Review

Fibromyalgia and Physical Trauma: The Concepts We Invent

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ABSTRACT. Despite weak to nonexistent evidence regarding the causal association of trauma and fibromyalgia (FM), literature and court testimony continue to point out the association as if it were a strong and true association. The only data that appear unequivocally to support the notion that trauma causes FM are case reports, cases series, and studies that rely on patients' recall and attribution — very low-quality data that do not constitute scientific evidence. Five research studies have contributed evidence to the FM-trauma association. There is no scientific support for the idea that trauma overall causes FM, and evidence in regard to an effect of motor vehicle accidents on FM is weak or null. In some instances effect may be seen to precede cause. Alternative causal models that propose that trauma causes "stress" that leads to FM are unfalsifiable and unmeasurable. (J Rheumatol First Release Aug 1 2014; doi:10.3899/jrheum.140268)

Key Indexing Terms:
FIBROMYALGIA MEDICOLEGAL TRAUMA ETIOLOGY CAUSATION

> … the concepts we invent to account for disease come to shape not only the observations we make and the remedies we prescribe, but the very manifestations of disease itself. *L. Eisenberg*[1]

In 1999, the US Social Security Administration abandoned multiple protracted and ultimately futile court fights against allowing disability awards for fibromyalgia (FM), and agreed that it was a medically determinable impairment on the basis of the presence of tender points. By 2013, Google Scholar Case Law reported 5220 published cases in US federal and state courts related to FM in the previous 10 years, representing perhaps 9% of all FM cases[2].

The controversy about traumatic causation of FM is deeply entwined with the controversy about the nature and legitimacy of FM — about whether FM is primarily a neurophysiological or psychosocial illness. In the United States, almost all physician authors of FM articles have tended to support FM "neurophysiopathogenesis"[3], and have almost universally supported the idea of physical trauma causation — albeit with varying degrees of support and explanation. In a 2009 deposition, Dr. Robert Bennett epitomized the view of FM supporters when in a testimonial reply to the attorney's question, "In the community of physicians and researchers who evaluate fibromyalgia patients or do research on them, is there any significant dispute in that community that physical trauma can cause the development of fibromyalgia in some people?" Bennett answered: "Not that I am aware of"[4]. The position that trauma plays little role in FM development was expressed by Wynne-Jones, *et al,* who, writing of the results of a prospective cohort study, stated that trauma "… is unlikely to have a major impact on the new onset of widespread pain [WP]. Any observed relation may, in part, be explained by psychological distress"[5]. Only a few others have published doubts about trauma and FM[6,7,8,9,10].

The Politics of Trauma — a Debate Begins

In the medicolegal contest that relates trauma to FM in the United States, both sides seek to show that there is general agreement or "consensus" with their position, and 2

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Accepted for publication April 29, 2014.

consensus documents have emerged to become central parts of the legal contest. In 1994, one of us (FW) organized a "disability conference" in Vancouver to address FM issues and describe standards of practice. We invited all physicians and researchers who were FM experts — choosing participants on the basis of publication record and known interest. The conference report that resulted was published in this journal and would later be cited in almost all FM tort cases[11]. It would also spark considerable controversy among rheumatologists and other experts.

The 2-day Vancouver Fibromyalgia Consensus Conference discussed and voted upon issues relating to FM and disability[11]. In general, most of the conclusions were widely agreed on, but there was disagreement about FM and trauma. The committee concluded that "… the data from the literature are insufficient to indicate whether causal relationships exist between trauma and fibromyalgia." This "… does not mean that causality does not exist, rather that appropriate studies have not been performed"[11]. Based on this uncertainty, it was recommended to "eliminate the terms 'reactive' and 'post-traumatic fibromyalgia.'"

A minority of the 34 attendees disagreed with the consensus conclusions about trauma and FM, and after further e-mail discussions a second vote by e-mail occurred before submission for publication. Still unsatisfied, the group (Robert Bennett, Thomas Romano, I. Jon Russell) led by Mohammad Yunus published an attack on the committee and its conclusions in a non-research journal of recent origin. Yunus actively recruited nonparticipants in the Vancouver conference who shared his opinion to be coauthors[12]. Of the 34 original Vancouver committee members, 12 (35%) subscribed to the changes, but 22 (65%) did not agree to the Yunus revision. The Vancouver report became an important reference document in US litigation, as did the Yunus rump effort.

