Review

Fibromyalgia and post-traumatic stress disorder: different parts of an elephant?

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ABSTRACT

Post-traumatic stress disorder (PTSD) and fibromyalgia (FM) are prevalent and debilitating conditions, conventionally delineated by distinct symptom profiles: PTSD is typified by intrusive thoughts and avoidance behaviours, while FM is essentially characterised by widespread pain, cognitive disturbances and fatigue. Despite these differences in definition, these disorders share a multitude of clinical features and risk factors, including persistent psychological distress. Furthermore, they often co-occur. Hyperactivity of the salience network, recognised as a key pathogenic feature of PTSD, has been recently suggested to also underlie FM, broadening the prevailing concept of central pain sensitisation. These observations prompt the hypothesis that these conditions have common vulnerability factors, characterised by a maladaptive response to stress perpetuated by a persistently heightened perception of threat and low ability to sooth the threats.

This paper explores this hypothesis, by analysing the commonalities between FM and PTSD, in line with the FITSS model, and how this may, eventually, foster cross-fertilisation of knowledge stemming from both perspectives, to the benefit of patients.

Introduction

Fibromyalgia (FM) affects around 2.7% of the world population and inflicts a heavy burden on patients and society at large (1). Despite this, it remains a controversial condition lacking effective treatments and robust pathophysiological explanations required to enabling fostering much-needed therapeutic progress.

A recently proposed innovative theory, the Fibromyalgia: Imbalance of Threat and Soothing Systems model (FITSS) model, suggests that FM may arise and persist due to continuous (hyper)activation of the salience network (SN) (2-5). This invites reflection on the commonalities between FM and post-traumatic stress disorder (PTSD), the prototypical condition attributed to hyperactivation of the SN (6, 7). The potential association between FM and PTSD is not a new concept. The term affective spectrum disorders (ASD), first defined in 1990, encompasses 14 different medical conditions, including FM and PTSD (8-11). Both were also included under the umbrella term central sensitivity syndromes (12).

Many labels grouping FM and other conditions with persistent physical symptoms are used, including dysfunctional syndromes (13), chronic overlapping pain conditions (14, 15), medically unexplained symptoms (16), functional somatic syndromes (17) and bodily distress syndromes (18). These overarching labels are focused on the manifestation of somatic symptoms rather than on the assumed underlying mechanisms. They retain the inclusion of FM while dropping PTSD, drifting them apart (13-15). As all lumping labels, these are controversial and require scrutiny for multiple sorts of bias (19). However, we should not lose sight of the risks of artificial splitting either. Overall, the medical conditions included under these labels are frequently comorbid/concomitant and share overlap of risk factors, family aggregation, symptoms and response to some antidepressants and to some forms of psychotherapy (20). It has been suggested that lower physical and emotional adaptability and stress system dysregulation may play a role in the persistence and fluctuations in severity of these conditions over time (21-25).

Table I. Classification and diagnostic criteria of FM and PTSD over time.

FM			PTSD		
Criteria	**Author, year**	**Main features**	**Criteria**	**Author, year**	**Main features**
ACR	Wolfe, 1990	1. Widespread pain 2. Tender points on palpation 3. Symptoms duration ≥3 months	DSM-III	APA, 1980	Trauma: stressor that would evoke significant distress symptoms in any individual Exposure: not defined Symptoms: re-experience, numbing, arousal and avoidance Symptom duration: not defined
ACR	Wolfe, 2010	1. Widespread pain[1] in combination with fatigue, sleep problems, cognitive symptoms and/ or somatic symptoms assessed by the physician[2] 2. Symptoms duration ≥3 months 3. No other explanation for the pain	DSM-IV	APA, 1994	Trauma: actual/ threatened death or serious injury/ threat to physical integrity Exposure: direct, witnessed, indirect/ through others Symptoms: re-experience, numbing, hyperarousal Symptom duration: ≥1 month
ACR	Wolfe, 2016	1. Generalised pain[3] 2. Widespread pain[1] in combination with fatigue, waking unrefreshed, cognitive symptoms and/ or somatic symptoms (self-report)[2] 3. Symptoms duration ≥3 months 4. Diagnosis is valid irrespective of other concomitant diagnoses	DSM-5	APA, 2013	Trauma: actual/ threatened death, serious injury, or sexual violence Exposure: direct, witnessed, indirect/ through others, repeated/ extreme Symptoms: intrusion, avoidance, altered cognition/mood, altered arousal/ reactivity Symptom duration: ≥1 month
AAPT	Arnold, 2019	1. Multi-site pain 2. Sleep problems OR fatigue assessed by the physician 3. Symptoms duration ≥3 months 4. Diagnosis is valid irrespective of other concomitant diagnoses			

AAPT: ACTTION-APS Pain Taxonomy; ACR: American College of Rheumatology; APA: American Psychiatric Association; DSM: Diagnostic and Statistical Manual of Mental Ilnesses.
[1]According to the Widespread Pain Index Score; [2]According to the Symptom Severity Score; [3]Defined as pain in at least 4 of 5 regions.

Many of the symptoms expressed can be seen as part of a normal and adaptive sickness response as they offen contribute to healing and recovery. However, if chronic and hyperresponsive to non-pathological triggers of various natures, these responses become maladaptive and pathogenic (26, 27).

