

Trauma and Fibromyalgia — Black and White? Or 50 Shades of Grey?



Fibromyalgia (FM) is relatively common, with prevalence estimates between 2.0% and 3.5%^{1,2,3}. Its prevalence increases with age, up to around age 60 to 70 years; FM is roughly 5 times more common in women than in men⁴, and is one of the most common reasons for referral to a rheumatologist⁵. The etiology and pathophysiology of FM are not well determined, and while there are several extant theories — including muscle dysfunction or misuse, central sensitization, sleep disorders, and altered stress axis function — there is little consistent evidence to support, or refute, any of these.

Although FM is often insidious in onset, it is part of the human condition to look for a cause for one's symptoms, and many patients report "trigger" events on which to blame their symptoms. Bennett, *et al* reported results of a large Internet survey of 2569 participants, and presented data on the 13 most commonly reported FM trigger events among questionnaire respondents (most of whom probably had FM, and most of whom probably completed the questionnaire only once, although one cannot be certain)⁶. Chronic stress (reported by 42%) and emotional trauma (31%) were the most frequently reported triggers. (The trigger events were not mutually exclusive.) However, physical trauma was also commonly reported, either due to or not due to, a motor vehicle accident (MVA; 17% and 16%, respectively).

In this issue of *The Journal*, Wolfe and colleagues⁷ review the evidence relating to the (causal) association between physical trauma and FM, and go into some detail about the debate that has persisted — in North America, specifically — over the past 20 years. There are very few studies, worldwide, that examine the relationship between trauma (most commonly MVA-related) and FM. Likewise, only a few other reports examine the relationship between MVA and chronic widespread pain — the cardinal feature of FM. Some are higher quality than others; all are discussed by Dr. Wolfe and colleagues. What is presented is not a structured review; there is no evidence of systematic literature search; and the authors present a very partisan

argument. However, the authors have identified the main articles in the field, and many of their comments are valid.

There are a number of case reports/case series that describe the development of FM following trauma — or, to be correct, that describe the reporting of prior trauma among persons with FM. While interesting on a case-by-case basis (pun very much intended), in the debate about cause and effect these reports, like Bennett's patient-perceived triggers, comprise only slim epidemiological evidence⁶.

The lack of high-quality evidence in this field reflects the complexity of such studies. All studies have difficulties with measurement, in terms of the exposure (trauma), the outcome (FM), and potential confounding variables. In case-control studies, the choice of an appropriate control group is difficult — compounded by the fact that cases, by definition, cannot be identified until at least 3 months after the onset of symptoms. In addition, the exposure of interest is relatively rare and the possibility of recall bias is strong, making this a suboptimal study design. Cohort studies, while more scientifically desirable, also face a number of methodological challenges. As with any rare outcome, one needs enormous sample sizes (and therefore considerable resources) to allow FM to occur in numbers sufficient for meaningful study. The annual incidence of chronic widespread pain in the general population is around 5.8%⁸. With a few assumptions (1:1 ratio of exposed to non-exposed; $\alpha = 0.05$; $\beta = 0.10$; loss to followup of 20%) it is possible to compute that 621 individuals are required who have been exposed to an MVA, plus 621 not exposed, to detect a doubling in the risk of chronic widespread pain onset over a 12-month period.

Wolfe and colleagues mention the medicolegal environment in which the trauma-FM literature is often discussed, and cite staggering statistics about the extent of litigation that occurs: > 5000 cases in US federal and state courts in the past 10 years. One must bear in mind the discordance between the role of epidemiology and the requirements of the legal system. The question for the

See FM and physical trauma, page 1737

epidemiologist is whether, among a group of individuals exposed to an MVA, a greater proportion will develop FM than in a group not similarly exposed. The court, by contrast, is not interested in the group, but attends to the issue of whether a particular trauma is responsible for particular symptoms in a particular individual. In epidemiology, a null hypothesis is proposed (that the proportion of those who develop FM is equal in both groups) and observations are made. Then, if an association is observed, one can estimate the probability of this, if in fact the null hypothesis is true. Or, more usefully, one can calculate the magnitude of any observed association, and estimate this with a certain level of confidence. In either case, information about causation is inferred, and there are often shades of grey.