Five years later, what was to become known as the "Canadian consensus document" appeared[13]. In his important legal review, Finch characterized it by saying, "More recently, a group of medical researchers, medical school faculty, and physicians issued the 2003 Consensus Report. This latest report concludes that, based on existing epidemiological, clinical, and biological evidence, there is a compelling argument that trauma does, in fact, play an etiological role in the development of fibromyalgia in some, but not all patients"[14]. He characterized the Vancouver report as a "self-styled 'Consensus Report,' … a private conference of physicians and researchers sponsored by insurance-related interests"[14]. Together with the Yunus "Additional Comments" article, the Canadian consensus became the opposing consensus viewpoint, and was introduced and cited in US court documents. The 14-member Canadian conference was a designed, 1-sided effort. Among its members were the 2 American rheumatologists, I. Jon Russell and Thomas Romano, who were leaders of the Yunus Additional Comments article. The 12 Canadian members were not rheumatologists, had little if any peer-reviewed publication record, and were certainly not the medical researchers and medical school faculty described by Finch. Russell, a coauthor of the report and the editor of *Musculoskeletal Pain*, published the report in his journal.

The Association of FM and Trauma: Research Studies

The Evidence Based Medicine initiative has provided guidelines ("GRADE") for the evaluation of study quality that may be applied to FM-trauma studies[15,16]. Case reports were the initial studies linking trauma to FM (Table 1). Goldenberg convincingly described a completely healthy individual who experienced substantial facial trauma and went on to develop FM[17]. Wolfe describes a similar patient, but becomes uncertain about the patient's veracity toward the end of the report[18]. In an unusual report, Mailis, *et al* described a minor car accident, 9 years of litigation, and refutation of FM experts' diagnosis by surveillance videos[19]. Although case reports may stoke our interest, they provide no useful evidence about the association of FM and trauma.

Case series and cross-sectional surveys (Table 1) also provide very low-quality evidence[20,21,22,23], where the overwhelming biases are selection and recall bias. Al-Allaf, *et al* compared recall of traumatic events in patients with FM and controls selected from medical clinics[23]. Of responders to the authors' questionnaire, 39% of patients with FM reported significant physical trauma in the 6 months before the onset of their illness, compared with 24% of controls, OR 2.1 (95% CI 1.2, 3.5), $p < 0.005$. Recall bias[24], together with selection problems, mark this report as uninterpretable and very low-quality evidence.

Two prospective Israeli cohort studies addressed the association between neck trauma and FM, but yielded strikingly different results, with the rate of FM 36 times greater in one study than in the other. Buskila, *et al* studied 102 persons with neck injuries[25] — "nonspecific soft tissue injuries, excluding, during the case selection process, those patients who had radiographic evidence of fractures, dislocations, and subluxations." Control subjects were 59 patients with lower extremity fractures. Cases were identified over an 18-month period by studying patients attending an occupational clinic. In Israel, "persons who are working and have received injuries must attend an occupational clinic." Although their "case review resulted in an almost complete capture of all cases" attending the clinic, subjects who were injured but did not attend the clinic for any reason, including low severity, were not studied or identified. In addition, a single examiner who determined the presence or absence of FM was not blinded to the nature of the injury. The results of the study showed that subjects with neck injuries were much more likely to be found to have FM [OR 16.0 (95% CI 2.4, 669.3; Table 1)]. Severe

Table 1. Studies of trauma with fibromyalgia and chronic widespread pain.