We herein review the clinical and neuropsychological similarities and differences between FM and PTSD, exploring the potential applicability and usefulness of cross-fertilisation of knowledge accumulated with each condition. To this purpose, we conducted a comprehensive narrative review of the literature. The complexity of the topic and scarcity of studies directly comparing the two conditions precluded a systematic and quantitative approach.

The case definitions of FM and PTSD and their evolution

Table I presents the classification and diagnostic criteria over time of FM and PTSD.

With regard to FM, the tender point count, which was a major pillar of the original ACR 1990 classification criteria, was replaced in the ACR 2010 preliminary diagnostic criteria by a score of self-reported widespread pain in combination with fatigue, non-restorative sleep and cognitive dysfunction on a continuum of *fibromyalgianess* (28, 29). The 2019 AAPT diagnostic criteria are limited to three core symptoms, multisite pain, with moderate to severe sleep disturbance or fatigue, as assessed by the physician, for at least 3 months (26).

The definition of PTSD has been the subject of continued controversy since its introduction as a psychiatric disorder in 1980, in the Diagnostic and Statistical Manual of Mental Disorders III (DSM-III). The most controversial issue in the latest DSM-5 criteria (31) concerns the new definition of trauma: 'actual or threatened death, serious injury, or sexual violence', which excludes the consideration of most psychosocial stressors as potential triggers of PTSD. In addition to trauma, which remains a mandatory criterion, the new criteria also highlight PTSD-related hyperarousal, altered cognitions, avoidance and intrusion (32, 33) (Table I).

The classification and diagnostic criteria portray FM and PTSD as different disorders. The former is construed around widespread pain, with no reference to trauma and only mentions psychological distress as one of multiple symptoms that need not to be present, while the latter is based on a triggering traumatic event and on psychological symptoms, with emphasis on intrusion and no reference to somatic symptoms including pain. Cognitive and mood disturbances are included (although not

mandatory) in current diagnostic criteria for each of the two conditions.

That diagnostic criteria picture of FM and PTSD as different disorders should not be surprising, as these criteria are designed to be specific, i.e to discriminate each condition from potential misdiagnosis, thus requiring the highlight of differences and waning of commonalities. In practice, however, the core features highlighted in criteria are associated with a diversity of nuances and features that largely overlaps and blurs this seemingly clear distinction. In fact, a large proportion of patients is indicated to concomitantly satisfy criteria sets of PTSD and FM (34-38).

Co-occurrence between the two conditions

Reports on the comorbidity of FM and PTSD are influenced by the diagnostic criteria that applied at the time of the research. Studies have found that 15-72% of FM patients concomitantly fulfills criteria for PTSD (36-39) and have a 3- to 5-fold increased risk of having PTSD (40, 41). Many additional FM patients may meet criteria for partial PTSD, meaning they present symptoms of 3 of the 4 mandatory domains defined in the DSM-5 diagnosis of PTSD (42).

The prevalence of PTSD is higher in chronic pain conditions, particularly in those with chronic widespread pain (CWP) (43). In fact, PTSD was indicated to be more prevalent in FM than in rheumatoid arthritis (RA) (37% *vs*. 9%, respectively) (44). Noteworthy, when RA is comorbid with FM the prevalence of PTSD is similar to that observed in primary FM (20.8 *vs*. 22.5%, respectively) (45). These observations suggest that the mechanisms underlying and the characteristics of chronic pain in these different conditions, rather than pain *per se*, may, together with other factors, account for the special epidemiological link between FM and PTSD. Conversely, 11–21% of individuals with PTSD also satisfy criteria for FM (34, 35).

The in-between diagnosis that never was: complex PTSD or DESNOS

The overlap of symptoms between FM and PTSD is central to an interesting and instructive piece of scientific history: that of disorders of extreme stress not otherwise specified (DESNOS).

Trauma has been classified into type I: single and unexpected trauma, and type II: repeated/extended and anticipated trauma, usually with onset in early life (46). Although both are considered risk factors for PTSD development, type I is highlighted to the point of being included in the diagnostic criteria. However, two prospective studies including a total of 696 patients, suggest that type II trauma is a stronger risk factor for PTSD than type I, and is associated with more alterations in attention and consciousness (dissociation), shame, intrusions and flashbacks (47, 48).

Some authors believe that the clinical picture resulting from type II trauma is different enough to be distinguished from classical PTSD and proposed the designation of DESNOS or complex PTSD (31, 49). This profile retains the four defining criteria of classic PTSD (intrusion, avoidance, negative cognitions/mood and hyperarousal/reactivity) while adding five new categories, all based on disturbances in self-regulatory capacities: dissociation, adversely affected belief systems and somatic symptoms of distress (somatisation) (50, 51). There are similarities between these symptom categories and features considered common in FM. The fact that FM is predominantly associated with type II trauma underlines and may contribute to the overlap of FM and PTSD (2, 52).

