The biopsychosocial model – a tool for rheumatologists

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Rheumatologists grapple in daily practice with many controversial syndromes including fibromyalgia, late whiplash syndrome, chronic fatigue syndrome, Gulf War syndrome, the adverse outcomes of silicon breast implants and so on. For decades, much of the debate surrounding, and the approach to these controversial syndromes has centred on a model creating two camps—organic versus non-organic. While each camp has its support, this model seems to have failed in achieving the desired understanding of these syndromes, most particularly in offering the rheumatologist a practical and coherent approach to effective treatment. This chapter will thus introduce the biopsychosocial model, its elements, its advantages over the traditional model and the practical application of this model. Examples will be given of how rheumatologists can approach the treatment of these syndromes through patient education and the implementation of a change in illness behaviour.

Key words: model; fibromyalgia; whiplash; chronic fatigue syndrome; Gulf War syndrome; silicon breast implants; symptom amplification; symptom expectation; symptom attribution.

Remember that all models are wrong; the practical question is how wrong do they have to be to not be useful.1

Rheumatologists often have to evaluate and manage patients with a symptom complex often described as one (or more) of the following syndromes: chronic fatigue syndrome, late whiplash syndrome, fibromyalgia, sick building syndrome, multiple chemical sensitivity, repetitive strain injury, the effects of silicon breast implants and Gulf War syndrome. These illnesses challenge the rheumatologist not only for a diagnosis, but also to provide explanation and treatment advice, often in the face of patients who already have their own diagnostic label and carry with them an armamentarium of literature on their particular illness. This in itself presents a challenge to the physicians’ art, but to add to this, many of these illnesses are characterized by medicolegal and disability issues with which the rheumatologist must also grapple.

To understand these poorly understood illnesses, one may look to models based on illness mechanisms. These may be of value in providing a coherent basis on which to formulate treatment and research avenues, as well as to provide patients with a logical explanation for their illness.

When considering these controversial illnesses the traditional model on which physicians have most often relied is the dichotomous (dualistic) approach (i.e. organic...
versus non-organic). Thus either there is an organic (biological) disease, or if there is ‘nothing wrong’, there must be a psychiatric/psychological (non-organic) problem.

This polarized approach has been unhelpful because (a) it is (being oversimplified) not an accurate description of the events leading to the clinical syndrome, and (b) such dichotomies are further focused by patients, insurance companies, lawyers and disability panels into discussions of the meaning of non-organic being ‘all in your head’ or malingering, and organic meaning ‘all in your body’, with anything psychological being secondary. The polarization thus creates duelling camps, research in each camp being advanced according to the preconceived paradigm.

What may be worse about this model, however, is that society understands these models and their implications all too well. Shorter explains that society has known for well over a century that to have a psychiatric/psychological illness label is to delegitimize oneself and risk being further labelled (depending on which time period one is in) as either being subject to demonic possession or engaging in witchcraft, or being a lunatic, weak and immoral, or simply being lazy and lacking a ‘positive wellness attitude’. Aaron et al demonstrated how differently insurers and disability panels treat patients depending on whether the illness is identified as being organic or non-organic. Aaron et al found that, with a similar presentation, fibromyalgia patients who reported their symptoms following physical trauma were much more likely to receive disability compensation than those reporting their symptoms following emotional trauma. Even with the same symptoms and findings of physical examination, and even the same syndrome label, a different response was given if the onset of the illness was judged as organic as opposed to non-organic.

The most probable basis for this apparent ‘discrimination’ lies in its social context. While the illness is largely a function of the individual’s response to biopsychosocial factors, the acceptance of the sick role is dependent on others’ perception of the basis of that illness. Thus the discriminatory aspect of this dualistic model is entrenched in the psyche of today’s culture and may pervade at an unconscious level. Patients (even those who may wish to explore some of the psychological aspects of their illness) are fearful of being labelled as having an illegitimate illness. Society is not as accepting of psychological or psychiatric disorders, or any disorder with a hint of abnormal psychology at its roots, as a legitimate basis for the sick role and disability. Those who are instead diagnosed – i.e. labelled – as having an organic illness are accorded attention, respect, as well as a disability confirmation much more readily. As a result of their fear of illegitimacy, patients focus on physical symptoms and physical cures to validate their illness to others as well as to themselves. They too begin to develop an inaccurate conception of their illness, and it becomes difficult for the rheumatologist to attempt treatment and education against this barrier.