Study Type	Author	Outcome	Quality[5,16]
Case report	Wolfe, 1994[18]	Describes FM development after trauma	Very low
	Mailis, 2000[19]	Describes FM development after trauma, in reality is malingering	Very low
	Goldenberg, 2011[17]	Describes FM development after trauma	Very low
Case series	Greenfield, 1992[20]	23% reported having antecedent trauma, surgery, or a medical illness	Very low
Cross-sectional surveys	Bennett, 2007[21]	Physical injury (not MVA) 17.1%, MVA 16.1%, surgery 16.1%	Very low
	Waylonis, 1994[22]	Of 176 patients with posttraumatic FM, 60.7% followed an MVA, 12.5% after a work injury, 7.1% after surgery, 5.4% after a sports-related injury, and 14.3% after various other traumatic events	Very low
Retrospective case-control	Al-Allaf, 2002[23]	39% FM patients reported significant physical trauma in the 6 months before the onset of their disease, compared with 24% of controls ($p < 0.007$)	Very low
Cohort	Buskila, 1997[25]	Compared FM neck injury vs leg fracture. FM found in 21.6% vs 1.7%, "13 times more frequent"	Low
	Tishler, 2006[27], 2011[28,54]	Compared FM following "whiplash" vs patients hospitalized for severe MVA trauma. 0.6% vs 0.0% developed FM in ~1 yr. Similar results after 3 yrs followup	Low
Nested case-control	Jones, 2011[30]	Cohort of 2069 non-CWP individuals: (11.6%) developed CWP over 4 yrs. Covariate adjusted OR for widespread pain from any traumatic event 1.01 (0.73–1.40), MVA 1.50 (0.89–2.52), surgery 0.77 (0.50–1.18)	Moderate
Prospective cohort	Wynne-Jones, 2006a[5]	Cohort of persons experiencing a MVA: the onset rate of CWP 6 mos later was low (8%), though in comparison with the non-MVA group there was an increased risk [RR = 1.9 (95% CI, 0.8 to 4.8, adjusted for age and sex)]; this was attenuated after adjustment for pre-MVA (prior) psychological distress and somatic symptoms [RR = 1.4 (95% CI, 0.5 to 3.2)]. A motor vehicle crash is unlikely to have a major effect on the new onset of widespread pain	Moderate
	Wynne-Jones, 2006b[31]	In cohort of persons experiencing a MVA, predictors of CWP were post-MVA symptoms (RR 2.5, 95% CI 1.2–5.1), precollision health-seeking behavior (RR 3.6, 95% CI 1.6–7.9), precollision somatization (RR 1.7, 95% CI 0.99–2.8), perceived initial injury severity (RR 1.7, 95% CI 0.9–3.3), older age (RR 3.3, 95% CI 1.5–7.1). In combination, these factors accounted for about a 20-fold difference in the risk of new-onset widespread pain	Moderate

MVA: motor vehicle accident; FM: fibromyalgia; CWP: chronic widespread pain.

selection bias, the possibility of an aggressive examiner, and the absence of blinding limit the validity of this report. In addition, the rate of FM in those with neck injuries is much greater than FM-like illness found in "whiplash" (WL) studies of motor vehicle crashes[26], but only slightly greater than in "patients with persistent neck pain 3 months after WL injury who were enrolled in a treatment program."[27] [Disclosure: FW was a co-author of the Buskila, *et al* article.]

The second Israeli study was performed by Tishler, *et al* 7 years later[28]. They identified 153 "patients diagnosed with whiplash injury after a car accident and followed them prospectively starting immediately after discharge from the emergency room." Their control subjects were "53 patients hospitalized with fractures of the limbs, spine, and ribs due to road accident." After 14.5 months 1 patient with whiplash and no control subject developed FM, $p = 0.574$. In a followup study 3 years later "three patients in the study group compared with one patient in the control group were diagnosed as having fibromyalgia"[29]. They concluded that their studies confirm previous short-term results showing that whiplash injury and road accident trauma are not

associated with an increased risk of fibromyalgia." A limitation to this study was that only "patients who complained of symptoms or signs suggestive of FM were invited for further evaluation that included a tender point examination, and that the signs and symptoms that suggested the possibility of FM were never documented in the article. Compared with a post neck injury rate of FM of < 1% in Tishler, *et al*, the prevalence of FM in the Buskila, *et al* study was 22%, a rate 36 times greater.