The suggestion for a complex PTSD classification was supported for a long-time by a large group of prestigious researchers and backed up by a remarkable wealth of evidence, leading to its recognition by the World Health Organisation in the latest ICD-11 (International Classification of Diseases, 11th Revision, Complex PTSD, Code 6B41). However, because no diagnostic criteria were ever developed for complex PTSD and almost all these patients fulfilled the criteria for PTSD, complex PTSD (or DESNOS) was never recognised by the American Psychiatric Association in the Diagnostic and Statistical Manual of Mental Disorders (DSM) criteria (53). Shortly before the latest DSM-5 came to light, the 2010 ACR preliminary diagnostic criteria for FM had been published. These criteria and those of 2016 included depression as a (non-obligatory) part of the symptoms used in diagnosis. Although the relative weight of depression remained quite small, this was the first time a psychological feature was included in the diagnostic criteria of FM. Despite this, the two constructs, FM and PTSD, continued to drift apart.

Commonalities and differences in the experiences of people with FM and PTSD

Published epidemiological studies on clinical features typically focus exclusively on either FM or PTSD, thereby emphasising the core symptoms associated with each condition and overlooking potential similarities or shared features.

Published descriptions and clinical experience suggest that the degree of overlap of clinical features associated with each condition is much higher if we leave aside the criteria used for classification and diagnosis, and encompass the complex experience of people with either of these conditions. To explore this, we started by defining a full set of clinical features of interest, including psychosomatic symptoms and psychological dimensions that emerged as relevant in the elaboration of the FITSS model for FM (2) and from recent reviews of PTSD (54). We took the *a priori* decision that all the features described in one or both conditions would be included in an extensive literature search, to explore both similarities and differences and also to highlight potential literature gaps and the need for further studies on these dimensions.

Our initial approach was to conduct a systematic review of the literature on each manifestation for each condition, but after a very exhaustive effort to do so, we had to conclude that this was not feasible, not only due to the large number of features under scrutiny but, essentially, to the limitations of the available evidence. While the literature search on some clinical features identified over one thousand potentially usable articles, for many others there were only with few or none at all. Analyzing

the search results of potentially usable articles for a number of randomly selected features we observed that, more frequently than not, objective prevalence data were lacking to compare both conditions (Supplementary File).

We could not find any study that systematically evaluated the prevalence of most selected features in each or in both conditions: the full scope of clinical features of these two conditions has never been directly contrasted before. As an alternative approach, we searched and thoroughly read the most cited reviews on FM and on PTSD, to gauge the prevalence of each manifestation of interest. However, these articles mostly focused on classical well-established clinical features of each disease, not venturing into less studied and more exploratory ones.

Finally, we decided to focus only on reviews that addressed both conditions. This search resulted in 7 review articles, all dedicated to explanatory models for the overlap between FM and PTSD (36, 55-60). This set includes, we believe, the most comprehensive reviews of clinical features of both conditions available in the literature. None of these publications presented original prevalence data, rather they were based on prior publications, which are inherently biased by the disease under focus and its putative core features. We took into account all the clinical features mentioned in each article. Table II is a semiquantitative representation of our summative appraisal of these reviews.

There is overlap of several reported clinical features between the two conditions, starting with experienced trauma (a specific PTSD feature) in FM and chronic pain (a specific FM feature) in PTSD. All the somatic symptoms commonly seen in FM are also described in PTSD (and mostly with similar reported strength of association with trauma severity. Similarly, all the classical features of PTSD (with the exception of intrusion) are also found in FM, though the number of studies and strength of association reported are generally lower than those for PTSD. Depression and anxiety stand out in both conditions and are mentioned in most reviews.

Table II. Clinical features reported in published reviews of FM *vs*. PTSD.

Estimates of the prevalence and association of trauma and individual clinical features in patients with FM and/or PTSD, according to 7 comprehensive literature reviews (36, 55, 57-60, 102)**.** Numbers 1 to 3 in both the above columns and the colour intensity reflect the reported strength of association of each manifestation to either diagnosis, as assessed in each publication: 1. association reported; 2. strong association; 3. very strong/(almost) universal association. (Please note: most often these classification terms were used as qualitative descriptors, without rigorous quantitative reference data.)

The size of the circle symbolises the number of articles mentioning the feature (the larger the circle, the higher the number of reviews mentioning the feature, within a minimum of 1 and a maximum of 7). This analysis was conducted by two independent reviewers and the results of each compared and contrasted. All divergences were discussed until consensus was reached.

The most noticeable difference relates to 'intrusion', which is mentioned only for PTSD and is generally considered a differential feature of this condition. Intrusion is defined, in the DSM-5 criteria, as the presence of at least one of the following: i. repeated, involuntary memories; ii. distressing dreams, iii. flashbacks of the traumatic event, iv) psychological distress, or v. physi. logical reactions when exposed to cues resembling the traumatic event. This means that intrusion can be presented in the form of distressing dreams and psychological distress, features commonly found in both conditions (61, 62).

Also all other features show similarities between PTSD and FM. It cannot be excluded that the differences reported reflect the diagnostic criteria of the condition of interest in each original publication. This aspect is highlighted by trauma, which, although reported in both conditions, acquires a prominent position in PTSD, in agreement with its defining role for this condition. The opposite can be said for chronic pain and fatigue: common in both conditions, but way more prominent in the literature portraying FM, in line with the diagnostic criteria.

