

Cognitive-behavioral and operant-behavioral therapy for people with fibromyalgia

K. Thieme¹, D.C. Turk²

¹Department of Medical Psychology, Philipps-University of Marburg;

²Department of Anesthesiology & Pain Medicine, University of Washington

SUMMARY

The current article reviews the cognitive-behavioral (CB) and operant-behavioral perspectives on chronic pain and suggests an answer to the question why changes in behaviors, attitudes, and emotions are associated with decreases in pain severity and impact discussing potential psychobiological mechanisms that may underlie cognitive and behavioral techniques. The impact of learning such as classical and operant conditioning in behaviors and physical responses including baroreflex sensitivity (BRS), as well as the influence of cognitions on pain perception and impact will be presented to explain general efficacy of cognitive-behavior therapy (CBT) and operant-behavioral therapy (OBT) in the treatment of people with fibromyalgia (FM) describing some of the limitations of published outcome studies. We discuss advances in moderation and mediation of treatment outcomes. Lastly, we will discuss the need for research that takes into account evidence-based medicine, methods that address treatment responders and non-responders, individual trajectories, how we might advance and refine CBT and OBT, and strategies related to relapse prevention, maintenance, and adherence-enhancement taking advantage of evolving, technological methods of service delivery. We provide recommendations of how to move forward in approaching studies of CBT and OBT efficacy as a function of better understanding of patient characteristics and contextual factors. We advocate for the potential of the CB perspective and principle of learning for all health care providers regardless of discipline or training and will give examples for making more effective the patient-rheumatologist-relationship by using the principles discussed.

Key words: Cognitive-behavioral therapy, operant-behavioral therapy, psychobiological mechanisms.

Reumatismo, 2012; 64 (4): 275-285

■ INTRODUCTION

The notion that pain is psychosocial as well as physiological is not a new with perhaps the first systematic discussion dating back almost 50 years (1). Both historical and recent models of the experience of pain assert that the experience of pain is maintained by an interdependent set of psychosocial, behavioral, as well as biomedical factors (2, 3). This multidimensional view of chronic pain forms the basis for the use of the cognitive-behavioral (CB) and operant-behavioral (OB) perspectives. It is important to distinguish between the underlying rationales and principles of these two perspectives and various psychological techniques that are based on psychosocial and behavioral principles. Following a description of the central assumptions of

the CB and OB perspectives, we will provide a brief overview of psychobiological mechanisms useful for selecting the relevant cognitive and behavioral techniques that are used to help individuals manage their pain and associated symptoms, and summarize evidence for the efficacy of cognitive-behavior therapy (CBT) and operant behavioral therapy (OBT) approaches that make use of different combinations of these techniques. We then discuss how these perspectives can be used in the collaboration among rheumatologist, rehabilitation professionals, and behavioral health providers to enhance adherence, generalization, and maintenance of treatment benefits. Specifically, the following questions will be addressed:

1. How is pain perception influenced by learning (*i.e.*, classical and operant conditioning)?

Corresponding author:

Dr. Kati Thieme

Department of Medical Psychology

Philipps-University of Marburg

Karl-von-Frisch-Str. 5

35032 Marburg, Germany

2. What are the central assumptions of the CB and OB perspectives?
3. What cognitive and behavioral techniques are used in within CBT and OBT, and how effective are they?

Psychobiological mechanism

Classical (responding) conditioning is widely known as Pavlovian learning from the research of the Russian physiologist. In his classic experiment, Pavlov (1927) found that a dog could be taught, or *conditioned*, to salivate at the sound of a bell by *pairing* the sound with food presented to a hungry dog. Salivation of dogs to food is a natural response; however, by preceding the feeding with the sound of a bell, Pavlov's dogs *learned* to associate the bell with an imminent feeding. Once this association was learned, or *conditioned* the dogs were found to salivate at the mere sound of bell *even in the absence of the food*. That is, the dogs were *conditioned* to anticipate food at the sound of a bell.

The influence of classical conditioning can also be observed across medical diseases. For example, a patient who received treatment that intensified symptoms may become conditioned to experience a negative emotional response to the presence of the health care provider and to any contextual cues associated with the nociceptive stimulus. The negative emotional reaction may lead to tensing of muscles and this in turn may exacerbate symptoms and thereby strengthen the association between the presence of a physical therapist and pain. For example, patients with fibromyalgia (FM) may experience and exacerbation of their pain when they engage in physical therapy (PT).

