Response to Letter to the Editor by Dr. Cohen and Dr. Quintner

Dear Editor,

I appreciate the opportunity to respond to the letter to the editor of Drs. Cohen and Quintner (the authors), prompted by the review of the Muscle and Myofascial Pain section of the 2005 IASP Core Curriculum (IASP CC) [1]. The authors are acknowledged for their persistence in challenging inconsistencies in the constructs of the myofascial syndrome (and fibromyalgia). Indeed, and unfortunately, they have offered essentially similar comments to this journal when it was called the Clinical Journal of Pain as early as 1994 [2]. Their position is that the myofascial pain syndrome (MPS) is ill conceived and does not represent a true clinical phenomenon, and that the signs and symptoms attributed to myofascial pain and trigger points (TrPs) are better explained by the concept of regions of secondary hyperalgesia of peripheral nerve origin, which the authors stated in 1994, as a hypothesis that is testable to achieve external validity. Their studies have not been carried out and their theory has not been validated, but many studies have been produced supporting the existence of the mechanisms underlying pain of muscular origin [3,4]. The authors challenge and attribute to Mense the concept of altered end-plate potentials in the pathophysiology of TrPs. As David Simons is the actual author of this theory, he has offered to respond in a separate letter below.

The authors refer to the IASP CC book review of Leano and Kalauokalani [1]. The section alluded to actually says:

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The authors state that Leano and Kalauokalani’s (the authors) position is that the myofascial pain syndrome (MPS) is ill conceived and does not represent a true clinical phenomenon, and that the signs and symptoms attributed to myofascial pain and trigger points (TrPs) are better explained by the concept of regions of secondary hyperalgesia of peripheral nerve origin, which the authors stated in 1994, as a hypothesis that is testable to achieve external validity. Their studies have not been carried out and their theory has not been validated, but many studies have been produced supporting the existence of the mechanisms underlying pain of muscular origin [3,4]. The authors challenge and attribute to Mense the concept of altered end-plate potentials in the pathophysiology of TrPs. As David Simons is the actual author of this theory, he has offered to respond in a separate letter below.

I agree that establishing the level of evidence in cited publications would add to the suggested validity of the MPS construct.

The authors go on to address specific deficiencies in Sections I, V and VI.

A. Understand that the term “myofascial pain” includes a general definition that refers to pain caused by myofascial trigger points. (Emphasis added)

1. Muscle pain and myofascial pain are synonymous; if myofascial trigger points cause myofascial pain—a classic circular argument—then they must cause all muscle pain. It must be noted that myofascial trigger points are, by definition, painful. It follows that a painful phenomenon causes all muscle pain. The tautology is blatant; the circular causative argument is brazen. Not very intellectually satisfying for a student.

This is a valid criticism. The term “myofascial pain” should not be synonymous with “myofascial TrPs.” There are multiple functional muscle pain syndromes; one such example is delayed onset muscle soreness [5,6]. Other functional muscle pain may be present without TrPs [7] and to avoid confusion, the actual designation that should be so stated in the IASP CC for TrPs should be Myofascial Trigger Points.

Section V. Assessment

Know that the lack of a formal, widely accepted, criterion-based diagnostic scheme has proved to be a serious impediment to proper diagnosis, clinical communication, and research.

B. Be able to identify a trigger point and know the common trigger points thought to be responsible for pain. Know that the inter-rater reliability for detecting trigger points is poor in untrained and inexperienced examiners. Know that the reliability, sensitivity, and specificity of trigger points are unknown. (Emphasis added)

After more than 20 years since the Travell & Simons publication [3], there is still no “formal, widely accepted, criterion-based diagnostic scheme”! And why are “the reliability, sensitivity, and specificity of trigger points” still “unknown”? Would a student’s eyebrows not be raised?

Hypotheses are the currency of scientific discourse. The Integrated Hypothesis of Trigger Points is an attempt to coalesce a number of clinical observations into a reasonable hypothesis that can be modified or, with a better proposition, replaced. It is not perfect. It addresses phenomenon associated with primary muscle pain such as taut band and tenderness with typical referral patterns in the absence of measurable muscle action potentials. In addition to the authors’ hypothesis, Rivner [8] summarizes three other hypotheses to compete with the muscle end-plate hyperactivity hypothesis.