Another point is the lack of reference to personality traits, such as neuroticism, perfectionism, and introversion, in the descriptive reviews contrasting FM and PTSD, despite the recognition of their relevance in studies focusing on each of the conditions individually (63, 64). For clinicians, the hypothesis that these apparent differences between the two diseases are a result of bias on diagnosis and reporting is unavoidable. To start with, most specialised centres and individual researchers are dedicated to either one or the other disease and this will inevitably affect the direction of

the clinical enquiry, additional investigations, final diagnosis and management (20). Clinicians and researchers will be confronted with the decision of whether, for instance, psychological trauma in early infancy or physical abuse in adulthood should be considered major and taken as a driving factor, favouring PTSD, or as a subsidiary aspect in the presence of generalised pain. Depending on the inclination of the evaluator (psychiatrist *vs*. rheumatologist, for instance), multisite pain may emerge as the defining feature, in favour of FM, or be relegated to the background in a PTSD-prone context. Repetitive thoughts, rumination and disturbing dreams may be considered by different clinicians as demonstrative of intrusion, favouring PTSD, or as a common, although diagnostically irrelevant, manifestation of FM. In sum, it seems possible, if not probable that, depending on the specific interest of clinician or researcher, different features may be seen as core in each case, and that the additional inquiry will be geared towards a specific diagnosis, ignoring the background (even if predominant) mass of nonspecific features.

A note on treatment

When it comes to treatment options, there are similarities and differences between the two conditions, as described in current treatment recommendations (65-69). Regarding psychological therapies, cognitive behaviour therapy is the first choice for both conditions, although the strength of evidence for the recommendation was only 'weak for' in FM (65, 70). In PTSD, trauma-focused cognitive behaviour therapy appears to be the most efficient intervention, although the dropout rate is higher compared with non-trauma-focused alternatives, such as eye movement desensitisation and reprocessing. Regarding FM, a Cochrane review on 23 randomised-controlled trials found a small benefit of CBT on pain, negative mood and disability. However, most studies had a high risk of bias and the overall quality of evidence was considered low (71, 72).

For pharmacological treatment, serotonin-norepinephrine reuptake inhibitors (venlafaxine in PTSD and duloxetine in FM) and amitriptyline are recommended in both conditions although recommendation strength is weak for FM. These similarities do not constitute a strong argument bringing the two conditions together as they are commonly used in a variety of stress disorders (65). There are, conversely, some (relative) differences worth mentioning. Selective serotonin reuptake inhibitors, the first-line drug treatment in PTSD, failed to show consistent efficacy in FM (73). Given the risk of comorbid substance use disorder, weak opioids such as tramadol, used in FM only for those with severe pain, are considered contraindicated in PTSD (65, 74). Physical exercise, a cornerstone in the treatment of FM, is not mentioned in any of the PTSD guidelines, although there is promising evidence for its use also in PTSD (75).

The diagnostic role of pain and trauma

The prevailing distinction between FM and PTSD appears to hinge on the predominance of CWP and other symptoms in the former *vs*. lingering consequences of trauma and intrusion symptoms in the latter. Emerging evidence, may be interpreted as suggesting that this demarcation is artificially accentuated by current diagnostic criteria.

Chronic widespread pain

CWP is the hallmark of FM, but a systematic review found that it is frequently observed in PTSD, according to 16 out of 19 (84.2%) studies (76). In their 2001 review, Sharp and Harvey reported that CWP is observed in 20-80% of PTSD individuals, and that criteria for PTSD are satisfied in 10-50% of CWP cases (60). Defrin *et al*. (77) evaluated i) pain perception through quantitative somatosensory testing, ii) the presence of chronic pain, iii) PTSD symptoms, and iv) anxiety levels in people with PTSD, anxiety disorders and healthy controls. They found that people with PTSD had a higher frequency and intensity of chronic pain and higher number of painful body regions, than subjects with anxiety disorders and healthy controls. On top of that, the PTSD group presented higher pain thresholds while, at the same time, perceived suprathreshold (pain visual analogue score of 8 on a 0–10 scale, 10 the most severe) thermal stimuli as more intense, compared with the other groups. In sum, they seem to have a hyposensitivity to pain while noxious pain stimuli are experienced as more intense and more widespread. These findings align with a 2020 meta-analysis by Tesarz *et al*., which showed that pain perception in PTSD varies by trauma type. Specifically, combat-related PTSD was associated with elevated pain thresholds, consistent with Defrin's cohort, while accident-related PTSD showed reduced pain thresholds, highlighting trauma-specific differences. The meta-analysis further linked dissociative symptoms (common in combat-related PTSD) to delayed pain detection and higher pain thresholds, whereas anxiety sensitivity amplified suprathreshold pain intensity and chronic pain experiences. This may explain Defrin's observation of heightened responses to noxious stimuli despite baseline hyposensitivity (78).

Among the processes mediating the relationship between CWP and PTSD is pain catastrophising, a cognitive response that involves a tendency to overestimate the threat value of a painful stimulus and to feel worried or helpless about pain. Catastrophising decreases the probability of pain resolution and increases the risk of progression to chronic pain (77, 79, 80). A similar mechanism is argued to exist in FM, where researchers posit that the experience of pain is interpreted as a threat and consequently develops into fear or trauma, leading to maladaptive behaviour and persistent pain disability (81).