The pairing of increased pain and exercise may become conditioned, like Pavlov's dogs. Thus, a patient may begin to perceive an increase in his or her pain when they enter the PT clinic even *before* they initiate activity. The facility and the PT may become conditioned stimuli accompanied by increased pain even before the physical exercises that might increase pain begin. For the FM patients the anticipation of pain following activity may lead them

to avoid more and more activities (*i.e.*, stimulus generalization) even ones that might not lead to increased pain in order to prevent anticipated pain. A consequence is that they may become more physically deconditioned and more activities will become difficult. Avoiding pain is a powerful reinforcer and a behavior that is difficult to extinguish.

Classical conditioning is likely one significant contributor to non-adherence to exercise. Figure 1 illustrates the way in which PTs may evoke a conditioned increase of pain in the FM patients they are treating.

Once symptoms persist, fear of motor activities becomes increasingly conditioned, resulting in avoidance of additional activities. The avoidance of pain and fatigue are powerful rationale for reduction of activity, whereas muscle soreness associated with exercise functions as a justification for further avoidance.

Thus, although it may be useful to reduce movement in the acute stage, limitation of activities can be maintained not only by symptoms but also by anticipatory fear that has been acquired through classical conditioning. This may be an important explanation for the high premature termination rates of FM patients with PT. We have often heard patient protest that although they know they should be more active, "I'll pay

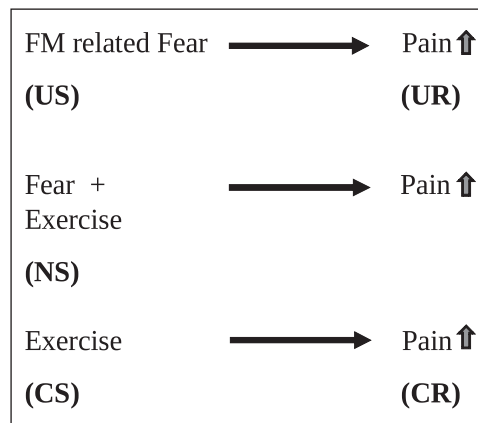


Figure 1 - Classical conditioning of increase of pain. CR, conditioned response; CS, conditioned stimulus; NS, neutral stimulus; UR, unconditioned response; US, unconditioned stimulus.

for it tomorrow.” Here it is the anticipation of aversive consequences that impedes engagement in physical exercise programs.

Anticipatory fear and anxiety also elicit physiological reactivity that may aggravate symptoms. Thus, psychological factors may directly affect nociceptive stimulation and need not be viewed as only reactions to symptoms.

Insofar as activity-avoidance succeeds in preventing symptom aggravation, the conviction that patients should remain inactive will be difficult to modify. By contrast, repeatedly engaging in behavior that produces significantly less pain and fatigue than was predicted (corrective feedback) will be followed by reductions in anticipatory fear and anxiety associated with the activity. Following the behavioral technique of systematic desensitization and exposure therapy, by starting with patient education regarding the relationship of muscle tension - fear - pain, combined with a muscle perception training for a stepwise increase of muscular activity as a quota-based physical therapy program would reduce the fear and progressively increasing their activity levels despite fear of injury and discomfort associated with renewed use of deconditioned muscles (5).

Stress responses and fibromyalgia

Another very important association in respondent learning is observed between stress and pain. Many FM patients report that their symptoms began following physical or emotional stress (6) here is little doubt that living with FM and related symptoms serves as an ongoing stressor. A large proportion of FM patients report stress is an aggravating factor (6). Thus stress, at least as perceived by the patient, may be both a causal and maintaining factor.

To understand stress as a causal factor, it is useful to consider the concept of autonomic stress response (7) that accentuates the individual-specific stress response pattern and shows the organ with the most frequent responses in stress situations has the highest risk to develop a disease. For example, studies have identified demonstrated augmented stress response of FM patients

compared to healthy controls based on plasma cortisol levels despite the comparable baseline level (8,9).

A number of psychophysiological studies in patients with FM (10-12) demonstrate heterogenous autonomic responses on stress. For example, Thieme and Turk (13) identified several subgroups of FM patients based on patterns of stress responses to imaginal stimuli (*e.g.*, perceived social conflicts).

One subgroup of FM patients displayed a high blood pressure (BP) and elevated heart rate (HR) along with stable skin conductance levels (SCL) and reduced electromyographic (EMG) responses to emotional stress, the other large subgroup shows reduced BP, HR SCLs, and EMG responses. Another subgroup showed increased SCLs, BP, and HR and reduced EMG responses in contrast to another small subgroup displayed an elevated EMG response, but stable BP, HR, and SCL responses (13). Similar results of blunted and increased sympathetic reactivity, especially, have been reported in response to physical stressor such as orthostatic examination and cold pressure test suggesting disturbed sympathovagal balance and reduced parasympathetic activation in patients with BP stress response, and reduced sympathetic activation in patients with low BP stress response, especially (12, 14).