British epidemiologists have evaluated the FM-trauma link by studying subjects who developed chronic widespread pain (CWP) following trauma[5,30,31]. CWP is a criterion of the 1990 American College of Rheumatology (ACR) FM criteria; and in a population study 20% of those with CWP satisfied 1990 ACR FM criteria[32]. FM in the 2010 ACR criteria differs from the chronic widespread pain concept by its inclusion of non-pain symptoms, including measures of severity of fatigue, unrefreshed sleep, cognitive problems, and somatic symptom reporting[33]. The use of CWP was driven by the practical impossibility of performing the tender point count required by the 1990 ACR criteria in large epidemiological and clinic studies. At the time of the UK studies[5,30,31], the 2010 ACR FM criteria and 2011 survey modification, which didn't require that examination, were not yet available[34,35]. However, CWP appears to be a workable surrogate for FM and has several advantages: principally, the ability to evaluate large groups of subjects without diagnosis bias.

Jones, *et al* evaluated a cohort of 2069 non-patients living in the community, after determining that they did not have CWP[30]. After 4 years, 11.6% had developed CWP. To put this rate into perspective, and assuming that in CWP ~20% will satisfy FM criteria, the FM prevalence would be about 2.4%. The authors noted that the adjusted OR for any traumatic event was 1.22 (0.90–1.65), MVA (motor vehicle accident) 1.84 (1.10–3.11), and surgery 0.89 (0.59–1.33). When further adjusted to include baseline (preaccident) variables such as psychological distress, anxiety, depression, adverse health behaviors, and sleep problems, the covariate adjusted OR for CWP from any traumatic event was 1.01 (0.73–1.40), MVA 1.50 (0.89–2.52), surgery 0.77 (0.50–1.18). The authors concluded that the relationship between any trauma and development of CWP was "completely attenuated after adjustment for confounding (OR 1.01; 0.73–1.40). However, there was some evidence to suggest that involvement in a road traffic accident, specifically, may confer an increase in the risk of CWP onset."

This was a difficult study to perform and analyze, and there were many factors (biases) that could have influenced the results. First, while it is appropriate to extrapolate the results to FM — because CWP is on the path to FM, the number of cases would have been much smaller if FM was the endpoint, and no statistical association would have been shown, assuming parallel results in FM and CWP. Second, there is an important potential bias that is discussed and discounted by the authors. The study assumes that in the 4-year followup period all the cases of CWP started after the traumatic event. But it is unlikely that such was the case. Of the 213 cases of widespread pain, 20 occurred with road traffic accidents (RTA). But if as few as 4 of the 20 cases preceded the RTA, the crude OR would have been 1.22 (95% CI 0.71, 2.10; p = 0.477); and if only 2 cases were misclassified the crude OR would have been 1.38 (95% CI 0.82, 2.33; p = 0.220). So if one believes that misclassification must have occurred, as we do, the association between RTA and CWP would be further attenuated. In addition, the association of trauma to MVA may be confounded by the tendency to "build up" or exaggerate, which is common with MVA claims in general. Also, data indicate that "the elimination of compensation for pain and suffering is associated with a decreased incidence and improved prognosis of whiplash injury."[36] It is possible that such confounding might be responsible for the observed small increase in CWP associated with MVA that is not found when other traumas are studied.

In an earlier prospective cohort study from this group, Wynne-Jones, *et al* studied persons enrolled in a single insurance company who had or had not experienced a motor vehicle crash[5]. All participants were sent a questionnaire to assess precrash (or for the noncrash group, prior) psychosocial factors and widespread pain. Participants reporting precrash (prior) widespread pain were excluded. At 6 months, participants were sent a followup questionnaire to ascertain new prevalent widespread pain. Among the cohort who had experienced a crash, the new onset rate of widespread pain 6 months later was low (8%), although in comparison with the noncrash group there was an increased risk [relative risk (RR) = 1.9 (95% CI 0.8–4.8, adjusted for age and sex)]; this was attenuated after adjustment for precrash (prior) psychological distress and somatic symptoms [RR = 1.4 (95% CI 0.5–3.2)]. Wynne-Jones, *et al* concluded that the findings suggest that a "motor vehicle crash (as an example of a physically traumatic event) is unlikely to have a major impact on the new onset of widespread pain. Any observed relation may, in part, be explained by psychological distress." As with the previous study[5], the association of trauma to MVA may be also confounded by the tendency to "build up" or exaggerate, which is common with MVA claims[37,38,39].

