


Reply to Quintner and Cohen

Dear Editor,

I thank Drs. Quintner and Cohen for their interest in my editorial [1] about the study by Albrecht et al. [2]. Drs. Quintner and Cohen have raised several concerns about my discussion of the implications of that study, especially of the concept that peripheral nociception by myofascial trigger points initiates central sensitization. They object to the idea that peripheral nerve sensitization is the fundamental “driver” of, as they put it, all possible somatic mechanisms of pain in fibromyalgia, which they claim is found in the editorial. I agree that this idea is extreme and is incorrect. However, my editorial states only that “peripheral nociceptive sites play an important role in initiating and maintaining pain . . . Pain is the outcome of a complex interplay between . . . central modulation and peripheral input.” However, Albrecht et al. [2] in their article do discuss in detail the implications of their work on some of the many manifestations of fibromyalgia, specifically fatigue, sleep, and cognition, through the potential of reduced blood flow to skeletal muscle caused by shunting blood from muscle to glabrous skin during exercise. The central issue raised by Quintner and Cohen is the role that active peripheral nociception plays in chronic widespread pain. This is a role that they doubt exists.

Quintner and Cohen question the evidence that I presented in the editorial that demonstrates that trigger points are a peripheral nociceptive input leading to central sensitization. They believe that it is a circular argument to state that because inactivation of trigger points in fibromyalgia (and for that matter, in myofascial pain syndrome, as they have included that in their discussion) reduces pain and decreases manifestations of central sensitization, then trigger points (which are peripheral nociceptors) are important in the initiation and maintenance of central sensitization. They state that I presuppose the end (peripheral nociceptors are important in the initiation and maintenance of central sensitization), and therefore my argument does not contribute to the support of any theory.

My argument is linear, to the contrary, not circular. The trigger point can be isolated as a cause of local and referred pain. Pain can be reproduced by trigger point activation. Local and referred pain can be diminished or eliminated by inactivating the trigger point with or without local anesthetic. All these conditions are met in Giamberardino et al.’s studies of trigger points in migraine headache [3]. The pain can be caused again by activating the trigger point. This is more in keeping with Koch’s postulates for a bacterial cause of disease than a tautological, circular argument. In the case of muscle pain, if trigger point activation induces both local pain and referred pain (a manifestation of central sensitization), and inactivation of trigger points reduces local pain and also referred pain, then trigger points can logically be considered related in a causative way to central sensitization. The hypothesis, therefore, is that trigger points are capable of inducing central sensitization and referred pain. To perform experiments that show an effect of trigger point inactivation on central sensitization does not seem to
me to be a circular argument. Quintner and Cohen claim that the conclusion is assumed before the evidence is presented. Rather, the evidence supports the hypothesis. Quintner and Cohen refer to Hans Kraus’ work with vapocoolant spray to claim that there is extant an incorrect theory of a “pain-reflex-pain self-perpetuating cycle interrupted by the vapocoolant.” Simons et al. [4], in their text on myofascial pain, specifically rejected this hypothesis as an explanation for muscle pain from trigger points.

Quintner and Cohen refer to Kellgren’s studies that demonstrated that pain could be referred to distant sites from injections of hypertonic saline into a variety of structures (muscle, ligament, and tendon). They go on to quote Kellgren as saying that the intensity of pain did not diminish when areas of induced pain (referred pain regions) were injected with local anesthetic. If pain originated centrally, or in peripheral nerves unrelated to the tender regions injected, one would not expect pain intensity to diminish. They did not refer to Kellgren’s article in which he reported relief or elimination of referred pain by injection of local anesthetic [5]. Kellgren states that “the full spontaneous pain has been reproduced by manipulating the tender spots and all symptoms then abolished by anaesthetizing these spots locally; in these cases, therefore, it is sound to conclude that the widespread referred pain arose from the tender regions of muscle.” Kellgren reported that the reduction or elimination of pain long outlasted the duration of action of the local anesthetic.

Quintner and Cohen claim that interrater reliability of trigger point identification cannot be demonstrated. However, there are now a number of very well-performed studies that demonstrate excellent interrater reliability in trigger point identification [6–9].

Quintner and Cohen state that referred pain still occurs when all afferent input is interrupted, showing that a peripheral nociceptor is not required to maintain central sensitization. I have no argument with that, as it is well known that central sensitization can be maintained with little or no ongoing peripheral nociceptive input [10]. However, because referred pain is a manifestation of central sensitization, then the modulation of referred pain by activating and inactivating trigger points, the latter both by injection of local anesthetic, and by mechanical means (trigger point compression and dry needling) without anesthetic, must imply that trigger points play a role in establishing and/or maintaining central sensitization.

Quintner and Cohen stated in their 1994 article that the “referred pain” of trigger points can be explained by pain that comes from peripheral nerve or nerve trunk pain because the pain is primarily in the distribution of a peripheral nerve or nerve root [11]. Referred pain from trigger points, like all referred pain from somatic and visceral tissues, is segmental, and primarily in the distribution of the innervating nerve. That means that a trigger point in a C5-innervated muscle will refer pain predominantly to C5-innervated structures. The mechanism of referred pain that arises from muscle through the mechanisms of peripheral and central sensitization is summarized by Graven-Nielsen and Mense [12].

Quintner and Cohen state that there is no demonstrated pathophysiological basis for the existence of trigger points. Mense and Gerwin have produced an elegant body of work elucidating the mechanism of peripheral muscle pain resulting in peripheral sensitization, and central sensitization from their research in animal models, summarized in their text Muscle Pain [13]. Studies over the past decade have imaged trigger points [14,15], have shown that trigger point activation results in CNS activation (fMRI scanning) [16], have demonstrated electrophysiologic activity at the trigger point [17], and have shown biochemical changes in the trigger point zone [18]. Further studies have shown that manipulation of the trigger point modulates muscle function [19], and induces local and referred pain [20–22].

In conclusion, Quintner and Cohen raise serious questions about the nature of muscle pain and its relationship to widespread pain. Their concerns are welcome as an opportunity to review the field of myofascial pain that has grown in the number of studies that demonstrate its nature and importance in musculoskeletal pain today, in contrast to the state of the field in 1994 when they published their article questioning the entire concept of myofascial trigger point pain.

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References