Given the high prevalence of anxiety and depression in FM and the basic findings demonstrating the relationship between anxiety and increased BP as well as depression and lower SCL and BP, a classical conditioning of BP by maladaptive cognitions have been proposed (15, 16). An early psychophysiological study with chronic low back pain patients who were asked for thinking on the most terrible pain that they have ever experienced as well as on an intensely stressful situation showed a significant increase of EMG followed by increase of pain perception (17).

Thinking about pain and stress mediated by maladaptive attitudes provoke an autonomic response that tended to persist. The conditioning on maladaptive attitudes resulting in BP stress reactivity and increas-

ing pain perception may be important in the etiopathogenesis in chronic pain. The non-inverse relationship of BP and pain is different to the response in healthy individuals as an important component of pain regulatory system defined by the interaction of pain sensitivity and cardiovascular dynamics that influenced baroreceptor sensitivity (BRS).

In healthy individuals, a functional interaction between the cardiovascular and pain regulatory systems appears as an inverse relationship between baroreceptor stimulation and acute pain sensitivity (18-20). Increased BRS leads to the activation of diffuse noxious inhibitory control (DNIC, also called conditioned pain modulation) and is expressed as an activation of both descending spinal and supraspinal pain inhibitory processes (21).

The dysfunction associated with the absence of an inverse relationship may be related to diminished baroreceptor sensitivity in people with chronic pain (22). Several studies reported that resting arterial blood pressure is not related to pain perception in chronic pain patients (14, 23), these results are consistent with the hypothesis that baroreceptor-mediated modulation of pain is altered in chronic pain patients (24). These findings support the view that painful chronic musculoskeletal pain disorders are associated with alterations in central nervous system inhibitory systems that facilitate the expression of activity in central pain channels and inhibits the expression of activity in central tactile channels.

Behavioral stress reduction techniques, HR-biofeedback, breathing exercises, and cardiovascular training can influence BRS. Future studies will investigate the mechanism-oriented use of these techniques to pain inhibition.

Operant conditioning - Environmental contingencies of reinforcement

The main focus of operant conditioning is modification in frequency of a given behavior by environmental reinforcement. If the consequence of the given behavior is rewarding, the likelihood of its occurrence increases; if the consequence is aversive, the likelihood of its occurrence decreases (Table I).

Behaviors associated with symptoms, such as distorted ambulation, rubbing painful body parts, lying down during the day, are called *pain behaviors* (25). When a person is exposed to a stimulus that causes tissue damage, the immediate response is withdrawal in an attempt to escape from noxious sensations. Such pain behaviors are adaptive and appropriate. Pain behaviors such as avoidance of activity and help seeking may effectively prevent or withdraw aversive consequences (e.g., pain, fatigue). This negative reinforcement makes such behaviors more likely to occur in the future. The operant view proposes that acute pain behaviors such as avoidance of activity to protect a wounded limb from producing additional noxious input may come under the control of external contingencies of reinforcement (responses increase or decrease as a function of their reinforcing consequences: see Table I), and thus may evolve into a chronic problem.

Pain behaviors may also be maintained by the escape from noxious stimulation by the use of drugs or rest, or the avoidance of undesirable activities such as work. In addition, *healthy behaviors* (e.g., activity, working) may not be positively reinforced, and the more rewarding pain behaviors may therefore be maintained (26).

Consider an example to illustrate the role of operant conditioning. When a person with FM's symptoms flare up, she may lie

Table I - Operant schedules of reinforcement.

Schedule	Consequences	Probability of the behavior recurring
Positive Reinforcement	Reward the behavior	More likely
Negative Reinforcement	Prevent or withdraw aversive results	More likely
Punishment	Punish the behavior	Less likely
Neglect	Prevent or withdraw positive results	Less likely

down to rest. Her husband may observe her behavior and infer that she is experiencing intensification of her pain symptoms. He may respond by offering to bring her some medication, to take children out to the part to give her quiet time, or to assume some household chores. Such response may positively reward the woman and her pain behaviors (*i.e.*, lying down) may be repeated even in the absence of symptoms. In other words, the woman's pain behaviors are being maintained by the learned consequences.

Another powerful way she reinforces her pain behaviors is by permitting her to avoid undesirable activities. When observing his wife lying down, the husband may suggest that they cancel the evening plans with his brother, an activity that she may have preferred to avoid anyway. In this situation, her symptom reports and behaviors are rewarded by her husband providing her with extra attention and comfort and the opportunity to avoid an undesirable social obligation.