The discourse surrounding the nature of TrPs does not provide clear support for all of the elements of Simon’s theory: Although there are recent findings that support the biochemical uniqueness of the TrP [9], there is also recent
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Kuhn in stone of T rP identification and treatment. Thomas conversely, that the taut band phenomenon is a T rPs as they now exist need to be changed or, muscle pain [14], the diagnostic distinctions of in many more patients than have complaints of authors do have a point: if taut bands are found clinical significance. If this is what they mean, the findings in MPS, which they feel also have no clear cing this work, but I assume they are saying that the finding of muscle abnormalities having no obvious clinical significance is similar to muscle findings in MPS, which they feel also have no clear clinical significance. If this is what they mean, the authors do have a point: if taut bands are found in many more patients than have complaints of muscle pain [14], the diagnostic distinctions of T rPs as they now exist need to be changed or, conversely, that the taut band phenomenon is a finding of uncertain meaning and not the cornerstone of T rP identification and treatment. Thomas Kuhn in The Structure of Scientific Revolutions [15] points out that prior to the discovery of oxygen, clinical investigations of the gas surrounding us have produced a multitude of theories to challenge the concept of phlogiston, the substance that the prevailing theory suggested we breathed. With the discovery of oxygen, the competing theories have vanished under the weight of the definitive discove- ery. Phlogiston is a necessary step to provide a theoretical framework upon which one could prove or disprove observable clinical phenomenon. Peptic ulcer disease has relatively recently been found to be caused in part by bacteria. Other causes exist, but the new information has clarified the inconsistencies in the pathophysiological construct and has led to more effective treatment. With new information derived from rigorous preclinical and human studies, we recognize that MPS is a collection of etiologies resulting in the final common pathway of muscle pain.

Although the authors’ wish to clarify the concepts relating to a complex pain problem, one is struck by their wish to ignore all the supportive data for the existence of myofascial pain and T rPs. MPS construct comes out of the recognition that the largest organ system by weight, muscles, needs to be investigated as a possible source of pain in common pain syndromes. The pain treatment community standard of existing models of diagnoses and treatments fails to adequately address the problems of chronic low back, neck, and shoulder pain, leading to needless, costly, and sometimes destructive tests and interventions. The absence of any articles on muscle pain in this journal in many of the years of publication is evidence of the gap in our intellectual curiosity concerning soft tissue pain.

Primary muscle pain, that is, pain originating in muscles related to some form of damage leading to sensitized muscle nociceptors and associated with known referral patterns and absent muscle action potentials, exists and is worthy of further clarification as to its possible pathogenesis and treatment. The absence of a valid diagnostic and treatment algorithm leading to reliable and effective treatment interventions is a clear challenge to the acceptance of MPS in the larger pain treatment community.

The authors therefore could not be suggesting that large-scale biopsies be performed on patients with muscle pain.

It is unclear what the authors mean to say in citing this work, but I assume they are saying that the finding of muscle abnormalities having no obvious clinical significance is similar to muscle findings in MPS, which they feel also have no clear clinical significance. If this is what they mean, the authors do have a point: if taut bands are found in many more patients than have complaints of muscle pain [14], the diagnostic distinctions of T rPs as they now exist need to be changed or, conversely, that the taut band phenomenon is a finding of uncertain meaning and not the cornerstone of T rP identification and treatment. Thomas Kuhn in The Structure of Scientific Revolutions [15] points out that prior to the discovery of oxygen, clinical investigations of the gas surrounding us have produced a multitude of theories to challenge the concept of phlogiston, the substance that the prevailing theory suggested we breathed. With the discovery of oxygen, the competing theories have vanished under the weight of the definitive discov-

The authors refer to the article below [13] presumably pointing out that muscle alterations of unknown significance are common and state out of context that “Heterogeneous myopathic changes are identified in >50% of patients . . .” The article actually reports:

Results: We have described five groups of patients based on muscle biopsy findings: 51.6% had heterogeneous myopathic abnormalities; only 19% of them had a specific myopathic picture, i.e., central nuclei myopathy, central core disease, myopathy with tubular aggregates or with trabecular fibers or abnormalities of fiber typing; 20% had signs of respiratory chain dysfunction but only one patient had a probable mitochondrial disease; 7% had a neurogenic pattern; 2.4% had a metabolic myopathy (phosphorylase or phosphofructokinase deficiency); and 19% had normal muscle biopsy. No clear-cut correlation between muscle biopsy and clinical data was observed except for those patients with a metabolic myopathy. Conclusions: The probability that a patient complaining only of muscle pain and with a normal neurologic examination has a definite muscle pathology is 2%. Only patients with sole exercise-related muscle pain and sCK seven times higher than the normal value are strongly suspected of having a metabolic myopathy. A rigorous selection of patients is needed before performing a muscle biopsy.

References
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