According to the fear-avoidance model of chronic pain, both pain catastrophising and psychological inflexibility contribute to fear of pain, which in turn lead to avoidance behaviours and bodily hypervigilance, features that are typically observed in both PTSD (55, 82) and FM (83). Psychological flexibility is defined as 'the ability to contact the present moment fully as a conscious human being, and to change or persist in behaviour when doing so serves valued ends' (84). Psychological inflexibility

has been indicated as a contributor in chronic pain conditions (85) including FM , being associated with worse outcomes (increased pain intensity, anxiety and depression and worse physical and mental functioning) (86, 87). In PTSD, psychological inflexibility was found to be associated with worse symptom severity after accounting for personality and classical PTSD risk factors (88).

A potential mechanism for the interaction between psychological inflexibility and pain relates to the presence of a dysfunctional anterior cingulate cortex, a brain region integrated in the SN, responsible for a host of cognitive-emotional functions, including the processing of the affective component of pain. Altered structure and function of the anterior cingulate cortex have been described both in individuals with PTSD (79, 89) and in patients with long-standing FM (90) although the issue of 'cause or consequence' is still obscure.

Trauma

While experienced trauma remains mandatory for the diagnosis of PTSD, its role in FM is less conspicuous and less often discussed.

A history of major trauma is reported by FM patients as preceding the onset of disease in 20% to 90% of all cases of FM, depending on the definition of trauma adopted (91, 92). This does not necessarily mean that trauma caused FM, as many of these patients already have, at the time of trauma, predictors of FM, such as health-seeking behaviour and somatisation symptoms (39, 93). In such cases, it is hypothesised that FM may be 'triggered or highlighted' as opposed to 'being caused' by trauma (94). Trauma could for some people operate as the drop off water that makes the glass overflow, the trigger responsible for pushing people with higher levels of fibromyalgianess beyond the cut-off that clinically defines FM (95). The same may hold true for PTSD. Contrary to the common belief, longitudinal studies have shown that, frequently, PTSD does not emerge after one single trauma event, but rather after an accumulation of significant stressors over time, until a diagnostic cut-off is crossed (96). In fact, despite the differences suggested by the diagnostic criteria, type II is the predominant form of trauma also in PTSD. (47, 48). This continuous accumulation of stressors resembles what is frequently observed in FM, as a correlate of the heightened perception of threat highlighted by the FITSS Model (2). This is similar to the previously described type II trauma, immersed as it is in what has been called 'The Generalised Unsafety Theory of Stress' (97).

The strict definition of trauma integrated in the DSM-5 criteria for PTSD, apparently enlarges the distance between this condition and FM, as it excludes psychological stress as a potential cause or trigger of PTSD. However, many studies and researchers adopt a broader and more subjective definition of trauma, or use self-reports to assess trauma. These broader definitions include not only physical, but also emotional, psychological, and sexual trauma, thus encompassing more psychosocial stressors than the DSM-5 definition (31, 33, 98-101). This viewpoint brings the concepts of trauma and chronic stress closer together (102, 103) by recognising them as essentially transactional constructs, dependent on the individual's perception, which is modulated by a variety of factors of biologic, psychosocial and cultural nature.

In summary, the difference between the two conditions regarding trauma seems to be, in reality, less striking than suggested by diagnostic criteria.

Risk/predisposing factors

Three published meta-analyses focused on the identification of risk factors for PTSD (104-106). No meta-analysis or systematic literature reviews is available for FM; only a few narrative reviews address this topic (103, 107-111). Below, we summarise the main risk factors identified for both conditions.

Psychosocial and cognitive factors

Published reports indicate that both people with FM (38, 107, 109, 112, 113) and people with PTSD (38, 54, 104, 105, 113, 114) have an increased prevalence of comorbid and premorbid affective disorders, cognitive-behavioural features and symptoms. These include depression and anxiety disorders, sleep disturbance, avoidant behaviour and catastrophising cognitions. The relationship between these conditions and the presence of FM or PTSD is likely dynamic and bidirectional: Hypervigilant responsivity, catastrophising, avoidance, intrusive thoughts, and chronic stress (dispositional or in response to pain or trauma) are assumed to enhance sensitivity to pain and induce other symptoms (115-118). These may, in turn, enhance behaviour that maintains the symptoms, closing a vicious circle that increases anxious and/or depressed states and pain amplification. This has been conceptually explored in FM (119), but the same mechanisms can be argued to be present in PTSD: increased attention to environmental cues (hypervigilance, catastrophising) may result in persistent monitoring for threatening stimuli and limit the ability to integrate new emotional and sensory functions (120). Increased risk of disease associated with poor social support, family dysfunction and lack of emotional intelligence have been proposed for both FM (109) and PTSD (104, 105, 117, 121).

Lower than general intelligence quotient (IQ) has been pointed out as a risk factor for PTSD (104, 114, 117). While very limited information is available on FM, one study from the 1958 British Birth Cohort found that children with higher IQ were less likely than those with a general IQ to report chronic pain as adults (122).