People with FM may not consciously communicate about their symptoms to obtain attention or to avoid undesirable activities. It is more likely to be the result of a gradual process of the shaping of behavior of which they patient is not aware.

Health care professionals may also reinforce symptoms by their responses (27). The physician who prescribes medication on the patient's complaint may be reinforcing reporting symptoms. That is, patients learn their behavior elicits a response from the physician, and if the response provides some relief, then the patient may learn to report pain in order to obtain the desired outcome. This is the case when pain medication is prescribed on a *take as needed* (PRN) basis. In this case the patient must indicate that the pain has increased in order to take the medication. If the medication provides some reduction of symptoms then, the attention to and self-rating of pain may be maintained by the anticipated outcome of relief. In several studies, the interaction of physicians and patients have been shown to unwittingly reinforce patients' pain reporting by providing further atten-

tion and more intensive treatments based on patients' reports rather than any evidence of physical pathology (28-30).

PTs who suggest that patients engage in exercises until the *pain and fatigue become excessive* are functioning in the same way as the physician. The reinforcement of reduction in activity to reduce pain will come to maintain complaints and inactivity. The alternative for the PT is to prescribe exercises on a work-to-goal rather than work-to-pain basis. Termination of the exercise is then paired with completion of a designated set of exercises, not pain. Here we can see how classical and operant conditioning become related. The pairing of the neutral and pain-evoking stimuli is classically conditioned, and the reinforcement schedule established by the health care professional leads to operant learning.

The combination of reinforced pain behaviors and neglected healthy behaviors is common in FM. The operant learning paradigm does not uncover the etiology of symptoms but focuses primarily on the maintenance of pain behaviors and deficiency in healthy behaviors. Adjustment of reinforcement schedules is proposed as a mechanism to modify the probability of recurrence of pain behaviors and healthy behaviors.

Operant technique focuses on the elimination of symptom behaviors by withdrawal of attention and increasing of healthy behaviors by positive reinforcement. The operant view has generated what has proven to be an effective treatment for select samples of FM patients (31, 32).

Cognitive-Behavioral perspective (C-B)

People learn to predict events based on experiences and information processing. They filter information through their pre-existing knowledge (2), and react accordingly. Their responses, consequently, are based not on objective reality but their idiosyncratic interpretations. As interaction with the environment is not a static process, attention is given to the ongoing reciprocal relationships among physical, cognitive, affective, social, and behavioral factors.

The C-B perspective incorporates princi-

ples of learning within an integrated perspective on the individual experiencing pain and pain management. The C-B model proposes that so-called conditioned reactions are largely self-activated on the basis of learned expectations rather than automatically evoked. The critical factor for the C-B model, therefore, is not that events occur together in time or are operantly reinforced, but that people learn to predict them based on experiences and information processing. They filter information through their preexisting knowledge, and organized representations of knowledge (2), and react accordingly. Their responses, consequently, are based not on objective reality but their idiosyncratic interpretations of reality. As interaction with the environment is not a static process, attention is given to the ongoing reciprocal relationships among physical, cognitive, affective, social, and behavioral factors.

There are five central assumptions that characterize the CB perspective on pain (2) (Table II).

People's beliefs, appraisals, and expectations about pain, their ability to cope, social supports, their disorder, the medicolegal system, the health care and their employers are all important as they may facilitate or disrupt the patient's sense of control. These factors also influence patients' investment in treatment, acceptance of responsibility for self-management, perceptions of dis-

ability, support from significant others, expectancies for treatment, acceptance of treatment rationale, and adherence to treatment.

Cognitive interpretations will also affect how individuals present symptoms to significant others, including health care providers and employers. Overt communication of pain, suffering, and distress will enlist responses that may reinforce pain behaviors and impressions about the seriousness, severity, and uncontrollability of the pain. That is, reports of pain may lead physicians to prescribe more potent medications, order additional diagnostic tests, and, in some cases perform surgery (e.g., 27, 33). Family members may express sympathy, excuse the patient from usual responsibilities, and encourage passivity thereby fostering further physical deconditioning. It should be obvious that the CB perspective integrates the operant conditioning emphasis on external reinforcement and the respondent's view of learned avoidance within the framework of information processing.

People with persistent pain often have negative expectations about their own ability and responsibility to exert any control over their pain, and they avoid activities that they believe will exacerbate their pain or contribute to additional injury (34, 35). Moreover, they often view themselves as helpless. Such negative, maladaptive appraisals about their condition, situation, and their personal efficacy in controlling their pain and problems associated with pain serve to reinforce their over-reaction to nociceptive stimulation, inactivity, and experience of demoralization. These cognitive appraisals are posed as having an effect on behavior; leading to reduced effort, reduced perseverance in the face of difficulty and activities and increased psychological distress.

