal chord to the mid brain. This occurs as wide dynamic range (WDR) second order neurones within the dorsal horn be-

come adaptively more sensitive to nociceptive input [32].

- The receptor field of dorsal horn neurones expand when adjacent WDR neurones become responsive to stimuli (noxious or not) to which they were previously unresponsive. Low-threshold sensory fibres activated by very light touch of the skin, for example, begin to activate WDR neurones in the spinal cord that normally only respond to noxious stimuli. As a result, an input that would usually evoke an innocuous sensation now produces pain.
- Profound stimulation of sensory nerves results in enduring sensitivity to noxious sensation [33]. In his original experiments Wall showed that musculoskeletal afferent stimulation produced significantly longer lasting central excitability than did cutaneous. The increased excitability is typically triggered by a burst of activity in nociceptors (such as that evoked by an injury), which alter the strength of synaptic connections between the nociceptors and the neurones of the spinal cord (so-called activity-dependent synaptic plasticity). This process is thought to be responsible for persisting tenderness after deep tissue trauma or surgery.

Dorsal horn hypersensitivity is damped by interneurons acting to inhibit nociceptive signalling. These are controlled in large part by signals descending from brainstem nuclei (nucleus raphe and reticular formation). There are two mechanisms for this; a tonic inhibition causing general damping, ensuring only persistent nociceptive signals are passed upwards and more profound 'descending noxious inhibitory control' (DNIC). In DNIC collat-

eral's from ascending second order nociceptors act in the brain-stem producing descending inhibition throughout the system, other than at their level of activation. They thus ensure that when nociception arrives at the higher centres it is carrying specific information about the origin of painful stimuli.

The descending pain control mechanisms primarily use opiate based neurotransmitters. With enduring pain states there is an increase in the gene expression of the neuropeptide cholecystinin (CCK) and its receptor protein within the dorsal horn. CCK inhibits the effectiveness of opiates used by descending inhibitory pathways and thus has a role in persisting pain by reducing second order pain inhibition [34].

Altered pathways within the higher centres

The limbic system is the final relay in the nociceptive pathways before the conscious appreciation of pain, it also acts in co-ordinating motor signals sent from the higher centres downwards. It plays a role in the gating of pain, and is responsible for generation of emotion and associated physiologic changes. Importantly through the cingulate cortex it ascribes suffering to pain. From a neuro-anatomic perspective, the 'mind-body interaction' may be considered to largely arise in the limbic system [35].

Hyper-vigilance in the in the limbic system has been described as a partial explanation of chronic pain.

Cognitive behavioural therapy for patients with chronic pain has been shown to reduce electrical activity in the limbic system. Where limbic activity changes are seen they have been accompanied by significant improvements in pain and

psychological functioning (e.g. anxiety, worry) [36].

McDermid and others demonstrated that chronic pain patients have a heightened sensitivity to pain (lower threshold and tolerance) because of increased attention to external stimulation and a preoccupation with pain sensations; states that are mediated through limbic activity [37, 38].

Limbic system hyper-vigilance results in increasing activity within pain processing pathways in the central nervous system. Fibres from the limbic system interact with the descending pain inhibitory pathways originating in the periaqueductal grey and flowing through the brain stem nuclei.

Limbic dysfunction also manifests as an abnormal efferent in-ervation of musculature, both visceral and somatic. The musculature undergoes tonic contraction as a result of limbic efferent stimulation, which may generate a further sensation of pain [39].

Interestingly Zhuo postulates that the formation of enduring synaptic links called long term potentiation (a form of neural memory), within the limbic system, may be a new model for understanding central sensitisation related to chronic pain, as well as pain-related cognitive emotional disorders [40].

Genetic factors

The literature has limited yet growing evidence for the role of inherited factors in increasing the chance of individuals developing severe or enduring LBP [41, 42]. Currently implicated are genetic links that predispose towards the production (or not) of an endogenous muscle relaxant, and a predisposition towards developing major depression when back pain occurs [43, 44]. There is also convincing work on

the role of genetic influences affecting disc degeneration [45].

Discussion.

Chronic low back pain is a very complicated condition involving a wide range of different yet highly linked processes (figure 1).