Personality

Several personality traits have been proposed as vulnerability factors, both for FM and PTSD. Blumer and Engel identified a so-called 'pain-prone personality' linked to FM, characterised by compulsive perfectionism, high arousal, increased stress perception, highly competitive profile, rumination and persistent negative cognitions (123, 124). While the subsequent efforts to identify a FM-specific personality profile have been generally considered failures (125-127), they demonstrated that the personality traits mentioned above and other temperamental and personality traits may differ between FM pa-

tients and healthy controls (128). These include higher levels of harm-avoidance, neuroticism, and alexithymia, and lower levels of extraversion and self-directedness (109, 129-132), identified both in cross-sectional and expert-based studies (133). It is important to emphasise that these associations cannot be seen as implying causality, which cannot be fully explored due to the paucity of prospective studies in individuals at risk to develop FM.

Similar traits have been shown to be more prevalent in PTSD, with some being considered strong risk factors (*e.g.* neuroticism) or protective factors (*e.g.* extraversion) for the development of this condition (114, 134-136). Some of these studies explored personality traits (negative affect, neuroticism, hostility, extraversion/introversion, harm avoidance, novelty seeking, reward dependence, persistence and alexithymia) in soldiers before the PTSD triggering traumatic event. Such observations, in a prospective design, suggest a moderator relationship between neuroticism-like personality traits (and other risk factors) and the risk of emergence of PTSD. No similar prospective data is available for FM.

Demographic and life-style factors

Female gender is one of the most established risk factors for both PTSD and FM (54, 104, 106, 107, 109). This may partly be explained by gender-associated biases (brave men and emotional women) in self-observation and observations by others (19).

Several lifestyle habits are recogniaed as risk factors for both conditions, including obesity (107, 109, 110, 137), lack of physical activity (109, 110, 138), smoking and alcohol abuse (107, 138).

The same applies for lower sociodemographic status, low education level and blue-collar occupation (104, 107, 114). Probably, these are not (all) independent risk factors.

Belonging to an ethnic minority has also been pointed out as a risk factor for PTSD (104, 106), while no data is available for FM in this matter. We could find no evidence that a risk factor in one condition can be excluded as relevant in the other.

Trauma specifics

The role of trauma and trauma characteristics in both FM and PTSD has been the focus of a previous section of this paper. Exposure to trauma has been pointed as a risk factor for FM (92), particularly physical trauma (103, 109). Less evidence is available regarding emotional/ psychological trauma. In PTSD prospective research, type of trauma (103) history of previous trauma (104, 105), cumulative exposure to trauma (54,106) and trauma severity (54, 104-106) are all well established risk factors.

Early adversity

The association between adverse childhood experiences (or early stress, early life adversity, childhood trauma, childhood neglect, and other possible nomenclatures) with both FM (42, 91, 131-138) and PTSD (104, 140) has been recognised for a long time, although causality is not unambiguously demonstrated in both conditions.

Exposure to adversity early in life may impair the learning of safety cues, disrupt reward processing and hinder effective socioemotional development. Learning deficits characterised by fear overestimation, blurred differentiation between safety and threat, and inept extinction of threat perceptions have been documented in both PTSD (144-148) and FM (81, 149-151) and may constitute a fundamental process in the emergence and maintenance of both conditions.

Repeated exposure to threatening experiences, especially in early childhood, can induce persistent neurophysiological changes that immortalise a negative perspective of life events and distort stress responses, which contribute to maintain and amplify symptoms and maladaptive responses over time (139, 152). The potential importance of these mechanisms in the development and maintenance of stress-related conditions, such as FM and PTSD, has been extensively debated (108, 139). Dysfunction of the hypothalamic-pituitary-adrenal axis, one of our main stress controlling mechanisms, has been shown to predict CWP among children exposed to adverse childhood experiences (153). It has also been demonstrated that these children have differences in their resting state neural networks, mainly in the default mode network and the SN at large (154). A recent analysis of data from the Adolescent Brain and Cognitive Development (ABCD) study, indicates that similar changes, including increased functional connectivity between the SN and other neural networks, precede the onset of CWP for at least one year (111).

Other medical conditions

History of other painful conditions is one of the most commonly recognised risk factors for FM (107, 109, 155), suggesting that this condition may actually be the tip of the iceberg of a mounting level of distress and pain developed through the years. Several chronic non-painful conditions have been identified as predictors of FM, including anxiety-depression disorders (107-109), sleep disorders (107, 109, 110) and chronic infectious diseases (109). A comprehensive review on the incidence and risk factors for FM is even more inclusive and concludes that the presence of any other medical condition, particularly sleep disorders, headaches, depression and illness behaviour, increases the risk of developing FM (107).

Regarding PTSD, research on comorbid medical conditions mostly focus on psychiatric disorders. Still, a previous history of any chronic or major physical disease has been indicated as a risk factor for development of PTSD (106). Besides personal psychiatric comorbidities, family psychiatric history is also a recognised risk factor, mainly in PTSD (104-106).

Genetic factors

The contribution of genetic factors for either FM and PTSD is supported by family aggregation and data coming from twin studies. Chronic pain syndromes, including FM, showed an estimated hereditability of about 66% (of which 27% attributed to genetics) (156), while in PTSD genetic factors are hold responsible for around 30% of the risk of developing the condition (117). Serotonin-related pathway genes were found to be associated with both FM and PTSD (109, 117).

Other psychosocial factors

The observations described in this section, highlight that the associated risk and predisposing factors identified for FM and PTSD are strikingly similar.

