# Fibromyalgia
## Mechanisms and management

**The clinical syndrome of fibromyalgia is commonly seen in primary care practice and presents significant challenges in management. A new understanding of the causation of fibromyalgia has led to the development of more effective treatments.**

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Fibromyalgia is a common condition. Using the American College of Rheumatology classification criteria, fibromyalgia occurs in about 2 to 5% of individuals in most Western communities. If less rigid clinical diagnostic criteria are used, the prevalence rises to about 5 to 8%.[1] The condition has a high impact on the individual affected and, as a result, on the society in which he or she lives. Consequently, fibromyalgia is extremely costly, not only in terms of the individual's health and loss of personal life choices but also in regard to social costs, including an inability to work, and the need for expensive safety net provision for individuals with the disorder. Due to its high prevalence and high impact, fibromyalgia presents as a significant and relevant clinical disorder requiring renewed interest and focussed management.

## Controversies

About 20% of the general population have persisting regional or generalised pain. Fibromyalgia (also known as fibromyalgia syndrome) is the most common of the defined disorders that contribute to this social health burden. There have been more than 60 synonyms for this disorder, including the old term fibrositis, which incorrectly implied there was an inflammatory cause for the disorder.

Fibromyalgia is a chronic pain syndrome that is characterised by changes in biochemical functions within the pain system rather than by a structural musculoskeletal abnormality. There has always been controversy with regard to the recognition, nomenclature and diagnosis of this disorder, usually because there is no 'gold standard' diagnostic clinical test.

Many clinicians are also concerned that 'labelling' an individual with a diagnosis of fibromyalgia might in itself worsen the patient's outcome. However, this has been shown not to be the case[2] and in practice an appropriate diagnosis of fibromyalgia is associated with less health care seeking and improved health outcomes.

**IN SUMMARY**

- **The key clinical elements of fibromyalgia are the presence of widespread chronic pain and widespread abnormal tenderness to gentle pressure.**
- **Fibromyalgia is due to abnormal sensitivity within the pain-related nervous system.**
- **Six or more abnormally tender sites, combined with widespread pain, will allow a diagnosis of fibromyalgia to be made.**
- **Fibromyalgia often co-exists with other conditions and it may have similar presenting symptoms to many other disorders.**
- **The basic management platform of education, exercise and psychological strategies must be initiated in all patients with fibromyalgia.**
- **Analgesics, antidepressants and antiepileptics have been shown to be effective for the treatment of fibromyalgia.**

**Table 1. Functional somatic syndromes with similar symptoms to fibromyalgia***

| |
|---|
| Chronic fatigue syndrome |
| Irritable bowel syndrome |
| Irritable bladder syndrome |
| Regional pain syndromes |
| Restless leg syndrome |
| Multiple chemical sensitivities |
| Cognitive dysfunction |
| Dysaesthesia |
| Hypotension |
| Dizziness, nausea |

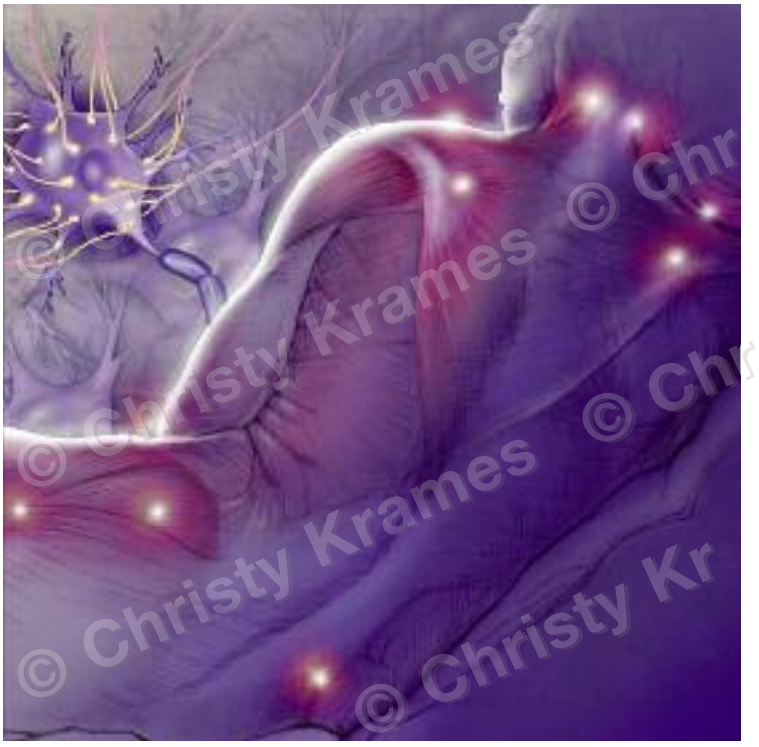
* There is a 20 to 30% overlap between fibromyalgia and other functional somatic syndromes.