The sensitisation of the spinal cord to pain is a natural and useful process facilitating the repair of damaged tissues by minimising further stresses upon them. However increased pain signalling from the dorsal horn may persist long after an injury has healed if there is a reduction in the normal damping mechanisms or ongoing nociceptive stimulation. Ascending inhibition of WDR cells through mechanoreceptor afferents action, via Renshaw interneurons, may be subdued in CLBP because of reduced movement [fear-avoidance, motor unit dysfunction, stiff/poorly co-ordinated movement patterns, increased muscular tone or adoption of sick roles] [46]. Descending inhibition may be reduced when there is hyper-vigilance in the limbic system [anxiety, distress or depression] or if CCK is being produced in the dorsal horn.

Most of the processes resulting in nociceptive afferent stimulation of the WDR cells will resolve quickly once external stimulus, local inflammation and tissue healing has occurred, however where there is direct irritation to a nerve axon (neuropathic signalling) continuing sensitisation will occur both centrally and in its tissue terminals. Additionally nociceptive afferent stimulation may continue if muscles are maintained in a state of tension [through limbic hyper-vigilance or altered posture], where tissues are stressed because motions are uncoordinated or inappropriate amounts of muscle contraction used through aberrant motor control strategies [changed central pathways &

attempted cognitive control over movement]. Poorly co-ordinated motions may precipitate the picking up of further micro-trauma as a consequence of not being able to respond quickly enough to unexpected external pressures.

It appears highly likely that the human system can be pre-loaded towards an exaggerated response to LBP. If an individual has been exposed to models of enduring suffering or disability through observing others or 'folk memories', then their beliefs and understanding can set them up towards fear-avoidance and undue anxiety. This and prior experiences with pain and illness will colour their perceptions of the possible consequences of LBP [catastrophisation]. Social roles enabling time to rest or avoid activity may set up negative behaviours, as illustrated by those able to claim sick pay reporting more enduring pain and disability than self employed individuals. Genetic factors are starting to be identified with predispositions towards prolonged muscle spasm and extreme psychological reactions to pain. There is no hard evidence for mechanical predisposition towards chronicity, however delayed trunk muscle reflex response to postural challenges is a pre-existing risk factor for LBP [47]. As alterations in motor control are a feature of chronicity it would appear likely that where this type of change exists in healthy subjects who then go on to develop LBP their problems will be more complicated from day one.

The health care professions can, and in the past have been responsible for encouraging chronicity in LBP. Until the mid 1980's bed rest and pain avoidance was standard advice. Even today many professionals and professional organisation use unqualified statements like the '*Don't forget - pain is a warning sign – do not*

ignore it' seen in some European chiropractic publications. Failed treatment of any type can lead patients to the assumption that their problem is complicated and potentially serious. The language used to discuss a problem may also have unexpectedly negative effects - telling a patient they have '*a little wear and tear*' may be meant to reassure, but for some it can create images of destruction and breakdown leading to increased anxiety and catastrophisation.

Summary

The timing of exactly when a back pain moves from acute to chronic looks less likely to be a simple matter of chronology and more likely to vary between individuals. Most people with an enduring pain will eventually develop coping strategies and often this will tend towards avoiding unpleasant sensations. However for some if they find the initial severity of the pain frightening, possibly because it is so far outside their previous experiences, or because of the unpredictability of the muscular spasms so often part of LBP, then the response may tend toward chronicity very early [activity avoidance, stiff movement, poor co-ordination & hyper-vigilance]. If work or domestic partners react to the pain insisting on avoidance of potentially painful activities [for reasons of kindness or concerns of litigation] this can encourage a sick or dependent role developing. Where this enables a patient to avoid tasks they find unpleasant giving them secondary gain it creates further disincentive towards activity/recovery.

The division of back pain by time alone appears overtly simplistic and is a consequence of inadequate information into how to identify characteristics within individuals likely to lead to enduring pain and disability early in their presentation.

Hopefully this will be corrected in time through further research.

Some people are set up for chronicity before the pain starts, others develop it very soon after the conditions onset, a third and possibly the largest group drift into it after suffering for a month or so because they, possibly with the unwitting connivance of others round them or their health advisors, mismanaged the original problem.

For most people LBP is a short lived inconvenience. Even when severe if the patient can be given appropriate information to disarm or prevent fear and anxiety, and if they can keep their motor systems functioning then the chance of chronicity can be dramatically reduced.