In evaluating the actual evidence that links trauma to FM, we must discard the very low-quality evidence based on case reports and cases series, as biases are too severe to allow any conclusion. The Buskila, *et al* study[25] has a number of severe biases, provides rates of FM that are at variance with all other studies, and would be classified as very low quality using GRADE criteria. By contrast, Tishler's reports, also assessing FM developing after WL-associated car accidents in the same country but with a

different selection and evaluation process, found no association between FM and trauma[28,29]. The 2 UK studies[5,30], although concerned with CWP rather than FM, represent a model of how the trauma-FM association could be studied because they had a relatively unbiased subject selection process and an unbiased assessment process. In addition, they were able to adjust for most relevant covariates. They were also able, to some extent, to separate MVA from other trauma. Their results suggest that trauma, overall, is not causally associated with CWP, but that there might be a slight association between MVA and CWP, an association that is weak overall, attenuated when covariate adjustment is used, but possibly confounded by "build-up."

To summarize the interpretable available data as to the FM-trauma association, the Tishler study[28] finds no association, and the 2 UK studies find modest or equivocal associations [RR 1.4 (95% CI 0.6–3.3) and 1.50 (0.89–2.52)] for MVA-associated trauma with CWP after covariate adjustment. The Buskila study[25] is problematic (and probably uninterpretable) for many reasons, but particularly because its selection process is severely biased toward inclusions of subjects with severe symptoms. In addition, with respect to the Buskila study, it has been suggested that "present criteria [ACR 1990] used in determining FM may result in spuriously inflated rates of diagnosis among WL patients because of persistent localized tenderness after an MVC [motor vehicle collision]. Further, the transient nature of FM 'symptoms' among WL patients should be taken into account before making a final diagnosis"[27].

The Causal Model of FM and Trauma

The causal model posits that when FM follows trauma, trauma is the predominant cause of FM, and that cause and outcome (or effect) may be modified by confounders (covariates). Thus we may ask, "Would fibromyalgia have occurred if there were no trauma?" Because information on counterfactual conditions at the individual patient level is unknown in almost all practical settings, we are limited to causal inference at the population level (e.g., comparing average risks)[40]. However, in nonexperimental studies, measurement error can occur not only in both X (trauma) and Y (FM/CWP), but also in the assessment of their temporal direction. In addition, the etiology of most physical diseases and almost all mental disorders is multicausal, resulting from a complex interplay between genetic and environmental factors, many or all of which may not be known or measurable; and most epidemiologic research is plagued by uncertainty about assumptions[40].

One method of model description makes use of causal diagrams, which can serve as a "visual yet logically rigorous aid for summarizing assumptions about a problem and for identifying variables that must be measured and controlled to obtain unconfounded effect estimates given those assumptions."[40] We have modeled the possible causal relations with such a causal graph, also called a directed acyclic graph. In Figure 1, trauma is the primary cause and FM (or CWP) the outcome (or effect). The model describes the direct path from trauma to FM. Other variables are confounders. "Assuming that exposure precedes disease, confounding will be present if and only if exposure would remain associated with disease even if all exposure effects were removed, prevented, or blocked."[40]

THE OUTCOME: DEFINING FM IS PROBLEMATIC

I. According to ACR criteria, FM is diagnosed when certain levels of severity variables are exceeded and present for at least 3 months. The 1990 criteria require at least 11 tender points and CWP; the 2010 criteria require certain (high) levels of FM symptoms and pain. But many authors provide exceptions to the tender point criterion. Yunus writes of the 1990 criteria, "… one does not need 11 tender points to make a diagnosis of FMS for clinical or patient care purposes. If a patient has the characteristic symptoms of FMS and has as few as 5 or 6 TPs, they may still be diagnosed with FMS."[41] Despite published criteria, primary care physicians often misdiagnose FM in clinical practice, and diagnosis may depend on the skill or beliefs of the physician[42]. If FM is dependent on satisfying criteria for proper diagnosis, improvement in symptoms can result in not meeting criteria. Even FM patients that were diagnosed using ACR criteria may be misclassified in as many as 25% of cases when physicians fail to realize that the patient no longer satisfies criteria[34].