Thus models, while they are always approximations, are only helpful if they are good approximations. The ‘either organic or non-organic’ approach is simply unhelpful, as it has been for centuries. A considerable amount of effort has, however, been directed towards developing what one would call the biopsychosocial model. In this model, the biological and psychosocial determinants of illness are considered in the assessment. In recent decades, a number of researchers have promoted the need to deal with the medico-legal and social dilemmas of these illnesses in this way.

Yet there is ongoing resistance to such models, perhaps because they are still poorly developed and poorly researched. Some of these attempts still tend to emphasize the cause of the symptoms (i.e. is it inflammation, depression or anxiety?) rather than the cause of the illness behaviour. That is, regardless of why the symptoms arise (their cause), one suggests that in these illnesses, what the patient interprets from those
symptoms, how others (i.e. the therapeutic and legal community) respond to those symptoms and so on are more important determinants of illness behaviour than is the cause of the symptoms themselves. Thus one may have a variety of biological (physical) causes for symptoms, but the behaviour in response to those symptoms, becoming maladaptive and disabling, is determined by psychosocial factors. In some cases, the patient is correct when he or she asserts that the symptoms lie in the body, but one can appreciate that the response to the symptoms, the fear related to the symptoms and the tendency to seek medical attention and litigation in response to those symptoms are not.

**OPERATIVE FACTORS IN THE BIOPSYCHOSOCIAL MODEL**

While one can accept that some aspects of the symptoms that these patients report arise as the somatic component of a depressive or anxiety disorder, it is equally reasonable that many of the symptoms have a physical source. The fact is that many of these same symptoms, with an often unidentifiable cause, occur frequently in normal individuals. That being the case, there is a substrate (symptom pool) immediately available upon which psychosocial factors may act, and this leads to further behaviours that become 'the illness'.

Thus the first aspect of the biopsychosocial model is that there is a general symptom pool. This pool includes headache, neck pain, back pain, numbness, fatigue, dry eyes, dry mouth, dizziness, joint or limb aches and pains, limb stiffness, poor concentration, poor hearing, abdominal pain and bowel irregularity, and sleep disturbance. Many occupations are associated with these symptoms as well, even in healthy workers. Yet the cause of these symptoms, even though at least some would be presumed to have a physical basis in the healthy person, is largely unknown.

Interestingly, when viewing this symptom prevalence in these various syndromes, one notes not only that the symptoms are non-specific, but also that the prevalence pattern (in terms of distribution of symptoms) is very similar to that reported in general population surveys. That patients are drawing from a common symptom pool also explains why there is such a great overlap in symptomatology between the supposedly varied syndromes.

Thus it is not necessary in the biopsychosocial model to state that psychological distress is the cause of all of the symptoms. Many of the symptoms may arise from a multitude of sources (including biological ones), and the more relevant aspect of the psychosocial factors (or psychological distress) is that they act on this substrate.

The first question then, is, how are these symptoms perceived and acted upon differently in patients from healthy people? The second question is, how does this maladaptive behaviour create a new source of symptoms? This brings us to the other factors operative in the biopsychosocial model—symptom expectation, amplification and attribution.

**EXPECTATION, AMPLIFICATION AND ATTRIBUTION–THE WHIPLASH EXAMPLE**

A new biopsychosocial model of the late whiplash syndrome suggests that we need to change our approach to chronic pain, and this probably applies also to many of the controversial illnesses listed above. This model is built on the assumption that most
patients are genuine and have a variety of physical sources for their pain, but that there is probably no chronic injury arising from the acute neck sprain as the source of chronic pain. The model also considers phenomena such as symptom expectation, amplification and attribution.

In North America, for example, there is overwhelming information regarding the potential for a chronic pain outcome after whiplash injury, with widespread knowledge of the expected symptoms even among individuals with no personal experience of having had an accident. This expectation will in turn lead the individual to become hypervigilant for symptoms, to register normal bodily sensations as abnormal, and to react to bodily sensations with affect and cognitions that intensify them and make them more alarming, ominous and disturbing – a process called symptom amplification. It is noteworthy that, in countries such as Lithuania, Germany and Greece, where the late whiplash syndrome is rare, recent studies using the methodology of Aubrey et al. and Mittenberg et al. find a lack of expectation of chronic symptoms, i.e. the whiplash injury is viewed as being benign.