The specific thoughts and feelings that patients experience prior to exacerbations of pain, during an aggravation or intense episode of pain, and following a pain episode can greatly influence the experience of pain, subsequent pain episodes, and response to treatment (e.g., 36, 37).

Table II - Assumptions of the cognitive-behavioral perspective (Flor and Turk, 2011).

People are active processors of information and not passive reactors.
Thoughts (e.g., appraisals, expectancies and beliefs) can elicit and influence mood, affect physiological processes, have social consequences, and also serve as an impetus for behavior; conversely, mood, physiology, environmental factors and behavior can influence the nature and content of thought processes.
Behavior is reciprocally determined by both the individual and environmental factors.
People can learn more adaptive ways of thinking, feeling and behaving.
People should be active collaborative agents in changing their maladaptive thoughts, feelings and behaviors.

Treatment and their effects

Operant-Behavioral pain therapy (OBT)

Based on general assumptions of the operant pain model, OBT assumes that pain, even though originally a reflex, is maintained through reinforcement controlled by operant conditioning.

At the first step patients learn to understand that the sensory, cognitive, affective, and behavioral components of pain memory have to interact to be able to perceive pain. This recognition let them understand that:

1. fear, sadness, and pain behaviors have to be associated with pain and
2. the inhibition of pain can be influenced by extinction of the conditioned associations.

In the 2nd step, the patients learn to detect pain behaviors such as problematic symptoms and behaviors ranging from mental problems such as anxiety and mood disorders (38), physical problems associated with medication misuse (27), deficient activity levels (39), excessive use of doctor visits (31), avoidance behaviours (40), and amplified pain perception (*e.g.*, 26, 28). At the 3rd step, the patients differ between pain and healthy behaviors in different areas of their life, and to recognize the maintaining factors of pain behavior related to spouses, children, friends and physicians as well as to their own reinforcing cognitions as catastrophizing.

To achieve the aims of treatment, a number of strategies are used, such as the contingent positive reinforcement of pain-incompatible behavior and reduced or absent positive reinforcement of pain behaviors, time-contingent intake and reduction of medication, increased bodily activity, reduction of interference of pain with activities, reduction of pain behaviors, and training in assertive pain-incompatible behaviors.

Active participation of spouses is important as the spouse can learn to reinforce the patient's pain-incompatible behaviors. Physical exercise is an essential part of OBT for training of motor perception, increasing personal physical activities and reducing avoidance behavior, intake of medication, and excessive solicitous spouse behavior (31, 41). OBT reduced pain perception and

pain related interference, pain behavior, medication, improves sleep intensity and increases physical activities. These changes are associated with physical changes such as an increase of BP that may be associated with the inverse relationship of BP and pain (42) and with changed central modulation of pain (43).

Cognitive-Behavioral pain therapy (CBT)

CBT usually contains a variety of therapeutic procedures based on the sensory, cognitive, affective, and behavioural components of pain memory.

The focus of CBT is targeted on changing negative emotions that result from dysfunctional thinking (44). CBT emphasizes the stress-tension-pain-model as an explanation why stress and the unwanted but automatically appearing cognitions such as *catastrophizing* (*Pain is increasing again and becomes worse and worse*) can influence pain perception.

The identification of catastrophizing related to stress situation and to pain is indispensable.

After changing essential stress-related cognitions (*I have so much to do, I don't know how to handle that*) to active cognitions (*I'll pace my activities, gradually built up my endurance*), and distraction as well as relaxation as the most important stress reduction techniques (45), and develop active strategies targeted on well-being and control (*When I remember that I reached that goal several times, why should I fail this time*).

The patient is taught problem-solving to be capable to modify emotions such as anxiety, helplessness, and depression that influence both the cognitive and affective components of pain and ultimately behavior. This approach also provides an experience of the ability to interfere with the perception of pain, which boosts the self-efficacy expectations.

Summarizing, psychological treatments have shown small to medium long-term effects for FM (46). Two meta-analyses reported that CBT is effective in the reduction of depression in FM patients (47,48) but the results have not been completely

Table III - Randomized, controlled treatment studies of cognitive-behavioral and operant- behavioral pain treatment for FM.