II. FM can also be seen as representing the extreme end of a continuous spectrum of polysymptomatic distress (PSD; FM symptoms or fibromyalgianess) that can be measured on the PSD scale[33]. The further along one is on this continuum, the more FM symptoms are present. A score ≥ 12/31 on the PSD scale derived from the 2010 criteria is the point where FM begins to be identified[33]. In this representation, patients have varying degrees of FM, which can be measured on the PSD scale, and FM cannot always clearly be distinguished from non-FM. For example, a PSD score of 11 (92% of the FM requirement) is not substantially different from a score of 12 (100% of the criteria requirement), except that one patient is criteria-positive and the other is not. The example extends naturally to a patient with 11 tender points compared with one who has fewer than 11 tender points. All of which raise the question of validity of the binary FM concept, even while it may be useful clinically.

III. As patients move in and out of the diagnosis by improvement or worsening of symptoms, and hence being criteria-positive or criteria-negative, the idea of "trait FM" comes up. Katz, *et al* described trait FM as the tendency of those with FM to respond to physical or mental stress in a stereotyped way — by increased pain, fatigue, and the other

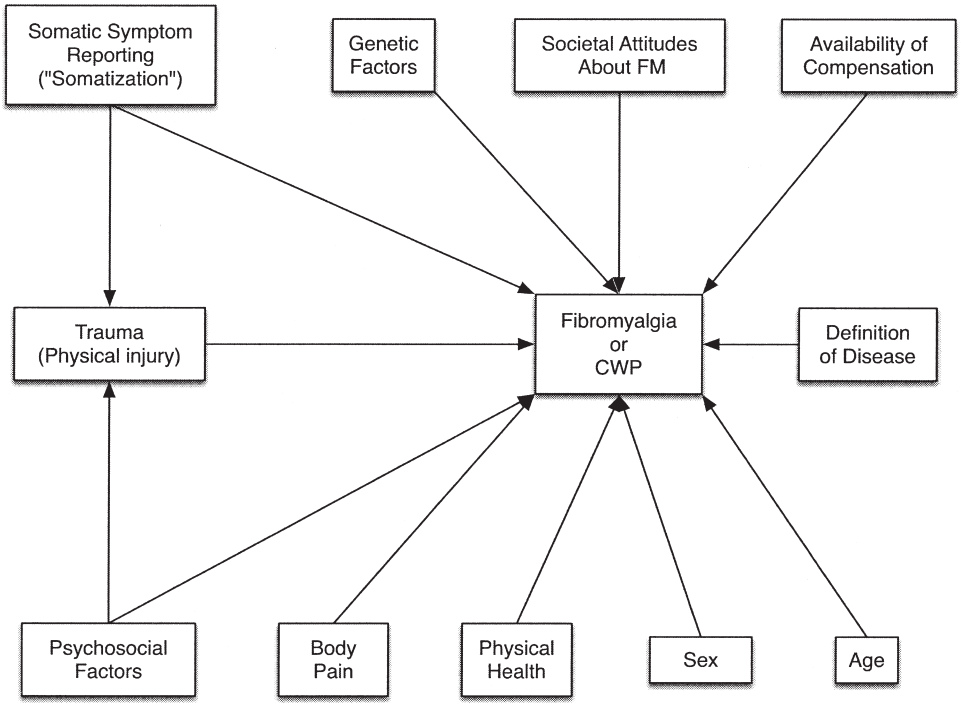

Figure 1. A directed acyclic graph modeling causal paths, emphasizing the trauma-post trauma pathway. FM: fibromyalgia; CWP: chronic widespread pain.