The circumstances of the accident immediately create an impression that the minor injury is not benign. The patient’s fear may start when paramedics take him out of his vehicle in a special stretcher, apply a hard collar and warn him not to move. This may well amplify his symptoms. Symptoms are intensified when they are attributed to a serious disease rather than more benign causes such as lack of sleep, lack of exercise or overwork.

Fear may also be generated later by the responses of physicians after the accident: ‘You had better see a specialist’, ‘You suffered a little nerve damage’, ‘I am not sure what’s wrong with you’, ‘It’s just some arthritis of the spine’, ‘Your X-ray shows degeneration of the spine’. The responses of the legal profession, such as ‘We had better wait for a few years before settling your claim because you never known how badly off you may become’ and ‘As the representative for the insurance company, we ask that you see one of our specialists’, can only serve to increase the patient’s concern.

Another aspect of symptom amplification occurs when others have the accident victim repeatedly draw attention to the symptoms (every time the patient sees a therapist or is asked to keep a diary of symptoms, for example). Attention to a symptom amplifies it, whereas distraction diminishes it. Thus the more frequently patients are asked to rate their pain, the more intensely they rate it.

This symptom expectation and amplification may co-operate to alter an accident victim’s behaviour in a detrimental way. Feeling severe pain and fearing future disability, patients develop the cognitions and behaviours that lead to a withdrawal from activity following minor injury and may develop a maladaptive posture. Yet it is known, for example, that postural abnormality, if induced in healthy subjects, causes pain. Whiplash patients, in response to their heightened pain and anxiety, have just created a new source of pain – and a physical source at that. This new source forms a further part of the substrate upon which symptom amplification can act: patients not realizing that they have a new source of pain, but instead feeling that their damage has progressed, such was their expectation. Psychosocial factors thus ultimately generate, in this example, a physical source of pain.

Another example of this approach includes the use of medication. The patient, experiencing amplified and fearful symptoms, seeks medicine. Yet the medications commonly used for pain have as their adverse effects dizziness, cognitive disturbance, and so on, forming a new physiological source of symptoms that patients will be told (from what they have read or from the input of their therapist) is part and parcel of their injury effects. As a further example, the use of a neck collar and the restriction of neck
range of motion resulting from anxiety will in turn become a cause of dizziness and visual disturbance, again a new physical source of symptoms, there to be amplified, there to be attributed to a ‘chronic injury’, all arising because of the initial behaviour of the accident victims and those in their environment.

The final factor of this triad is thus symptom attribution. As an accident victim becomes hypervigilant for symptoms, and given the common expectation of chronic symptoms, the problem of symptom attribution is a natural result. In the setting of amplification, previously unintrusive symptoms, largely ignored in daily life, become far more intrusive after the accident. The patient regards them as new (i.e. they are now being registered) and attributes them to the accident.

The symptom pool for new symptoms is drawn upon while the acute injury resolves. This pool arises from life’s aches and pains, occupational sources, symptoms from medication use and potential symptoms that arise from maladaptive posture and a change in physical fitness occurring as patients withdraw from normal activity. It is true that one would expect these various benign, physical sources to not be capable of causing severe or significant pain (and they probably did not do so in the past), but that is the effect of symptom amplification – altering the naturally benign appearance of the symptoms. Thus there may be a host of physical problems and sources for symptoms, but these sources would seem benign were it not for the psychosocial setting in which the symptoms were being registered and acted upon. A biopsychosocial model is therefore not a ‘psychogenic model’. It merely suggests that what patients expect, how they perceive their symptoms and how they focus and attribute their symptoms will in turn alter the character of those symptoms and the patients’ behaviour. Following this, entirely new physical problems may arise to contribute to the symptom pool.