Author, year	Number of sessions and treatment hours	Follow-up in month	Measurement of pain intensity	Effect size* of changes in chronic pain	Intervention of the treatment group	Intervention of the Control Group
None effects						
De Voogd, 1993	10 10 h	none	VAS	0.00	Psychomotor therapy and marital counseling (N=50)	Non-treatment group (N=50)
Nicassio, 1997	10 10 h	6	MPQ	0.00	CBT	Educational
Vlaeyen, 1996	12 12 h	12	MPQ	-0.25	Cognitive-educational (N=44)	Waiting list (N=45)
	12 12 h			0.00	Educational (N=44)	
Williams, 2002	4 6 h	12	MPQ	0.00	Standard medical treatment combined with 6 CBT sessions (N=76)	Standard medical treatment with pharmacotherapy and aerobic exercises (N=69)
Without any stable effects						
Redondo, 2004	8 20 h	6	FIQ	0.43 (previous:0.4)	CBT (N=21)	Physical exercises (N=19)
Soares, 2002	10 20 h	6	MPQ	0.07 (previous: 0.3)	Behavioral (N= 18) Educational (N= 18)	Waiting list (N=17)
Wigers, 1996	14 14 h	48	VAS	0.1 (previous: 0.4)	Stress management (N=20) Aerobic exercises (N=20)	Treatment as usual (N=20)
Clinical significant changes of pain intensity						
Bennett, 1996	24 36 h	24	FIQ	0.9	CBT (N=104)	Waiting list (N=29)
Burckhardt, 1994	6 12 h	1.5	FIQ	1.1	- Self-management with education (N=33) - Education with physical exercises (N=33)	Physical training (N=33)
Garcia, 2006	9 18	3	FIQ	1.87	- Pharmacotherapy (cyclobenzaprine) (N=7) - CBT (N=7) - CBT+Pharmaco (N=7)	No treatment (N=7)
Kashikar-Zuck, 2005	8 16	16	VAS	0.81	Coping skill training (N=15)	Self-monitoring (N=15)
Keel, 1998	15 30 h	3	VAS	0.53	CBT (N=14)	Autogenic Training (N=13)
Thieme, 2003	25 75 h	15	MPI	2.14°	Operant Behavioral therapy (N=40)	Amitriptylin and Physiotherapy (Relaxation) (N=21)
Thieme, 2006	12 24 h	12	MPI	1.14	CBT (N=42) Operant Behavioral therapy (N=43)	Social discussion as attention placebo (N=40)
	12 24 h	12	MPI	1.10		

VAS, Visual Analogue Scale; FIQ, Fibromyalgia Impact Questionnaire; MPI, Multidimensional Pain Inventory; MPQ, McGill Pain questionnaire. *The effect sizes for treatment groups were computed based on the formula $CG (MeanT2-4) - CBT/OTG (MeanT2-4)/CBT/OTG (standard deviation)$. °Inpatient setting. Reprinted from Thieme K, Gracely RH. Are psychological treatments effective for fibromyalgia pain? *Curr Rheumatol Rep* 2009; 11: 443-50.

consistent (49). The amount of treatment may be an important factor. Glombiewski (47) showed that higher treatment dose was associated with better outcome following CBT. Recently, Thieme and Gracely (50) (Table III) reported that the studies that included more than 20 treatment hours achieve long-term improvements. In addition, CBT and OBT customized to patient characteristics may enhance the treatment effects (51,52).

The mechanisms by which such individualized treatments produce benefits still have to be examined. It was recently reported that positive responses to CBT are related to higher levels of affective distress, lower coping, less solicitous spouse behavior, and lower pain behaviors; whereas responders to OBT displayed significantly more pain behaviors, greater physical impairment, more physician visits, more solicitous spouse behaviors, and higher reported catastrophizing (51).

■ CONCLUSIONS

We described important behavioral principles that appear to be important in understand persistence of pain and disability in FM and other chronic pain conditions. These principles are relevant to all health care providers and not just behavioral health practitioners. There are a number of cognitive and behavioral techniques, based on these principles that form the basis of CBT and OBT. Additional research is needed to identify the necessary and sufficient components of CBT and OBT, the best methods of intervention, and patient characteristics associated with differential treatment outcomes (51).

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

KT provided the preparation of the manuscript, and DCT was involved in the interpretation of the results, and preparation of the manuscript.