symptoms of FM[43]. "… a diagnosis of FM is most often permanent in the sense that it tends to represent a trait rather than a state. As such, a person may have 'a little' or even no FM for a while and much FM during other periods. For example, FM characteristics may become more prominent during periods of physical and/or mental stress and may relent or decrease during periods of better health or tranquility…. Patients considered to have FM may not meet formal ACR criteria on some or all occasions."[43] The idea of FM as trait accurately describes how it is perceived and treated in the community: a "fibro" patient is one who has now or has had a diagnosis of FM in the past. If the trait assumption is correct in some or all persons with FM, then many patients who are classified as not having FM actually have FM, but are misclassified and will not be diagnosed as FM.

For medicolegal purposes — where it is necessary to know FM status at the level of the individual patient — one would have to review a patient's lifetime medical records to understand whether FM was present in the past. But medical records are problematic. The specialist physician usually has incomplete records because patients move between many different physicians over a lifetime. Physician experts who interview and examine claimants may conclude the patient was "well" before the trauma based on historical data presented by the patient, even while comprehensive medical records that are not available to the expert show symptoms were present previously. Wallace writes of FM and trauma, "In our experience, a review of the medical record would show that 90% of the time, myofascial or FM-associated complaints were present prior to the injury"[44]. At the time that litigation or assignment of responsibility occurs, additional factors may play a role. Patients can emphasize or deemphasize symptoms and history, and physicians come with their own sets of beliefs. This perhaps universal "Rashomon effect" can add further uncertainty to the classification process. If FM exists prior to the trauma, the effect of trauma may be seen to be stronger than it actually is. However, in any causal model, trauma cannot cause FM if FM existed before the trauma.

Exposure

It has been difficult to define which physical trauma(s) might lead to the development of FM. Possible events have included fractures, surgery, various work injuries, MVA, childbirth, and acute illnesses[21,23]. However, no study or report has been able to link severity of trauma to FM development[44]. In the UK study that found some evidence for an MVA-FM association, no support was found for associations with fracture, injury at work, hospitalization, and childbirth[35], but they were unable to measure the severity on the injury during the MVA. While biological gradient (trauma severity-response) is not required to show causation[45], its presence is generally accepted as evidence toward a causal relationship[46].

Confounding factors

A number of confounding factors have been found to be associated with FM development, including some detailed in Figure 1[5,30,31]. In a separate publication, Wynne-Jones, *et al*, writing for the UK group, identified "pre-collision health-seeking behavior (RR 3.6, 95% CI 1.6–7.9), pre-collision somatization (RR 1.7, 95% CI 0.99–2.8), perceived initial injury severity (RR 1.7, 95% CI 0.9–3.3) … and older age (RR 3.3, 95% CI 1.5–7.1)" as independent predictors of new-onset widespread pain[31]. We have also identified the widespread pain index, which is used in the diagnosis of FM[34,35], as a powerful predictor of FM development in rheumatoid arthritis[47]. In addition, FM can even be diagnosed by somatic symptoms reporting. Using somatic symptoms reporting and widespread pain as covariates allows determination of the effect of trauma by removing confounders. However, in clinical care and medicolegal settings, many patients who will "develop" FM following MVA will have high preaccident levels of these variables, raising once again the issue of when FM begins and whether effect may precede cause.

Models

Among FM experts there is usually substantial support for a causal relationship between trauma and FM, although the support is hedged by invoking the idea of a "trigger"[48,49,50]. Yunus identifies trauma as a trigger to central sensitization[41], and Clauw speaks of "triggers," "initiating," and "inciting" events: triggers that include "trauma, emotion; stress, or cessation of aerobic activities. Individuals may be asymptomatic throughout life until an inciting event, but more commonly on questioning there is a lifetime or current history of many of these allied conditions (e.g., [irritable bowel syndrome], migraines, affective disorders, dysmenorrhea, non-bacterial cystitis) even before the 'triggering' event"[51]. This concept is consistent and pervasive in the FM literature. Because "trauma, emotion, and distress" are universal conditions and FM is an uncommon illness, authors argue that trauma "play[s] an etiological [read causal] role in the development of FMS in some, but not all patients"[12,13], an argument that is impossible to falsify; and it represents backward causal reasoning: given that one has seemingly developed FM, how shall we explain what we have seen?