MORE EXAMPLES

One can extend this triad of symptom expectation, amplification and attribution to many more of these controversial illnesses. In chronic fatigue syndrome, for example, the initial fatigue from a viral infection may on this occasion, unlike that of a viral infection in the past, be amplified in the setting of various life stressors predating the infection. With this, the symptoms seem more ominous and patients seek medical attention. They are told to rest, given medication and asked to keep track of their symptoms, which only amplifies them further. Sufferers then begin to experience the adverse effects of the medications they are taking, and attribute this to their illness. Eventually, often after a wide range of negative and anxiety-provoking tests, they are labelled as having chronic fatigue syndrome, then attributing a wide variety of symptoms from many sources to their ‘disease’. They may dramatically reduce their physical activity, generating cardiovascular dysfunction, which in turn causes further symptoms.

Patients then become physically unfit and gain weight (back pain following on from this). When attempting any return to normal activity or sport, they feel stiff and sore (as one would expect as a result of their inactivity), but attribute this part of their symptoms to the ‘fibromyalgia component’ of their chronic fatigue syndrome. They expected, from what they have read, that chronic pain could eventually arise as part of the syndrome. Thus psychosocial factors have again led to a behaviour that in turn generates a new physical source of symptoms, which in turn reinforces this behaviour.

As a further example, throughout preparations for the Gulf War and during the war itself, the soldiers and their families were made aware of the very real potential for toxic exposure, the soldiers even taking drugs and other agents to prevent such toxicity.
When the war was over, particularly as there was a relatively low number of casualties, concerns other than survival were on the minds of the veterans and their families. Some soldiers returning from the Persian Gulf would, over several months or years, notice illnesses, as would their families and physicians. Furthermore, out of the 750,000 returning troops, some were bound anyway to develop symptoms or illnesses as that is a normal part of civilian/veteran life. Yet, as with most people, they naturally looked for a cause for the illness. Obviously, given the discussion that had already taken place before the Gulf War about toxic exposure, the tremendous media attention and the current trend in Western society regarding environmental illness, together with an understandable paranoia that the government and the military are both capable of conspiracy and covering up and denying any serious threat in which they may have been involved, the conclusion of the soldiers and their physicians was a natural one – that the Gulf War was to blame. With the expectation of there being a Gulf War syndrome, hypervigilance and thus symptom amplification, followed by the problem of attribution, leads to soldiers reporting chronic, non-specific symptoms that they honestly believe are caused by a toxic exposure.

**BIOPSYCHOSOCIAL TREATMENT**

Extending the whiplash example, the biopsychosocial model leads to an obvious and applicable treatment approach, which for most patients requires the rheumatologist not suddenly to become a psychotherapist but more to be an educator. This model provides new approaches to treatment and truly reaffirms for patients that their pain, for example, is not ‘all in their head’. The treatment is not ‘cognitive or behavioural therapy’, which implies therapy being aimed at ‘psychological disorder’ but physical therapy (e.g. progressive exercises and posture correction/maintenance measures), which prevents a behaviour allowing psychosocial factors to foster chronic pain. Together with a critical re-education component, it is biopsychosocial therapy. Thus the patient has to ‘buy in’ to the new approach.

One is thus telling patients that if they withdraw from normal activity because of neck pain, and fail to mobilize their neck with exercises, they will develop postural abnormality, which will cause more pain. This gives patients the opportunity to recognize that the therapist or physician is judging psychological factors not to be the cause of their pain but to be the cause of their behaviour, behaviour that, if unmodified, will produce physical effects. It legitimizes patients’ beliefs (and they are probably correct) that they have a physical source of chronic pain. But that physical source of pain comes about because of the behaviour chosen following, for example, an acute injury. A further explanation to patients that some of their symptoms arise from collar use and medication is again reaffirming for them a physical source for their symptoms but indicating that their behaviour must ultimately change in order to rid themselves of this source.

From an understanding of the effects of symptom expectation, amplification and attribution, one can immediately consider other forms of sound, practical advice. Patients, should, for example, be encouraged to voice their concerns about what they have heard or known about the outcome of their illness. This then, is an opportunity to explain to patients that while chronic disability can occur, it occurs often because of factors over which the patient can have control, for example, whether they withdraw from activity, take too many medications, avoid exercise, gain weight and so on. Knowing what causes symptom amplification, the physician can discourage a pain/symptom diary, discourage frequent visits to therapists and instead encourage an
independent exercise programme. Furthermore, he or she may discourage litigation as, apart from the inherent stresses involved, litigation often compels patients to focus on symptoms and attend therapists frequently, as well as ‘reliving’ their symptoms during independent medical examination and discovery.