A further problem lies in the fact that many patients with fibromyalgia become disabled and are therefore involved in assessment for social safety net provision, such as workers' compensation, disability support pensions or personal litigation. Clinicians who only see patients in this setting are often concerned that the diagnosis labels a functional and potentially reversible problem as being 'organic' and permanent. This results in the individual being locked into long-term disability with the need for significant social support.

Fibromyalgia is caused by abnormal processing of pain mechanisms within the brain and spinal cord. It is influenced by psychological and stress-related factors, so there is often a dichotomy of views about the diagnosis among clinicians. Some consider the problem to be a psychological condition and others as a physical illness. The physical symptoms of the patient with fibromyalgia may be labelled as any number of conditions ranging from 'soft tissue strain' through to 'generalised osteo - arthritis', 'age-related changes' or specified 'abnormalities' of regional musculoskeletal structures. Unnecessary and extensive investigation to attempt to identify an 'organic cause' often occurs.

Although the condition remains controversial and much work needs to be performed to bring the different approaches discussed above together, it is argued that the clinical features of fibromyalgia are characteristic and robust. The diagnosis and management are, in fact, quite logical.

**Fibromyalgia: mechanisms and management**




Fibromyalgia is a common condition caused by abnormal processing of pain mechanisms within the brain and spinal cord. Management of the condition includes education about fibromyalgia, exercise and psychological strategies and appropriate drug therapy to control the pain.

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## Clinical features

The key clinical elements of fibromyalgia are the presence of widespread chronic pain and widespread abnormal tenderness to gentle pressure. These two features indicate that there is dysfunction of the pain-related nervous system. The pain is generally widespread but at times may be more localised and may alternate between different sites. The clinical features fluctuate with time and may be influenced by external factors such as levels of stress or activity, or changes in weather. Patients with a regional pain syndrome have pain and abnormal tenderness within a specific bodily region only, usually an upper or lower quadrant of the body or the low back or low neck.

Other symptoms of sensory dysfunction may occur in patients with fibromyalgia. These include

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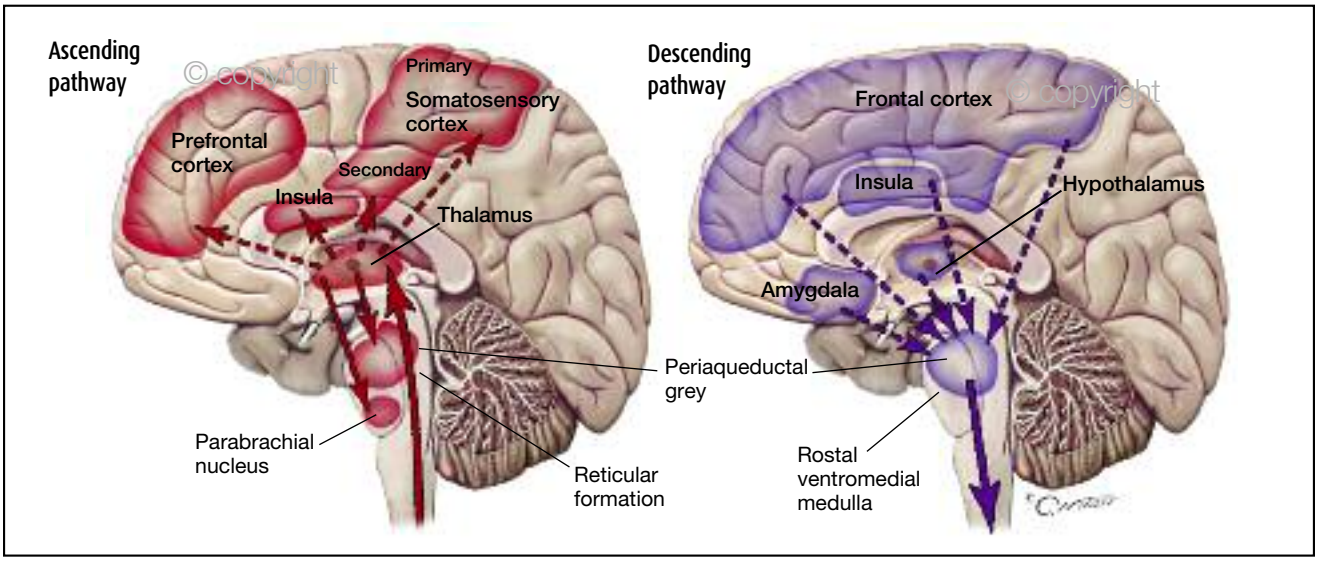





Figure 1. Ascending (left) and descending (right) pathways in normal pain perception. Signals from the dorsal horn of the spinal cord enter the pain neuromatrix (shown in red) to initiate pain processing and response. Descending signals from other centres (shown in blue) send pain modulatory signals to the dorsal horn. Emotional centres are prominent in the descending pathways.

peripheral nonanatomical dysaesthesia, tinnitus, irritability to noise or light, and sensory dysfunction in the bowel or bladder. Hence, many patients with fibromyalgia present with functional somatic disorders involving a range of organ systems to a variety of specialists (Table 1).