■ REFERENCES

1. Melzack R, Wall PD. Pain mechanisms: A new theory. *Science*. 1965; 19: 971-9.
2. Flor H, Turk DC. Chronic pain: an integrated biobehavioral approach. Seattle, IASP Press. 2011.
3. Turk DC, Swanson KS, Wilson HD. The biopsychosocial model of pain and pain management. In M Ebert, RD Kerns (eds.). Behavioral and pharmacological pain management. New York, Cambridge University Press. 2011; 16-43.
4. Pavlov IP. Conditioned reflexes. London and New York, Oxford University Press. 1927.
5. Vlaeyen JWS, de Jong JR, Sieben JM, Crombez G. Graded exposure in vivo for pain-related fear. In: DC Turk, RJ Gatchel (eds) Psychological approaches to pain management: a practitioner's handbook. New York, The Guilford Press. 2001; 210-33.
6. Bennett RM, Jones J, Turk DC, Russell J, Matallana L. An internet survey of 2,596 people with fibromyalgia. *BMC Musculoskeletal Disord*. 2007; 8: 27.
7. Wenger MA, Clemens TL, Coleman DR, et al. Autonomic response specificity. *Psychosom Med*. 1961; 23: 185-93.
8. Neeck G. Neuroendocrine and hormonal perturbations and relations to the serotonergic system in fibromyalgia patients. *Scand J Rheumatol (Suppl.)* 2000; 113: 8-12.
9. Crofford LJ, Engleberg NC, Demitrack MA. Neurohormonal perturbations in fibromyalgia. *Bailliere's Clinical Rheumatology*. 1996; 10: 365-78.
10. Qiao ZG, Vaeroy H, Morkrid L. Electrodermal and microcirculatory activity in patients with fibromyalgia during baseline, acoustic stimulation and cold pressor tests. *J Rheumatol*. 1991; 18: 1383-9.
11. Martinez-Lavin M, Hermosillo AG, Rosas M, Soto ME. Circadian studies of autonomic nervous balance in patients with fibromyalgia: a heart rate variability analysis. *Arthritis Rheum*. 1998; 41: 1966-71.
12. Bou-Holaigah I, Calkins H, Flynn JA, et al. Provocation of hypotension and pain during upright tilt table testing in adults with fibromyalgia. *Clin Exp Rheumatol*. 1997; 15: 239-46.
13. Thieme K, Turk DC. Heterogeneity of psychophysiological stress responses in fibromyalgia syndrome patients. *Arthritis Res Ther*. 2006; 8: R9.
14. Kelemen J, Lang E, Balint G, et al. Orthostatic sympathetic derangement of baroreflex in patients with fibromyalgia. *J Rheum*. 1998; 25: 823-5.
15. Cohen DH, Randal DC. Classical conditioning of cardiovascular responses. *Ann Rev Physiol*. 1984; 46: 187-97.