Figure 1. Interrelations of processes involved in maintaining lower back pain. J Field

1. *Wensters New World Medical Dictionary*. 2008: Wiley Publishing inc.
2. *Low Back Pain: The Acute Management of Patients with Chronic (longer than 6 weeks) non-specific low back pain*. NICE guidelines. DRAFT. 2008, NICE.
3. Descarreaux, M., C. Lalonde, and M.C. Normand, *Isometric force parameters and trunk muscle recruitment strategies in a population with low back pain*. J Manipulative Physiol Ther, 2007. **30**(2): p. 91-7.
4. Popa, T., et al., *Adaptive changes in postural strategy selection in chronic low back pain*. Exp Brain Res, 2007. **177**(3): p. 411-8.
5. Lamoth, C.J., et al., *Effects of attention on the control of locomotion in individuals with chronic low back*

- pain*. J Neuroeng Rehabil, 2008. **5**: p. 13.
6. Lamothe, C.J., et al., *Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking*. Clin Biomech (Bristol, Avon), 2004. **19**(6): p. 551-63.
 7. Bruno, P. and J. Bagust, *An investigation into motor pattern differences used during prone hip extension between subjects with and without low back pain*. Clinical Chiropractic, 2007. **10**: p. 68-8-.
 8. Edwards, J., *The importance of postural habits in perpetuating myofascial trigger point pain*. Acupunct Med, 2005. **23**(2): p. 77-82.
 9. Magnusson, M.L., et al., *Motor control learning in chronic low back pain*. Spine, 2008. **33**(16): p. E532-8.
 10. Henry, S.M., et al., *Decreased limits of stability in response to postural perturbations in subjects with low back pain*. Clin Biomech (Bristol, Avon), 2006. **21**(9): p. 881-92.
 11. Moseley, G.L. and P.W. Hodges, *Reduced variability of postural strategy prevents normalization of motor changes induced by back pain: a risk factor for chronic trouble?* Behav Neurosci, 2006. **120**(2): p. 474-6.
 12. Gill, K.P. and M.J. Callaghan, *The measurement of lumbar proprioception in individuals with and without low back pain*. Spine, 1998. **23**(3): p. 371-7.
 13. O'Sullivan, P.B., et al., *Lumbar repositioning deficit in a specific low back pain population*. Spine, 2003. **28**(10): p. 1074-9.
 14. Strutton, P.H., et al., *Corticospinal excitability in patients with chronic low back pain*. J Spinal Disord Tech, 2005. **18**(5): p. 420-4.
 15. Hides, J., et al., *Multifidus size and symmetry among chronic LBP and healthy asymptomatic subject*. Manual Therapy, 2008. **13**(1): p. 43-49.
 16. Leinonen, V., et al., *Low back pain suppresses preparatory and triggered upper-limb activation after sudden upper-limb loading*. Spine, 2007. **32**(5): p. E150-5.
 17. Gerwin, R.D., *A review of myofascial pain and fibromyalgia--factors that promote their persistence*. Acupunct Med, 2005. **23**(3): p. 121-34.
 18. Lantz, ed. *Subluxation: The Vertebral Subluxation Complex*. Foundations of Chiropractic, ed. Gatterman. 1995.
 19. Waddell, G., *1987 Volvo award in clinical sciences. A new clinical model for the treatment of low-back pain*. Spine, 1987. **12**(7): p. 632-44.
 20. Burton, A.K., et al., *Psychosocial predictors of outcome in acute and subchronic low back trouble*. Spine, 1995. **20**(6): p. 722-8.
 21. Pincus, T., et al., *A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain*. Spine, 2002. **27**(5): p. E109-20.
 22. Vlaeyen, J.W., et al., *Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance*. Pain, 1995. **62**(3): p. 363-72.
 23. Vlaeyen, J.W. and S.J. Linton, *Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art*. Pain, 2000. **85**(3): p. 317-32.