## Mechanisms

Fibromyalgia is due to abnormal sensitivity within the pain-related nervous system. Studies of patients with fibromyalgia using various neuroimaging techniques have shown marked changes in the pain-control centres of their brains.[3]

The pain-control regions of the brain connect to the dorsal horn of the spinal cord where normal pain modulation occurs. Altered functions at the dorsal horn are involved in the mechanism of pain in patients with fibromyalgia. The cause for this 'top-down' dysfunction remains unclear but it is particularly associated with emotional and stress factors. Defined pathways from the brain that modulate sensory inputs to the spinal cord involve opiate receptors within the brain and serotonin and noradrenaline actions within the dorsal horn.

The resultant abnormal control of sensory input at the spinal cord results in the neurobiological process of sensitisation. Normal subclinical sensory information

**Table 2. A proposed screening instrument, the Moldofsky Fibromyalgia Score, aimed at identifying patients likely to have fibromyalgia[4]***

**Describe how you have been feeling over the past month**

| | Never | Sometimes | Often | Always | Don't know | Item score |
|---|---|---|---|---|---|---|
| I have pain or stiffness in most parts of my body | 0 | 1 | 2 | 3 | 0 | |
| My body is sensitive to any tightness or pressure | 0 | 1 | 2 | 3 | 0 | |
| I feel energetic | 3 | 2 | 1 | 0 | 0 | |
| My sleep is refreshing | 3 | 2 | 1 | 0 | 0 | |
| I feel sad or nervous | 0 | 1 | 2 | 3 | 0 | |
| I am content with my life | 3 | 2 | 1 | 0 | 0 | |

* Scores less than five are less likely and those greater than 15 are more likely to predict fibromyalgia. Further clinical assessment is always necessary.

from mechanoreceptors in muscles and joints 'accesses' the pain pathways, translating otherwise innocuous regional sensory inputs into regional musculoskeletal pain. This effect is particularly prominent where there is a high input of mechanoreceptor information, particularly from the low neck, low back or areas of the spine that might have been compromised by disease or dysfunction. Thus, pain and tenderness in fibromyalgia is invariably located around the neck, shoulder girdle, low back and buttock areas and regions that refer from these sites. It is important to note that in fibromyalgia there is no tissue damage in the area where the pain is perceived (Figures 1 and 2).

## Recognition in primary practice

In a patient who has a complaint of persisting pain and on examination has abnormal tenderness to gentle pressure, a possible diagnosis of fibromyalgia should be considered. The characteristic features of the disorder can be summarised by a screening questionnaire that focuses on some key elements of the disorder. Patients with high scores are more likely to have fibromyalgia (Table 2).[4]

The emotional distress that is often in the background of a case of fibromyalgia is usually identified by the clinician. The concept of yellow flags is relevant in regard to these emotional factors and is summarised in Table 3.

## Diagnosis

Fibromyalgia is a specific diagnosis. The two key components are the patient's complaint of widespread pain and the clinical finding of widespread tenderness. Tenderness, although subjective, is usually easy to detect by palpation with the thumb or forefinger in areas of the body that are normally more sensitive to touch than other areas. These are known as tender points, and are clustered mainly around the neck, shoulder girdle, chest wall, low back and buttock areas. Palpating the patient's mid-trapezius area, the front of the chest wall