16. Reiff S, Katkin RS, Friedman R. Classical conditioning of the human blood pressure response. *Intern J Psychophy*. 1999; 34: 135-45.
17. Flor H, Birbaumer N, Schugens MM, Lutzenberger W. Symptom-specific psychophysiological responses in chronic pain patients. *Psychophysiology*. 1992; 29: 452-60.
18. Dworkin BR, Elbert T, Rau H, et al. Central effects of baroreceptor activation in humans: Attenuation of skeletal reflexes and pain perception. *P Natl Acad Sci USA*. 1994; 91: 6329-33.
19. Fillingim RB, Maixner W, Bunting S, Silva S. Resting blood pressure and thermal pain responses among females: effects on pain unpleasantness but not pain intensity. *Int J Psychophysiol*. 1998; 30: 313-8.
20. Myers CD, Robinson ME, Riley JL, et al. Sex, gender, and blood pressure: contributions to experimental pain report. *Psychosom Med*. 2001; 21: 853-60.
21. Bruehl S, Chung OY. Interactions between the cardiovascular and pain regulatory systems: an updated review of mechanisms and possible alterations in chronic pain. *Neurosci Biobehav Rev*. 2004; 28: 395-414.
22. Millan MJ. Descending control of pain. *Progressive Neurobiology*. 2002; 66: 355-474.
23. Maixner W, Fillingim RB, Kincaid S, et al. Relationship between pain sensitivity and resting arterial blood pressure in patients with painful temporomandibular disorders. *Psychosom Med*. 1997; 59: 503-11.
24. Evers AW, Zautra A, Thieme K. Stress and resilience in rheumatic diseases: a review and glimpse into the future. *Nat Rev Rheumatol*. 2011; 7: 409-15.
25. Fordyce WE. Behavioral methods for chronic pain and illness. St. Louis, CV Mosby Co. 1976.
26. Thieme K, Spies C, Sinha P, et al. Predictors of pain behaviors in fibromyalgia syndrome. *Arthritis Rheum*. 2005; 53: 343-50.
27. Turk D, Okifuji A. What factors affect physicians' decisions to prescribe opioids for chronic noncancer pain patients? *Clin J Pain*. 1997; 13: 330-6.
28. Turk DC, Okifuji A. Perception of traumatic onset and compensation status: impact on pain severity, emotional distress, and disability in chronic pain patients. *J Behav Med*. 1996; 9: 435-53.
29. Turk DC, Okifuji A. Evaluating the role of physical, operant, cognitive, and affective factors in the pain behaviors of chronic pain patients. *Behav Modification*. 1997; 21: 259-80.
30. Von Korff M, Barlow W, Cherklin D, Deyo RA. The effects of practice style in managing back pain. *Ann Intern Med*. 1994; 121: 182-95.
31. Thieme K, Gromnica-Ihle E, Flor H. Operant behavioral treatment of fibromyalgia: a controlled study. *Arthritis Rheum*. 2003; 49: 314-20.
32. Thieme K, Flor H, Turk DC. Psychological pain treatment in fibromyalgia syndrome: efficacy of operant behavioural and cognitive behavioural treatments. *Arthritis Res Ther*. 2006; 8: R121.
33. Martell BA, O'Connor PG, Kerns RD, et al. Systematic review: opioid treatment for chronic back pain: prevalence, efficacy, and association with addiction. *Ann Intern Med*. 2007; 146: 116-27.
34. Vlaeyen JW, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. *Pain*. 2012; 153: 1144-7.
35. Leeuw M, Goossens ME, Linton SJ, et al. The fear-avoidance model of musculoskeletal pain: current state of scientific evidence. *J Behav Med*. 2007; 30: 77-94.
36. Burns JW, Kubilus A, Bruehl S, et al. Do changes in cognitive factors influence outcome following multidisciplinary treatment for chronic pain? A cross-lagged panel analysis. *J Consult Clin Psychol*. 2003; 71: 81-91.
37. Merrick D, Sjolund BH. Patients' pretreatment beliefs about recovery influence outcomes of a pain rehabilitation program. *Eur J Phys Rehabil Med*. 2009; 45: 391-401.
38. Thieme K, Turk DC, Flor H. Comorbid depression and anxiety in fibromyalgia syndrome: Relationship to somatic and psychosocial variables. *Psychosom Med*. 2004; 66: 837-44.
39. Romano JM, Turner JA, Jensen MP, et al. Chronic pain patient-spouse behavioral interactions predict patient disability. *Pain*. 1995; 63: 353-60.
40. Nicassio PM, Radojevic V, Weisman MH, et al. A comparison of behavioral and educational interventions for fibromyalgia. *J Rheumatol*. 1997; 24: 2000-7.
41. Flor H, Birbaumer N. Psychobiologie und interdisziplinäre Therapie chronischer Wirbelsäulensyndrome [Psychobiology and interdisciplinary treatment of chronic back pain]. München, GSF Forschungszentrum. 1994.
42. Evers AW, Zautra A, Thieme K. Stress and resilience in rheumatic diseases: a review and glimpse into the future. *Nature Review Rheumatology* 2011; 7 (7): 409-15.
43. Diers M, Yilmaz P, Rance M, et al. Treatment-related changes in brain activation in patients with fibromyalgia syndrome. *Exp Brain Res*. 2012; 218: 619-28.
44. Bennett RM, Nelson D. Cognitive behavioral therapy for fibromyalgia. *Nat Clin Prac Rheum*. 2006; 2: 417-24.
45. Turk DC, Meichenbaum D, Genest M. Pain and behavioral medicine: a cognitive-behavioral perspective. New York, Guilford. 1983.
46. Rossy LA, Buckelew SP, Dorr N, et al. A meta-analysis of fibromyalgia treatment interventions. *Ann Behav Med*. 1999; 21: 180-91.

47. Glombiewski JA, Sawyer AT, Gutermann J, et al. Psychological treatments for fibromyalgia: a meta-analysis. *Pain*. 2010; 151: 280-95.
48. Morley S, Eccleston Ch, William A. Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain* 1999; 80: 1-13.
49. Bernardy K, Füber N, Köllner V, Häuser W. Efficacy of cognitive-behavioral therapies in fibromyalgia syndrome - a systematic review and metaanalysis of randomized controlled trials. *J Rheumatol*. 2010; 37: 1991-2005.
50. Thieme K, Gracely RH. Are psychological treatments effective for fibromyalgia pain? *Curr Rheumatol Rep*. 2009; 11: 443-50.
51. Thieme K, Turk DC, Flor H. Responder criteria for operant and cognitive-behavioral treatment of fibromyalgia syndrome. *Arthritis Rheum*. 2007; 57: 830-6.
52. van Koulil S, Kraaimaat FW, van Lankveld W, et al. Cognitive-behavioral mechanisms in a pain-avoidance and a pain-persistence treatment for high-risk fibromyalgia patients. *Arthritis Care Res*. 2011; 63: 800-7.