In the court, however, it must be either black or white; a decision needs to be made. In civil proceedings, the plaintiff will attempt to demonstrate that the defendant's actions, on the balance of probability (sometimes referred to as "preponderance of the evidence"), caused his or her symptoms. Thus, to rule in favor of the plaintiff, the court must be satisfied that there is a greater than 50% chance that the assertion is true. Here, attributable risk is a useful concept (i.e., the proportion of disease in an exposed population that would be avoided, were it possible to completely eliminate the exposure), and where this is $\geq 50\%$, one could surely argue that, on the balance of probabilities, the exposure caused the outcome.

So, do MVA cause FM? Well, clearly, MVA are neither necessary nor sufficient. Also, based on the current best estimates of the association between MVA (and other traumas) and FM, one must conclude that, on the balance of probabilities, they do not. However, the computation of attributable risk (the inverse of the risk ratio associated with exposure) means that any exposure that exerts only a small (risk ratio < 2.0) but genuinely causal effect can never cross the threshold of 50% and therefore can never be deemed to be "causal" by this criterion. This seems unsatisfactory. Also, the ecological fallacy posits that any individual within a group need not behave like the mean of the group to which he or she belongs, and it may be the case (in fact, I would

suggest that it is highly likely) that particular traumas are responsible for particular symptoms, in particular individuals.

Wolfe and colleagues cite an instance from legal testimony in which an expert witness was asked whether there was "any significant dispute ... that physical trauma can cause the development of FM in some people?" There is debate about the relative importance of trauma in the etiology of FM, at the population level. But the counter-argument to the above would suggest that that physical trauma cannot cause the development of FM in any people. This is a position that cannot be easily defended.

GARETH T. JONES, PhD,

Musculoskeletal Research Collaboration (Epidemiology Group),
University of Aberdeen, Polwarth Building,
Foresterhill, Aberdeen, AB25 2ZD, UK

Address correspondence to G. Jones; E-mail: gareth.jones@abdn.ac.uk

REFERENCES

1. Carmona L, Ballina J, Gabriel R, Laffon A. The burden of musculoskeletal diseases in the general population of Spain: results from a national survey. *Ann Rheum Dis* 2001;60:1040-5.
2. Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L. The prevalence and characteristics of fibromyalgia in the general population. *Arthritis Rheum* 1995;38:19-28.
3. White KP, Speechley M, Harth M, Ostbye T. The London Fibromyalgia Epidemiology Study: the prevalence of fibromyalgia syndrome in London, Ontario. *J Rheumatol* 1999;26:1570-6.
4. Wolfe F, Ross K, Anderson J, Russell IJ, Hebert L. The prevalence and characteristics of fibromyalgia in the general population. *Arthritis Rheum* 1995;38:19-28.
5. White KP, Speechley M, Harth M, Ostbye T. Fibromyalgia in rheumatology practice: a survey of Canadian rheumatologists. *J Rheumatol* 1995;22:722-6.
6. Bennett RM, Jones J, Turk DC, Russell IJ, Matallana L. An internet survey of 2,596 people with fibromyalgia. *BMC Musculoskelet Disord* 2007;8:27.
7. Wolfe F, Häuser W, Walitt BT, Katz RS, Rasker JJ, Russell AS. Fibromyalgia and physical trauma: the concepts we invent. *J Rheumatol* 2014;41:1737-45.
8. McBeth J, Macfarlane GJ, Benjamin S, Silman AJ. Features of somatization predict the onset of chronic widespread pain: results of a large population-based study. *Arthritis Rheum* 2001;44:940-6.

J Rheumatol 2014;41:1732-3; doi:10.3899/jrheum.140865