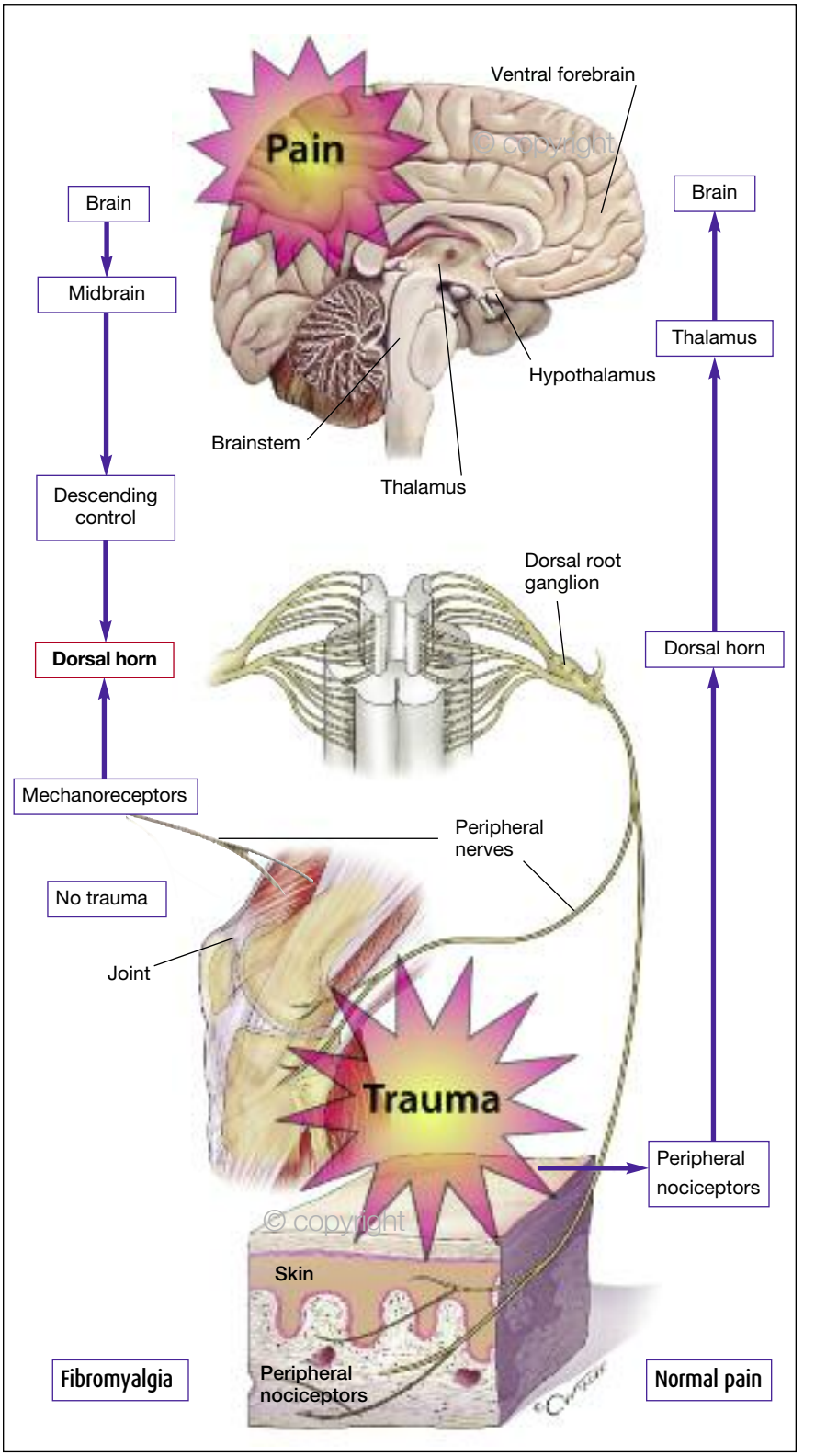



Figure 2. Pathways of pain in fibromyalgia and normal pain. The familiar nociceptive pathway (right) starts at the 'bottom' with stimulation of specific nerves in the periphery leading to pain. Brain-related modulation occurs in the dorsal horn. The fibromyalgia pathway (left) starts at the 'top' allowing for translation of innocuous peripheral mechanoreceptor stimuli to be processed as 'pain' through the overactive sensitised dorsal horn activity (highlighted in red).

**continued**

## Table 3. Yellow flags indicating various psychosocial stressors

| |
|---|
| Positive family history of fibromyalgia |
| Previous pain syndrome |
| Medical condition causing prognostic concern, e.g. systemic lupus erythematosus, rheumatoid arthritis |
| Family, work or personal stressors |
| Sleep disturbance |
| Pain-related work issue |
| Spinal injury |
| Events in the past that may link to a stress reaction |
| Vulnerable personality – poor coping or catastrophising |
| Presence or past history of a mood disorder |
| Previous or current substance abuse |

## Table 4. Medical condition that may mimic fibromyalgia

| |
|---|
| Cancer |
| Metabolic disorders |
| Infection |
| Connective tissue disease |
| Inflammatory arthritis |
| Osteoarthritis |
| Mechanical back pain/disc disease |
| Depression |

and the inner scapular region and noting abnormal tenderness can be used as a quick screening examination.

For research purposes, classification criteria exist for the diagnosis of fibromyalgia and specific sites are designated as being necessary to examine for abnormal tenderness. In clinical practice, however, abnormal tenderness can usually be gauged without performing a specific widespread examination (Figure 3). Generally, six or more abnormally tender sites, combined with widespread pain, will allow a diagnosis of fibromyalgia to be made.

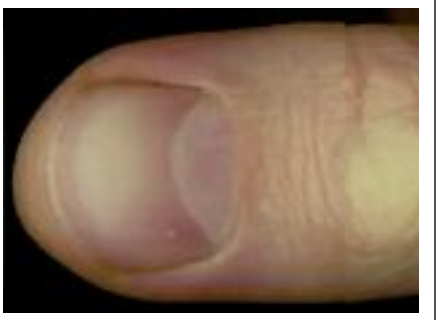


Figure 3. An abnormally low threshold for pain is considered to be present in a patient when pain is elicited at a tender point from pressure that blanches the finger nail.