24. McCracken, L.M. and R.T. Gross, *Does anxiety affect coping with chronic pain?* Clin J Pain, 1993. **9**(4): p. 253-9.
25. Asmundson, G.J., P.J. Norton, and G.R. Norton, *Beyond pain: the role of fear and avoidance in chronicity.* Clin Psychol Rev, 1999. **19**(1): p. 97-119.
26. Woby, S.R., M. Urmston, and P.J. Watson, *Self-efficacy mediates the relation between pain-related fear and outcome in chronic low back pain patients.* Eur J Pain, 2007. **11**(7): p. 711-8.
27. Waddell, G., et al., *A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability.* Pain, 1993. **52**(2): p. 157-68.
28. Woby, S.R., et al., *Adjustment to chronic low back pain--the relative influence of fear-avoidance beliefs, catastrophizing, and appraisals of control.* Behav Res Ther, 2004. **42**(7): p. 761-74.
29. Leeuw, M., et al., *The fear-avoidance model of musculoskeletal pain: current state of scientific evidence.* J Behav Med, 2007. **30**(1): p. 77-94.
30. Lewandowski, W., et al., *Chronic pain and the family: theory-driven treatment approaches.* Issues Ment Health Nurs, 2007. **28**(9): p. 1019-44.
31. Magnussen, L., et al., *Motivating disability pensioners with back pain to return to work--a randomized controlled trial.* J Rehabil Med, 2007. **39**(1): p. 81-7.
32. Mendell, L.M. and P.D. Wall, *Responses of Single Dorsal Cord Cells to Peripheral Cutaneous Unmyelinated Fibres.* Nature, 1965. **206**: p. 97-9.
33. Wall, P.D., *The painful consequences of peripheral injury.* J Hand Surg [Br], 1984. **9**(1): p. 37-9.
34. Wiesenfeld-Hallin, Z., et al., *Central inhibitory dysfunctions: mechanisms and clinical implications.* Behav Brain Sci, 1997. **20**(3): p. 420-5; discussion 435-513.
35. Jones, M.P., et al., *Brain-gut connections in functional GI disorders: anatomic and physiologic relationships.* Neurogastroenterol Motil, 2006. **18**(2): p. 91-103.
36. Lackner, J.M., et al., *Cognitive therapy for irritable bowel syndrome is associated with reduced limbic activity, GI symptoms, and anxiety.* Behav Res Ther, 2006. **44**(5): p. 621-38.
37. McDermid, A.J., G.B. Rollman, and G.A. McCain, *Generalized hypervigilance in fibromyalgia: evidence of perceptual amplification.* Pain, 1996. **66**(2-3): p. 133-44.
38. Giesecke, T., et al., *Evidence of augmented central pain processing in idiopathic chronic low back pain.* Arthritis Rheum, 2004. **50**(2): p. 613-23.
39. Fenton, B.W., *Limbic associated pelvic pain: a hypothesis to explain the diagnostic relationships and features of patients with chronic pelvic pain.* Med Hypotheses, 2007. **69**(2): p. 282-6.
40. Zhuo, M., *A synaptic model for pain: long-term potentiation in the anterior cingulate cortex.* Mol Cells, 2007. **23**(3): p. 259-71.
41. Leboeuf-Yde, C., *Back pain--individual and genetic factors.* J Electromyogr Kinesiol, 2004. **14**(1): p. 129-33.

42. Manek, N.J. and A.J. MacGregor, *Epidemiology of back disorders: prevalence, risk factors, and prognosis*. *Curr Opin Rheumatol*, 2005. **17**(2): p. 134-40.
43. Mishra, B.K., et al., *Do motor control genes contribute to interindividual variability in decreased movement in patients with pain?* *Mol Pain*, 2007. **3**: p. 20.
44. France, R.D., K.R. Krishnan, and M. Trainor, *Chronic pain and depression. III. Family history study of depression and alcoholism in chronic low back pain patients*. *Pain*, 1986. **24**(2): p. 185-90.
45. Battie, M.C., et al., *Heritability of low back pain and the role of disc degeneration*. *Pain*, 2007. **131**(3): p. 272-80.
46. Waters, A.J. and B.M. Lumb, *Descending control of spinal nociception from the periaqueductal grey distinguishes between neurons with and without C-fibre inputs*. *Pain*, 2008. **134**(1-2): p. 32-40.
47. Cholewicki, J., et al., *Delayed trunk muscle reflex responses increase the risk of low back injuries*. *Spine*, 2005. **30**(23): p. 2614-20.