The designation here of stable or unstable refers to movement that occurred from an initial more stable MT2. Thus the state (S-1 displacement from Fig. 1 to Fig. 2). Thus the S-1 body is displaced from Fig. 1 to Fig. 2. This would mean that in Fig. 2 we would find a very discrete olisthesis which there may be some kind of "microinstability," a sign that "Modic Types 1 and 2 are interchangeable and equipo-
transformation in 14.5% of cases, and maintenance of MT1 changes in 48%, but never spontaneous regression.

Kuisma et al. affirmed that Modic changes at L5–S1 and MT1 lesions are more likely to be associated with pain symptoms than other Modic changes or changes located at other lumbar levels. Ohtori et al. stated that endplate abnormalities are related to inflammation and axon growth induced by tumor necrosis factor.

So, we disagree with Dr. Marshman’s conclusion that “Modic Type 1 and 2 are equipotent in symptom-generating capacity, a fact that may more accurately reflect their associated endplate bone marrow changes.” The endplate changes are intimately related with the associated adjacent bone marrow changes. In fact, the endplate changes seem to precede the associated adjacent bone marrow changes. The endplate changes are probably a consequence of the lack of support from the degenerated disc (to the endplates). The intervertebral disc is largely avascular; hence, it depends on nutrition supplied to the endplates and the subchondral bone. The association between the endplates, the subchondral bone, and the intervertebral disc is of paramount importance. When degeneration occurs, natural disc nutrition would be impaired. The adjacent bone marrow would react and display the known MT1 changes, through inflammation and hypervascularization. Thus, endplate changes and associated adjacent bone marrow changes are not distinct things.

Malinin and Brown used nonhuman primates to investigate changes in the vertebral bodies adjacent to acutely narrowed intervertebral discs. By inducing acute disc degeneration, they caused adjacent bone marrow changes.

Their study can be considered an animal model of accelerated disc degeneration. They affirm that disruption of the endplate vasculature by sudden loss of the disc supporting the endplates may explain the pathogenesis of lesions found in their study. Their study confirms the role of the normal nucleus pulposus as a distributor of weight-bearing forces evenly onto the adjacent vertebral bodies. Thus, when there is modification of the normal weight-bearing properties of the nucleus pulposus, weight-bearing forces on the spine become distributed unevenly on the adjacent vertebrae, resulting in microfractures and bone necrosis. The same authors anticipate that, as the result of healing and stabilization of disc function, the lesions in the vertebral bodies may heal. Might this experiment confirm the passage from MT1 to MT2? Unfortunately the authors did not perform sequential MR imaging.

This study clearly demonstrates that the bone lesions adjacent to acutely narrowed discs are due to changes in the biomechanical environment around the vertebral bodies. Thus, the most important cause of acceleration of disc degeneration would be the lack of support to the endplates, but not their integrity. The restoration of support to the endplate would theoretically allow the transition to a more stable state. When the endplate is in an inflammatory, hypervascular state (MT1), the restoration of support would permit the stabilization of the discovevertebral unit and thus the transition to an MT0 state.

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Response: We thank for Dr. Pinheiro-Franco for his interest in our article. However, we are obviously concerned that he “completely disagrees” on “all our assertions.” Our conclusions were straightforward, logical statements that accurately summarized our clinical findings. We described 2 cases in which florid MT2 changes were associated with invariant, disabling chronic low-back pain (CLBP); and in whom florid reverse MT2–MT1 transformation occurred despite invariant CLBP severity.

Because CLBP severity remained invariant during the MT2–MT1 radiological transformation in both our cases, MT2 lesions were therefore associated with as much pain as with MT1 lesions: in other words, they were “equipotent” (our first conclusion).

Modic et al. stated that MT2 lesions were “stable” lesions that did not change with time. In contrast, MT1 lesions were frequently “unstable,” transforming over time into either MT2 or MT0 lesions. Quite clearly, both our cases contradicted this view: MT2 lesions were evidently unstable over time (our second conclusion).

Some of the difficulty may have related to an apparent confusion over the context used for the word “stable.” For example, in the second paragraph of his letter (in which Dr. Pinheiro-Franco appears to contradict himself by appearing to accept our second conclusion), Dr. Pinheiro-Franco conjectures that “the conversion from MT2 to MT1 would be explained by the movement that occurred from an initial more stable MT2.” As we made abundantly clear in our original article, however, the use of the word “stable” related—as in Modic’s original article—to invariance over time: it did not refer to mechanical “microstability.”
Short of doubting the actual veracity of our case reports, however, we are unable to offer any explanation at all as to why Dr. Pinheiro-Franco should disagree with our first conclusion.