### Other diagnostic signs

Another diagnostic sign present in patients with fibromyalgia is muscle tightness, often noted as muscle co-contraction (whereby there is inappropriate activation of agonist and antagonist muscles) or give-way weakness. Also common are dermatographia (where light stimulation with the finger nail results in a brisk and exaggerated wheal and flare response) and nonanatomical sensory symptoms (where the patient has symptoms of sensory dysaesthesia, such as localised numbness, 'pins and needles' or burning, but no objective evidence of neuroanatomical sensory abnormality).

### Mimicking conditions

Although the diagnosis of fibromyalgia is usually straightforward the condition often co-exists with other conditions and presenting symptoms may be similar. Examples of conditions that may mimic fibromyalgia are listed in Table 4.

Thus, a positive diagnosis of fibromyalgia must be accompanied by a considered appraisal of other possible co-existent problems. In particular, indicators of possible serious disease or red flags must always be considered (Table 5) and, if present, require further investigation in their own right.

## Investigations

In the average patient with fibromyalgia, it is usually prudent to perform a baseline set of investigations to ensure that subtle general medical conditions are not mimicking fibromyalgia. Although the requirement for each test needs to be considered in the context of the patient's clinical presentation, many mimicking diseases are initially nonspecific and subtle, and relevant investigations may be critical to reaching an early diagnosis (Table 6).

An 'investigation gradient' exists. The average patient does not require much investigation, whereas someone who has an atypical presentation or red flags present might require more significant and wide-ranging investigations.

Many patients with fibromyalgia undergo multiple investigations while seeking other possible causes for their complaints. In this context it is common for test abnormalities to be found, depending on the sensitivity and specificity of the particular test that has been ordered. For example, about 15% of normal patients will have a 'positive' result on an anti-nuclear antibody test. If this positive result is incorrectly connected to the symptoms of fibromyalgia, a misdiagnosis

**Table 5. Red flags suggesting serious pathology**

| |
|---|
| Older age at new symptom onset |
| Weight loss |
| Night pain |
| Focal pain |
| Fevers or sweats |
| Neurological features |
| History of malignancy |

**Table 6. Investigations that may be required in the initial work-up of patients with fibromyalgia**

| |
|---|
| Full blood count |
| Measurement of erythrocyte sedimentation rate |
| Measurement of C-reactive protein level |
| Liver and renal function – including measurements of levels of creatinine, electrolytes, calcium and phosphate |
| Thyroid function |
| Measurement of creatine kinase level |
| Measurement of vitamin D level |
| Presence or absence of rheumatoid factor |
| Presence or absence of antinuclear antibody |

continued

**Table 7. Treatment targets checklist in patients with fibromyalgia**

| Factor | Target/Area of assessment |
|---|---|
| **Does the patient have an appropriate understanding of fibromyalgia?** | |
| Diagnosis | Widespread pain and tenderness |
| Mechanisms | Pain sensitisation, effect of stress |
| Dimensions | Variable severity, duration or level of disability |
| Management principles | Education, exercise, psychology and medications |
| Outcome | Potentially reversible |
| **Is the patient a good self-manager?** | |
| Weight | Ideal |
| Exercise | Regular aerobic, stretching and strengthening |
| Simple relaxation | Time-out and breathing techniques |
| Advanced relaxation | Meditation and yoga |
| Motivation | Willingness to improve and positive attitude |
| Other | Coping strategies and good social support |
| **Have social predicaments been addressed?** | |
| Personal | Relationships, goals and level of distress |
| Economic | Safety-net issues |
| Domestic | Spouse and family |
| Work | Relationships, autonomy and injury |
| Other | Stressors |
| **How does each of the key fibromyalgia domains contribute?** | |
| Pain | Medication review |
| Stiffness | Fitness level and painful muscle trigger points |
| Fatigue | Pacing |
| Sleep | Quality and duration, and whether patients awake refreshed |
| Mood | Secondary or primary changes |
| Global effects | Function |
| **Are there emotional issues?** | |
| Mood disorder | Depression |
| Distress | Anxiety |
| Other | Catastrophising |

of a connective tissue disease may occur.

Similarly, a small percentage of patients with regional pain syndromes may have abnormal nerve conduction of the wrist, despite having normal neurological function. These patients may be incorrectly diagnosed with a peripheral nerve disorder, such as carpal tunnel syndrome or peripheral neuropathy, based on misinterpretation of the characteristics of the test and not on correct clinical assessment of the medical problem. In other words, the test that is applied needs to be carefully interpreted in the context of a patient's symptoms.

