Letter to the Editor

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Core stability and low-back pain: a causal fallacy

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Dear Editor,

We would firstly like to congratulate Seo and Kim on the publication of their research article *"The effects of Gyrotonic expansion system exercise and trunk stability exercise on muscle activity and lumbar stability for the subjects with chronic low-back pain."* However, we find some of the claims made by the authors to be unsubstantiated and reductionist of the complex biopsychosocial phenomenon which is chronic low-back pain. We feel that the methods utilised and the discussion that followed largely did not see the forest for the trees they were looking at.

Firstly, the authors state that back pain refers to "pain around the lumbar vertebrae" and do not use appropriate evidence to support their claim that chronic low-back pain is caused by posture or "imbalanced and weak muscles." Evidence clearly suggests that the majority of the low-back pain presentation is nonspecific, meaning that it is not possible to identify a single origin of the patient's symptoms (Deyo and Weinstein, 2001; Hartvigsen et al., 2018; Koes et al., 2006; Maher et al., 2017). Therefore, stating that posture is the cause of chronic low-back pain is, at best, reductionist and not evidence-based. At worse, it promotes a narrative potentially harmful to patients (Darlow, 2016; Darlow et al., 2013). High-quality evidence suggests a clear lack of association between incidence of low-back pain and awkward postures (Roffey et al., 2010), physical features such as facet joint degeneration (Hartvigsen et al., 2018; Maas et al., 2017) or magnetic resonance imaging changes (Brinjikji et al., 2015; Steffens et al., 2014; Tonosu et al., 2017). It could be argued that the increased rates of structural failure observed *in vitro* models within the "neutral" zone (Gooyers and Callaghan, 2015) support a biomedical approach to low-back pain. However, these changes have been deemed largely unavoidable (Arjmand and Shirazi-Adl, 2005). Furthermore, the validity of cadaveric *in vitro* models to humans is a debate in itself, largely opposed by the high variability and the adaptability of the body. While high loads applied in end-range could potentially lead to structural changes, these changes are rarely directly related to pain (Brinjikji et al., 2015; Steffens et al., 2014; Tonosu et al., 2017). It is more scientifically accurate to think of posture change as a symptom modification (Lehman, 2018) rather than a direct treatment of a disease, of which low back pain is rarely (Deyo and Weinstein, 2001; Waddell, 1987).

The literature supports the premise of the intervention so far as that exercise is beneficial for low back pain (Gordon et al., 2016). However, the claim that core stability exercise is one of the most effective options for low-back pain is without merit and clearly lacks supporting evidence. It has been demonstrated thoroughly that in those with low-back pain there is no long term difference between different exercise modalities including different modes of "core stability" (Michaelson et al., 2016; Saragiotto et al., 2016; Shnayderman and Katz-Leurer, 2013; Smith et al., 2014; Vasseljen et al., 2012; Wang et al., 2012). Perhaps their utilisation may be more effective acutely. However, acute response to physiotherapy treatment is a complex phenomenon, which could be influenced by many nonspecific factors (e.g., therapeutic alliance or patient expectations) and specific factors (e.g., symptom modification or neurophysiological changes) (Bialosky et al., 2009; Bjorbækmo and Mengshoel, 2016; Kinney et al., 2018; O'Keeffe et al., 2016). Moreover, core activation may not even vary between symptomatic and healthy asymptomatic people (Gorbet et al.,

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2010), nor can core stability changes predict improvements in disability following specific exercise (Mannion et al., 2012). Strength training can be beneficial but the larger effect sizes are noted only for full body programmes (Searle et al., 2015), and given that strength training is a complex intervention that has multiple biological and psychosocial benefits, further work is required before we can make any assumptions about cause and effect (Jay et al., 2015; Pedersen and Febbraio, 2008). Therefore, it would appear that the common denominators at present for improvements in the literature were seeking or receiving care for low-back pain, and an increase in activity (Alzahrani et al., 2019). This is hardly surprising when we consider the overall literature suggesting that systemic markers (Klyne et al., 2018), sleep, physical activity, diet (Yang and Haldeman, 2018), depression and mental health (Jarvik et al., 2005; Klyne et al., 2018; Pinheiro et al., 2015) are all partial mediators of development and prognosis. This is confounded further by the comparison of Gyrotonic exercise to another treatment and not including an appropriate control group. As highlighted earlier, there is a wide range of good-quality literature to support the complex and biopsychosocial nature of chronic low-back pain beyond "neutral" posture. Relying on a biomedical paradigm to explain a complex biopsychosocial problem is inadequate (Engel, 1977; Waddell, 1987).

Finally, several of the claims made with regards to the Gyrotonic exercise are unsubstantiated. For example, the statements "these natural movements improve muscle strength and flexibility along with breathing and collaborative muscle contraction" and "the arch and curl motion has been considered to create a connection between the erector spinae and the external oblique" are used to develop a discussion regarding the importance of these exercises for chronic low-back pain. However, the authors failed to provide any credible evidence to support their claims. Only one reference was used to support the first statement (Campbell and Miles, 2006). Upon reading this article, it rapidly becomes apparent that the authors only offer a description of the method, with no evidence or data to support their claims (Campbell and Miles, 2006). As per Hitchen's razor: what can be asserted without evidence can be dismissed without evidence.

In conclusion, we believe that the authors have failed to consider the wider literature and make bold claims without considering the evidence-based biopsychosocial model within their study. As humans, we are all prone to confirmation bias; however, as scientists we have a unique responsibility to display clinical equipoise and follow systematic processes in methodology and reporting to progress our field forward on stable foundations.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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