Back pain research: Skirting around the edges?

Low back pain is the leading cause of disability worldwide. The fact that it is seldom possible to identify a specific nociceptive cause is thought to be a major barrier to its successful management. At the same time, the need to identify verifiable subgroups of patients has long been the greatest priority for research. Attempts to do this have, however, been frustrated by the discovery that the condition, once chronic, seldom has only one pain site, but is more often a collection of problems that contribute to pain and disability and reduce quality of life.

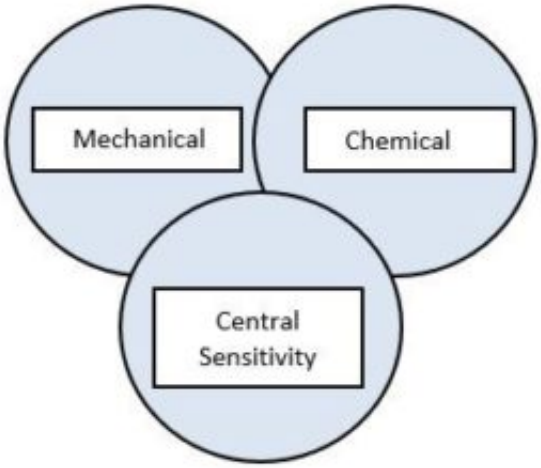

Fig. 1. Sub classification of biological factors in chronic back pain.

Perhaps for this reason, explanatory models of back pain are less common than prognostic, or risk based ones, where attempts at subgrouping have moved more towards natural history and treatment outcomes than mechanisms. This has caused management guidelines to concentrate mainly on attempting to modify the prognostic factors based on natural history and psychosocial profiles. Unfortunately, the effects of these are often small and the evidence weakened by unrecognised confounders, making clinical prediction rules unachievable. Although some progress in slowing the growth of disability has been made by addressing prognostic factors, the overall trend has been upward, and with some alarming features. These stem largely from the fact that the population is ageing and from opiate dependency. In an ageing population, chronic conditions are chronic for longer, and the inactivity caused by prolonged chronic back pain over years is associated with depression, obesity, diabetes and cardiovascular disease, while in some countries opiate addiction is a rising cause of death.

While evidence based practice recognises that biological, psychological and social factors influence the prognosis of an episode of back pain, knowledge of the first of these is the least abundant. Apart from trauma, biological labels such as ‘inflammation’, ‘arthritis’, ‘degeneration’

and ‘instability’ lack clear case definitions. However, some sub classification based on biological factors is becoming more achievable. This breaks the mechanisms of back pain into mechanical, chemical and central sensitisation components (Fig. 1), all of which are becoming accessible to new and innovative methods of objective assessment. Critical to progress however, is the ability by clinicians to focus in terms of the concept that these factors can all be in play to a greater or lesser degree.

The first of these advances has been the emergence of quantitative imaging technologies that have begun to identify mechanical biomarkers for chronic nonspecific back pain, although not yet to explain them. Inflammatory markers have also been identified in subgroups of chronic back patients, (notably TNF-alpha), while neuroimaging has been used to explain the central sensitisation associated with chronic back pain. Together, these represent a more integrated and objective approach to diagnosis that is worthy of research and development.

It is concluded that in order to make greater progress in reducing the burden of chronic back pain disability, a greater research effort that includes objective biological markers and their roles in individuals is needed. The assessment of multifaceted management packages that address those markers that are found to have particular relevance in chronic back pain in individuals could follow and sit alongside the psychosocial assessment. In order for clinicians to be able to implement these, considerable additional work on practitioner awareness and conceptualisation will be needed.

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