### Comorbidities

Characteristic comorbid conditions often occur in patients with fibromyalgia. These conditions are most likely to be related to the underlying mechanisms of fibromyalgia and will need to be treated in their own right.

It is common to see fibromyalgia co-existing with other chronic painful or distressing rheumatic conditions such as the inflammatory joint disease of rheumatoid arthritis (20% of patients) and systemic lupus erythematosus (50% of patients). It is also common to see fibromyalgia in patients who have generalised osteoarthritis or regional degenerative spinal diseases. Patients with other medical illnesses, such as inflammatory bowel disease, are also prone to co-existent fibromyalgia.

Many patients with fibromyalgia have co-existent depression. It has been shown that depression does not cause fibromyalgia but may co-exist in about 25% of patients at the time of presentation with fibromyalgia. Anxiety is also a common comorbid disorder in patients with fibromyalgia.

## Management

### Basic platform

Education, exercise and psychological strategies must be initiated in all patients with fibromyalgia. This basic platform of management cannot be overemphasised even though the thrust of this article is on targeted drug therapies. A checklist is provided to aid assessment of these issues (Table 7). In most patients, adjustment in these areas will be associated with significant improvement.

Education can be facilitated through appropriate website information (e.g. the website sponsored by the US National Fibromyalgia Association and Eli Lilly www.knowfibro.com, or www.pathoutof

continued

**Table 8. Medications to consider in the treatment of patients with fibromyalgia**

| Class | Type | Comment |
|---|---|---|
| Analgesics | Simple | Paracetamol is often not effective |
| | NSAIDs | Their analgesic effect may help some patients |
| | Opioids | No adequate trials to support their use |
| | Mixed | Tramadol with or without paracetamol is beneficial |
| Antidepressants | Tricyclic | Low-dose amitriptyline is beneficial (off-label use) |
| | SSRI | A high dose of fluoxetine may be beneficial (off-label use) |
| | SNRI | A higher dose of venlafaxine may be beneficial. Duloxetine is also effective. (Both used off label) |
| Dopamine agonist | – | Pramipexole may be beneficial (off-label use) |
| Antiepileptic | Alpha-2-delta ligands | Gabapentin and pregabalin are effective (off-label use) |
| Central acting | NMDA action | Possible target, no adequate trials (off-label use) |

pain.com.au) or through contact with Arthritis Australia.

## Simple analgesia

Pain is the dominant symptom of fibromyalgia. Consideration of analgesia is a common starting point in the medication management of the condition (Table 8).

Paracetamol is often trialled at doses of up to 4 g per day. Compliance issues and a lack of response are common. The use of extended-release paracetamol at 665 mg per tablet improves compliance. However, patients seem to prefer NSAIDs to paracetamol, presumably because of their long-acting analgesic effect and not their anti-inflammatory action. No appropriate trials have been performed on these agents in patients with fibromyalgia and caution regarding their effects on other organ systems is always necessary. Anti-inflammatory agents may reduce inflammation in peripheral pain generators (e.g. joints with osteoarthritis or rheumatoid arthritis), thus reducing the aggravating influence of these painful sensory inputs into the pain amplification state of fibromyalgia.

### Opioids

In general, pure opioid medications such as oxycodone or morphine preparations are not clinically useful in the sensitised pain system that characterises fibromyalgia. There are no available trials of pure opioid medication use in fibromyalgia to guide the clinician.

Recent studies suggest that opioid receptors in the brains of patients with fibromyalgia are maximally activated.[5] This indicates that the endogenous opioid system is working well in patients with fibromyalgia and that exogenous opioids may not further improve the central effects of this agent on the fibromyalgia mechanism. Some opioids, such as buprenorphine, may act on the fibromyalgia sensitisation mechanism but clinical trials are lacking.

### Tramadol

The unique opioid tramadol combines a mu-opioid agonist with a serotonin noradrenaline reuptake inhibitory action on the dorsal horn. This agent has been shown to be effective in pain control and improves function in patients with fibromyalgia at a dose of 150 to 300 mg per day. Tramadol often causes adverse events such as nausea or constipation and may interact with other agents, particularly psychoactive drugs such as various antidepressants.

## Pain system modulators

The use of drugs that target the mechanism involved in the sensitisation process of fibromyalgia is more logical and they are now more frequently used.