Also other psychosocial factors including personal and familiar psychiatric comorbidities, lack of social support and maladjusted behaviour, are regularly mentioned as risk factors for both conditions (103-110, 157). In fact, pre-trauma factors (as opposed to peri or post-trauma factors) are highlighted as being most strongly associated with PTSD (106). This observation is in line with the most recent data from the World Mental Health Surveys, based on advanced machine learning techniques (158). The role of chronic stress (repeated trauma) in FM was underlined in a recent review (159).

Explanatory models for the overlap between PTSD and FM

Several theories have been presented to explain the similarity and overlap of PTSD and FM (40, 55, 57-59,102).

In the Mutual maintenance theory (60) pain is seen as the traumatic stimulus responsible for PTSD development. PTSD-associated hyperarousal and hypervigilance, in turn, increase and perpetuate pain and enhance the risk of developing FM.

Other authors argue for a Shared vulnerability (160): common risk factors, such as anxiety, facilitate the development and maintenance of both PTSD and FM, in the presence of trauma.

The Multiplex or triple vulnerability model (161) integrates three types of vulnerabilities: generalised biological (heritable), generalised psychological (sense of control over salient events, based on past experiences) and specific psychological (focused anxiety on specific objects/situations). An 'alarm' develops during traumatic situations and remains latent for a long time. Every time the alarm is activated, fear is unleashed leading to avoidance behaviour, hypervigilance and catastrophising, all of them have been observed in both FM and PTSD.

As an extension of the fear-avoidance model, the perpetual avoidance model (162) suggests that physical arousal resulting from autonomic nervous system activation may enhance fear of pain and avoidance behaviour which maintain chronic pain. According to this model, fear is typically elicited in response to an actual or perceived threat, while anxiety may increase the likelihood of a fear response. This model suggests that trauma may trigger both FM and PTSD. There is a dearth of research to either support or refute each of these models. It is possible that more than one, or even all of them, are operative in different degrees and at different stages in different individuals. There is a definite emphasis on psychosocial mechanisms across all these models, such as fear and its avoidance, anxiety, and catastrophising, but none of them goes as deep as to propose common underlying neurobiological mechanisms.

The FITSS model

The recently proposed Fibromyalgia Imbalance of Threat and Soothing Systems (FITSS) model (2) is an attempt to integrate the myriad factors involved in FM by highlighting the mutual cross-talk and dynamic interplay between neurophysiological and psychosocial mechanisms involved. According to this model, FM is multifactorial in nature and is best conceptualised as the end result of multiple mechanisms/factors that are interconnected as the pieces of a hanging mobile toy, so that deviations in any given mechanism/factor produce variable changes in the remaining ones and in the overall balance. Each mechanism is simultaneously a potential cause and consequence of the process, which may have different origins or causal dynamics in different people, thus accounting for the heterogeneity observed in FM. Adopting a biopsychosocial perspective, the FITSS model suggests that this condition is characterised by an imbalance between an overactive threat and an hypoactive soothing emotion regulation system, which generates a continuous influx of threat perceptions that keeps the SN in constant overdrive (3, 4). This generates a myriad of efferent signals that generate and maintain the clinical features of FM. Whether the threat/soothing imbalance is the primary cause of FM or a consequence of other operating mechanisms or of the disease features is to be considered in the framework of the hanging toy allegory.

The FITSS model for FM is based on the three pillars addressed below. Similar and well-supported mechanisms apply to PTSD.

Pillar 1: heightened threat

According to the generalised unsafety theory of stress, the default stress response is activated through the interaction of three domains: bodily state, social context and stress-related contexts (97). Similar distrurbances in all these domains can be found in FM and in PTSD. To start, both conditions are characterised by autonomic dysregulation (163, 164). Also, chronically heightened stress emerges as a feature of the psychological matrix in both conditions: both have been associated with shame and guilt feelings, with stigma and invalidation from others, and with some degree of social disconnection, (165-168) which seemingly contribute to a 'threat ecology'. Shame and guilt have been associated with higher self-critical and lower self-reassuring thinking in PTSD which have been argued to contribute to the sense of pervasive threat observed in this condition (168). Similar feelings are also reported in FM, for instance, when patients are confronted with recommendations to take better care of themselves to the detriment of others (165). The ultimate consequence of persistence of these states of mind is threat-avoidant behaviour which, in the absence of a learned soothing environment, leads to social isolation and disconnection, establishing yet another stress-provoking spiral (169, 170).

The concept of an hyperactive threat system is also supported by neuroimaging studies which typically reveal, in FM and PTSD, hyperactivation of the prefrontal cortex and the amygdala, core structures of fear regulation (171), and of the insular cortex, an integrating part of the SN (172).

Pillar 2: hypoactive soothing

The exaggerated perception of threat described above is reinforced by a lack

of safeness, soothing and affiliation that might buffer the sense of unsafety and promote well-being and positive affect states (101, 173). Soothing, in opposition to threat, is not inborn but rather acquired through learning in the early years of life, which underlines the importance of safeness and strong social bonds during childhood (2). This is better seen in the light of attachment theory according to which humans (particularly children) seek proximity to others (attachment figures) for safety and support and that this search is intensified in the presence of (perceived) threat (174). The lack of attachment figures can lead to an underdeveloped soothing-affiliative system fostering threat-avoidant behaviour (175). This makes children the more vulnerable to adverse childhood experiences that may play a role in the development of both FM and PTSD (already discussed previously).