We understand that cherished views may prove difficult to reject when conflicting evidence arises. Nevertheless, as clinicians, we have a duty to apply basic scientific principles in our practice. In physical science, just one validated exception is sufficient to invalidate any theory. In biological science, however (where individual “natural” variation prevails) less rigor must frequently be tolerated. It may be the case that MT2 lesions are more generally stable than MT1 lesions over time: this, indeed, would help to account for the observed MT1/MT2 ratio (1:4.9.11; as, indeed, we amply discussed in our article). However, it is clearly erroneous to assume that MT2 lesions are always stable16 (our second conclusion).

Similarly, it may subsequently prove to be the case that MT1 lesions are more frequently associated with painful discs than MT2 lesions.4,7,8 However, MT2 lesions can be associated with just as much pain as MT1 lesions (that is, they are equipotent),1,7,10,14 even in the same patient (our first conclusion). Mitra et al.8 confirmed the findings of Modic et al.9 in that most MT1 lesions evolved into MT2 lesions within 72 months: nevertheless, even MT1 lesions that became more florid during this interval were not associated with significantly worsened pain or disability than those that evolved into MT2 lesions.9 Retrospectively attributing significantly less pain to those patients with MT2 lesions purely by virtue of poor fusion results in that group (and without using discography in all cases—as in Dr. Pinheiro-Franco’s study) is unreliable: variable outcomes could have reflected inconsistent diagnoses.

Dr. Pinheiro-Franco contradicts Modic et al. by asserting that “endplate changes and associated adjacent bone marrow changes are not distinct . . . ” As we pointed out in our article, Modic et al.9 demonstrated identical histological changes in the endplates of both MT1 and MT2 lesions. Indeed, it was only in divergent adjacent bone marrow appearances that each lesion actually did differ.9 In particular, the endplates were equally fissured, and equally associated with vascular granulation tissue, in MT1 and MT2 lesions.9 As we subsequently argued, such endplate changes represent a likely source of de novo pain afferents2,5 and, therefore, of discogenic pain. Divergent adjacent bone marrow appearances may thus be less clinically significant and, in cases of MT2 lesions, may even represent epiphenomena that potentially mask clinically more significant endplate changes.1,9 This may explain why MT1 and MT2 lesions are present in up to 10% of an asymptomatic population.13

The starting axiom for any consistent theory must be the irresistible fact that both MT1 and MT2 lesions can be associated with painful discs.1,7,10,14 By choosing a hyper-vascular “inflammatory” bone marrow response as his starting axiom, however, Dr. Pinheiro-Franco inevitably encounters an impasse: because such a response is singularly exclusive of MT2 lesions, it cannot explain why pure MT2 changes may be associated with pain. His hypothesis is, therefore, logically inconsistent: that is, it permits a contradiction, or an inexplicable fact. Ironically, Dr. Pinheiro-Franco did quote one study10 (in apparent support of his view) that actually included patients with discography-proven MT2-associated discogenic pain: curiously, however, he completely ignored this in his quotation. Similarly, in another quoted study, Dr. Pinheiro-Franco completely failed to highlight one of the key findings: that is, that “both Modic 1 and Modic 2 lesions at L5/S1 were associated with the occurrence and intensity of pain.”14

One way to circumvent the inconsistency of Dr. Pinheiro-Franco’s hypothesis is, therefore, as he appears to have chosen to either completely disregard14 or to fail to equitably quote7,8,10 any study that has demonstrated MT1 and MT2 equipotency. This approach, however, is not recommended. Instead, by choosing the correct starting axiom, a consistent mechanism can be deduced that adequately explains why MT1 and MT2 (and, indeed, the entire gamut of “mixed” MT1/2 lesions1,6,8,9) may prove equipotent. One such mechanism potentially resides in the extent of endplate vascularized granulation tissue present—the only common denominator among all such lesions in the study of Modic et al.9

We fully understand Dr. Pinheiro-Franco’s desire to see his own study quoted on this subject. However, “his” view was amply represented by more original studies that predated his, and which we did quote3,8,11,12 Moreover—and unlike Dr. Pinheiro-Franco, in his letter—we balanced that view against others in which contrary findings were established.1,14 On balance, the published findings support our view that “ . . . MT2 lesions are neither as stable nor as quiescent as originally believed . . . .” It may be solely in the degree to which endplate vascularized granulation tissue (the only common denominator in lesions of all Modic types) might recruit de novo pain afferents, that differences in pain are likely to be explained between patients. Nevertheless, and as we stressed in our original article, more representative radiopathological correlates are required to determine the precise pathological nature of all Modic lesions, since the latter had only been based on 3 patients.9

In summary, Dr. Pinheiro-Franco’s hypothesis is lacking in both novelty and logical consistency. Any novel hypothesis on this subject must (as Dr. Pinheiro-Franco’s hypothesis does not) explain why pure MT2 lesions can be associated with painful discs. Excessive use of Occam’s razor to cut out bona fide evidence merely because it conflicts with a preconceived hypothesis should be avoided.

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