Tricyclic antidepressants (TCAs) and serotonin noradrenaline reuptake inhibitors (SNRIs) both increase the levels of noradrenaline and serotonin in the dorsal horn through reuptake inhibition. SNRIs are more selective and have preferential effects on noradrenaline rather than serotonin and have better effects on the fibromyalgia mechanism. However, the TCA amitriptyline, which provides a relatively balanced reuptake inhibition of these monoamines, has been shown in a number of meta-analysis to be effective in patients with fibromyalgia.[6]

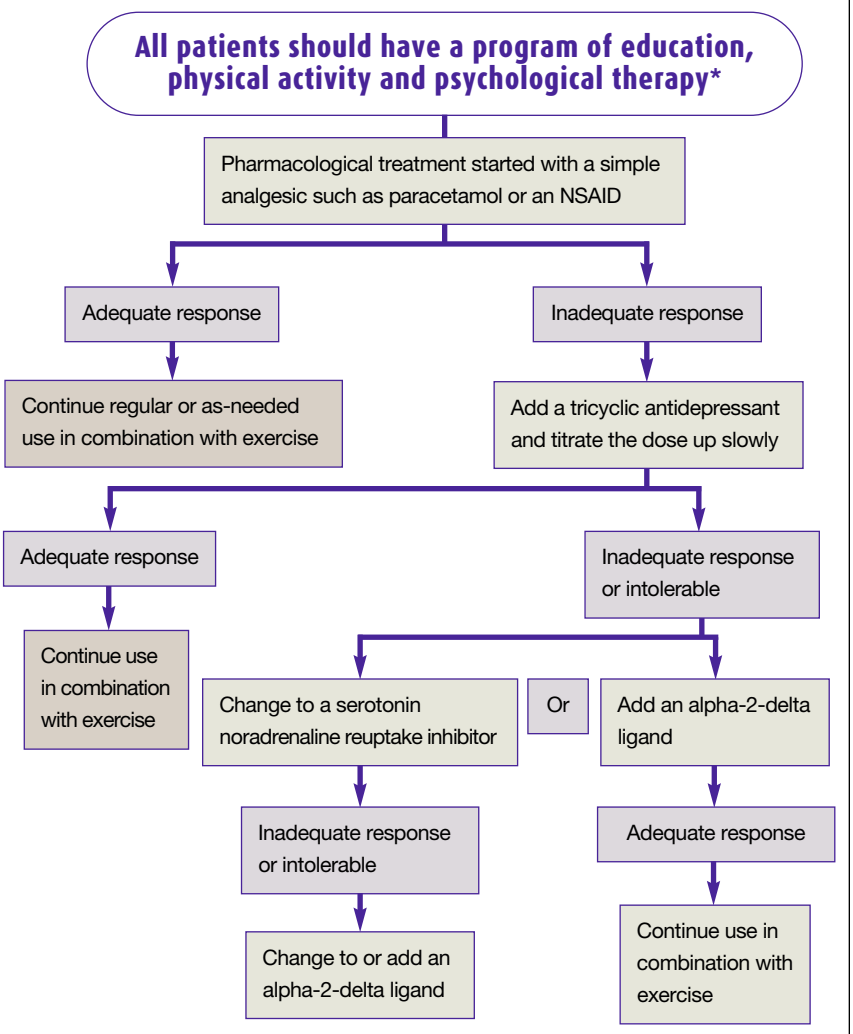
Amitriptyline is currently the most commonly used drug of these classes (off-label use for fibromyalgia). As is the case with most medications used in patients with fibromyalgia, amitriptyline should be given at a low dose and the dose only slowly increased. The medication is used for its pain modulation effect rather than its antidepressant effect, and hence the initial dose is usually about 10 to 20 mg in the mid evening, several hours before bedtime. The dose may be increased over several weeks to 30 to 50 mg in some patients but, in general, lower doses are better tolerated and are as effective. About 40% of patients will respond well to amitriptyline. Side effects can include morning drowsiness and dry mouth and maximal effect might take two to four weeks to be achieved at any one dose.

Drugs in the SNRI class include duloxetine and milnacipran (not available in Australia), which have both been approved by the US Food and Drug Administration for the treatment of fibromyalgia. Their effect on fibromyalgia is independent from any effect on mood. Duloxetine (used off label for fibromyalgia in Australia, but indicated for use in patients with diabetic neuropathic pain) may be effective and better tolerated at a lower dosage of 30 mg per day but many patients require 60 mg per day. Typical side effects include nausea, dry mouth and constipation. Another SNRI, venlafaxine, has benefits in fibromyalgia when used at higher dosages (off-label use), usually more than 150 mg per day.

In contrast, selective serotonin reuptake inhibitors (SSRIs) have much less effect on fibromyalgia (used off label). For example, SSRIs that are highly selective for serotonin, such as citalopram, have little benefit on fibromyalgia symptoms while others, such as fluoxetine, may be beneficial at higher doses where additional noradrenaline effects are seen.

Patients with fibromyalgia who have comorbid depression that has responded well to a SSRI can have low-dose amitriptyline added at a low night-time dose in order to gain extra benefit for the fibromyalgia symptoms. Caution is needed when combining high doses of drugs that act on serotonin because this may increase the risk of serotonin syndrome.

**A possible approach to the management of fibromyalgia**




* If sleep disturbance is particularly prominent, consider the use of a specific sleep agent. If restless leg syndrome is particularly prominent, consider the use of a dopamine-receptor agonist. Psychiatric disorders must be treated fully on their merits by an appropriate professional.