In addition to insecure attachments and high levels of negative affect, FM and PTSD are also marked by low levels of positive emotions (176, 177). Studies have shown that positive stimuli are perceived as inferior, in terms of both intensity and valence, in FM patients, suggesting a disruption in the reward system (178). The same applies to PTSD: neuroimaging studies show lower activation of neural regions involved in emotional processing (such as the amygdala) when exposed to positive stimuli, compared to healthy controls (179).

This threat-soothing imbalance and disrupted reward system will compromise the real vs. perceived threat-value of all neural inputs, which are postulated to translate into a hyperactive SN.

Pillar 3: the salience network

The SN is a large functional brain network responsible for the detection and rapid response to biologically salient (*i.e.* threatening) stimuli in the inner and outer environment. Structured around the insula and the anterior cingulate cortex, it works as the multimodal central alarm station, keeping a comprehensive and continuous threat surveillance and launching the timely activation of the fight/flight response. The implication of the SN is well established in PTSD (120, 180, 181). A quantitative meta-analysis and systematic review confirmed the presence of hyperactivity of the SN in PTSD patients in resting-states (182). This indicates increased threat and salience processing in task-free resting-state PTSD patients, supporting the clinical observations of a constant state of hypervigilance in these patients.

Similar observations have been made in FM, namely through stronger and longer responses of the SN to a variety of painful and non-painful neutral and/or negative stimuli, compared to healthy controls (173, 183-185).

Functional alterations in the anterior cingulate gyrus and insular cortex, which integrate the SN, are reportedly the most reproducible features, both in FM (186) and in PTSD (187). This is hardly surprising, as these structures are pivotal in the regulation of both pain and emotion, supporting their intricate interplay (59, 92).

In summary, the FITSS model provides an integrated psychosocial and neurophysiological basis to explain the commonalities between FM and PTSD, from risk factors to clinical manifestations and treatment strategies, centred around the hyperactivation of the SN.

Discussion

PTSD and FM have been for long presented, conceived and investigated as independent nosological, although overlapping, entities. However, there is evidence that PTSD and FM share, more than generally assumed, in different shades and degrees, several disease features, treatment modalities, associated risk factors and triggers, including psychosocial factors. Functional MRI studies have shown that they also share similar neurobiological underpinnings, namely the hyperactivation of the SN (7, 173).

One might argue that there are more arguments to bring these conditions together, than there are to keep them as separated entities. It is important to recognise, however, that the overlap arguments of this perspective can be criticised. In fact, having a (perhaps any) single disease or symptom will enhance the risk of developing nonspecific symptoms, *e.g.* fatigue, sleep problems or even pain, which are paramount in the conditions discussed in this document. Moreover, several other characteristics and epiphenomena are often observed in people with any disease, *e.g.* a higher prevalence in women or people with lower education, the presence of emotional distress and a sedentary lifestyle, or experiencing social invalidation. The use of similar treatments is also not a decisive argument to group diseases. If the pathological substrate is unknown or cannot be treated, physical exercise, cognitive behavioural therapy, or pharmacological interventions may be recommended to mitigate the consequences of the disease or to prevent further damage. Even the similarity of the underlying mechanisms does not guarantee that diseases commonly considered as different should be lumped together. In fact, it is an impossible task to decisively quantify whether similarities outweigh differences between diagnoses and even quantified arguments would be differently valued by different observers. Conversely, there may be reasons to keep separate diagnoses for diseases, *e.g.* because other organs are involved, the aetiology is different, more than one pathological process plays a role in maintaining the disease, or a different emphasis at treatments is needed.

To a certain extent this is also not important, because both differences and similarities between diseases may offer valuable information. In this article we presented arguments to look beyond the 'automatic' criteria-based distinction between FM (focused on widespread pain and other somatic symptoms *vs.* PTSD (nucleated around intrusions related to a traumatic event). The main function of diagnostic criteria is to differentiate between diseases. This may bring and keep patients in the most specialised settings, which may beneficial. A drawback, however, of emphasising differences between diagnoses is that similarities may be overlooked, including the possibility that in both PTSD and FM the brain is essentially hypersensitive because of a persistently heightened perception of threat and low ability to sooth the threats. This can be

decisive in guiding research and selecting therapy.

We do not want to refute that differential diagnostics may be useful to obtain homogeneous groups in research and find the most appropriate treatment for patients. However, with this review we wish to encourage researchers and clinicians to adopt a transdiagnostic approach in theory, research, and treatment, *e.g.* by examining structural and functional similarities in the psychological processes and the brain of people with PTSD and FM or by developing therapies guided by these common mechanisms. Exploring the commonalities of FM and PTSD, in line with the FITSS model may foster cross-fertilisation of knowledge stemming from both perspectives, to the benefit of patients.

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Competing interests

W. Häuser is member of the medical advisory board of the European Network of Fibromyalgia Associations. He has received honoraria for consulting and educational lectures from Otto Bock, UCB and Vidal.

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