One can manage the problem of attribution by explaining to the patient that pain and fatigue, for example, feel like pain and fatigue no matter what the cause. It is thus easy to mistake the original cause of the fatigue (e.g. a viral infection) for a current cause (e.g. narcotic analgesia and poor physical fitness). Patients with fibromyalgia can be told that their neck and back pain are related in part to poor posture, and that their leg pain is in part related to arch disorder and poor flexibility, each of which has a separate treatment but neither of which is part of one disease process, even though the symptoms seem coincident.

Adding to this approach, one must at some point ask, why has this person developed this behaviour now? People are exposed to viral infection and injury during many phases of their life. What causes them to behave differently on this occasion? One can usually find a series of life stressors predating the illness, or an acutely detrimental event (such as engaging in litigation) that is prompting the behaviour in the first place. Although the patient may be reluctant to pursue these issues, if the physician is first clear with the patient that the symptoms are legitimate, and have many physical sources, the psychological factors may also be accepted as part of the clinical picture, to be dealt with.

**SUMMARY**

The dualistic model of these illnesses are either organic or non-organic is simply too wrong to be useful. What seems more helpful is the biopsychosocial model, which considers an effect of cultural expectation, cultural factors that generate symptom amplification and attribution, and the possibility that physical and psychological causes for symptoms co-exist. This model takes away the stigma of the psychiatric label while at the same time explaining that individuals’ behaviour in response to their infection, injury or exposure may generate much of the illness and that the illness is therefore not an incurable disease.

Life’s anxieties or stresses, which may amplify otherwise benign bodily symptoms or transform minor injuries or exposures into ones that are viewed as serious and generate anxiety, may set in motion the phenomenon of symptom expectation and amplification. These processes eventually lead one to attribute new or even previous symptoms to a ‘disease’ process. This re-attribution then further amplifies the symptoms themselves, since they now take on a different significance and become more intense, noxious and worrisome. The concern that one is seriously ill, together with medical scrutiny and media-induced attention to the latest syndrome, may corroborate one’s fears.

A change in behaviour because of these fears and the influence of others may in turn generate whole new physical problems, generating more symptoms, a self-validating and self-perpetuating cycle of symptom amplification and disease conviction ensuing. At that point, the cause of the symptoms (physical, psychological or both) is less relevant than the illness behaviour. Rheumatologists, if they hope to assist patients in altering that behaviour, need to be compassionate, recognize the validity of the symptoms, recognize that they may have various physical causes and be able to communicate to patients that the various chronic symptoms often arise from the steps one takes in response to the initial problem.
**Practice points**

- it is unhelpful to view these controversial syndromes in terms of organic versus non-organic disorders
- in these patients, psychosocial factors (psychological distress) may cause symptoms, but, more prominently, psychosocial factors act on a substrate of physical sources of symptoms, amplifying them and causing the patient to attribute these sources to a ‘disease’
- symptom expectations derived from the background culture lead one to become hypervigilant, amplifying otherwise benign symptoms that commonly exist in healthy individuals
- the therapeutic and legal communities promote further symptom amplification and misattribution
- patients need to be told that their symptoms may have many physical sources that result in part from how they responded to the original problem, and these new sources must be addressed
- the physician can identify the patient’s symptom (or outcome) expectation at an early stage and help to reduce this expectation by explaining why some patients develop chronic problems whereas others do not

**Research agenda**

- more research is needed in understanding what happens to healthy subjects if they are obligated to focus on life’s benign symptoms (i.e. keep a symptom diary), and more research is needed to identify what other behaviours (including those of the therapeutic and legal communities) lead to symptom amplification
- the variance in outcome resulting from symptom expectation and factors that promote symptom amplification needs to be measured in whiplash patients, cases of fibromyalgia and so on. Identifying ‘high expecters’ and ‘high amplifiers’ early on may lead to more effective intervention
- more research is needed to understand why patients attribute symptoms in the way in which they do so that education can intervene on maladaptive attributions

**REFERENCES**