### Alpha-2-delta ligands

Drugs that bind to the alpha-2-delta subunit of the voltage-gated calcium channels of neurones reduce calcium influx and inhibit release of neurotransmitters such as substance P and glutamate. This decreases neuronal excitability and as a consequence these drugs are used effectively as anti-epileptics. They are also effective in the management of patients with chronic pain such as those with postherpetic neuralgia or diabetic neuropathy.

Pregabalin is a gamma-aminobutyric acid (GABA) analogue although it has no effect on GABA mechanisms. It has been shown in clinical trials to be effective for the pain, sleep disturbance and fatigue of fibromyalgia,[7] although its use is off label. The most common side effects include dizziness, drowsiness, headache, dry mouth and, in a smaller percentage of patients, weight gain. Patients who respond to this medication can maintain sustained benefit over time.

Pregabalin also has benefits for sleep and in patients with fibromyalgia it is best to start with a low dose at night. Common practice is to begin with a low dose of 25 to 75 mg just after the evening meal in order to encourage slow absorption and to limit dizziness and other side effects. The dose can be increased on a weekly basis to a maximum dose of about 450 mg per day or until maximum benefit is achieved. Although clinical trials indicate the most effective dosages may be about 300 to 450 mg per day,[7,8] many patients require a lower dose which is better

tolerated. Also, the lower dose is financially better for the patient.

Gabapentin targets the same mechanism as pregabalin and is also effective in fibromyalgia although it is prone to cause more side effects and be less well tolerated. Gabapentin is often required in higher doses, up to 1800 mg per day, and intolerance often limits its application in patients with fibromyalgia. Initial doses should be low dose, about 100 to 300 mg at night, with titration occurring one week at a time.

### Dopamine agonist

The dopamine agonist pramipexole, which is approved for use in patients with restless leg syndrome and Parkinson's disease, may also be useful in patients with fibromyalgia (off-label use). A dose of 125 mg two to three hours before bedtime, with increasing weekly doses of 125 mg to a dose of 750 mg, as needed, is often used. Nausea and dizziness and, rarely, compulsive gambling among other side effects may occur.

The currently available drugs are summarised in Table 8.

### Drug combinations

Many of the above mentioned drugs can be used together, particularly if used at a low dose.

It is usual to commence with a simple analgesic and to then consider tramadol or an anti-inflammatory drug depending on the circumstances. A low-dose TCA is particularly useful in the evening and can be added to the above treatment. If there is depression occurring independently, a SSRI can be combined as a morning dose with the evening dose of amitriptyline. Alternatively, the SNRI duloxetine, which has effects on both depression and fibromyalgia, can be used as monotherapy at a standard dose instead of amitriptyline. Pregabalin can be added to any of these agents because there is no cross reactivity and the drug is well metabolised unless there is severe renal impairment.

A suggested algorithm is shown in the flowchart on page 25.

## Treatment response and prognosis

Fibromyalgia is a multicomponent disorder and patients may receive benefits from one of a number of areas. Pain control is a key area and, if achieved, will allow the patient to initiate or further improve on their aerobic fitness and stretching programs. Relief of pain also decreases stress, improves sleep and often reverses some of the other components of the vicious cycle that is frequently associated with fibromyalgia.

Some drugs are more effective for certain components of fibromyalgia such as sleep disturbance, fatigue, muscle stiffness or global function. Even if full control of all symptoms can not be achieved with medication, the goal of return to normal life activities, such as routine recreational, household and work activities, can often be met despite variable symptoms still being present.

Prognosis is improving with this targeted approach. This is particularly so in the typical community patient with mild to moderate symptoms and when a modifiable trigger is present. Where there are intractable psychosocial stressors the outcome, although better than before, is still guarded.

## Future strategies

### New drugs

Many drugs are being evaluated for use in treating various components of fibromyalgia. These include drugs effective for the sleep disturbance of fibromyalgia. The drug sodium oxybate has benefits in this regard but is unavailable in Australia due to drug regulation issues. Drugs active on the alpha-receptors, such as propranolol 10 to 40 mg per day, have been used off-label where sympathetic nervous system activation is prominent – for example, where anxiety and related symptoms are prominent.

A number of drugs targeting various aspects of the pain-related nervous system are being developed for use in fibromyalgia. Clinical trials of efficacy and tolerance will dictate their clinical role in fibromyalgia.

### Experimental treatments

Treatments that modulate the pain-control centres in the brain may be beneficial in fibromyalgia. These include transcranial magnetic stimulation devices. These approaches are considered experimental at this time.

## Conclusion

Fibromyalgia is common and has high impact on individual and social health. Better understanding of symptom mechanisms is associated with a number of more effective therapies.

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## References

*A list of references is available on request to the editorial office.*

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