The word "trigger" is often used instead of "cause" when speaking of trauma, perhaps because it implies causation without actually saying it. "Trauma doesn't cause FM, it triggers it" is a common way to put it. Finch's legal review makes it clear that cause and trigger might be the same thing when he writes, "It also appears that most of the doctors who specialize in the treatment of FM believe that trauma can cause this condition"[14]. And Yunus also slips from trigger to causal when he writes, "Trauma, from a motor vehicle accident, for example, is associated with local tissue inflammation and may cause CS [central sensitization] and subsequent FMS"[41]. Bennett's strong affirmation of trauma causation in his deposition ("…that physical trauma can cause the development of FM in some people") attaches practical meaning to the causation debate[4]. Such a conclusion is important for "when courts are satisfied that proponents have presented sufficient proof of general causation, they usually admit testimony that a specific plaintiff has suffered traumatically induced FM ("specific causation" testimony)"[14].

McLean, Williams, and Clauw (MWC) presented their proof for a causal model for trauma as a triggering event (or cause). In a 2005 review, they wrote that "the evidence that MVC trauma may trigger FM meets established criteria for determining causality…"[50]. This quotation was then repeated by others, including Buskila[48]. However, the "established criteria" cited were not criteria to determine causality[52]. Instead, they represented a "first step" unofficial study group committee recommendation of "proposed attribution elements to assess exposures [in order] to publish findings of a possible causal relationship between an environmental exposure and a clinical syndrome."

With time, Yunus's idea that "Trauma, from a motor vehicle accident, for example, is associated with local tissue inflammation and may cause CS and subsequent FMS"[33] has given way to the more inclusive concept of MWC: "MVC trauma appears capable of triggering FM, but generally not through direct biomechanical injury. Instead, the evidence suggests that MVC trauma can act as a 'stressor,' which in concert with other factors, such as an individual's biologic vulnerability, psychosocial factors, cultural factors, and so on, may result in the development of chronic widespread pain and other somatic symptoms"[50]. In paraphrase, it might be said that anything can cause FM. That "trauma acts as a stressor" means that any trauma can trigger FM, and that the extent of trauma need not play a role. And patients report any number of antecedent events that could be considered stressors[21].

The studies of Table 1 were designed to examine the direct effect of trauma on FM development under the original assumption of direct causation. Because ideas about FM held by its proponents have changed somewhat, the arrow from trauma to FM depicted in Figure 1 might be directed into MWC's more inclusive concept: "…other factors, such as an individual's biologic vulnerability, psychosocial factors, cultural factors, and so on"[50]. But those factors are not measureable and are hard even to describe. At least for now, if we want to measure the association between trauma and FM, we have do it directly, as illustrated in Figure 1.

Hill's often-cited causation criteria[46] do not provide much support for trauma-FM causality. The strength of association in all but very low-quality studies is weak. The available data are inconsistent and not specific. The

temporal association is uncertain and may be often in the wrong direction. There is no biological gradient. Experimental evidence offers, at best, weak support. Yes, there is biological plausibility — but "biological plausibility is subject to the prior beliefs of individual researchers"[45]. The data are not coherent, and experimental evidence is weak at best. Only for analogy do the criteria support the FM-trauma association.

Despite weak to nonexistent evidence regarding the causal association of trauma and FM, the literature and court testimony continue to point out the association as if it were a strong and true association. Such assertions influence legal evaluations and beliefs of expert and non-expert physicians, and further the social construction of FM. In 2007, Smith described pseudoevidence-based medicine "as the practice of medicine based on falsehoods that are disseminated as true evidence, then adopted by unwitting and well-intentioned practitioners of evidence-based medicine"[53]. FM recommendations are subtler. They have taken weak truths and added them together to give the impression of an overall association of FM and trauma, while rarely addressing study quality, bias, and strength of effect.

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