I recently attended the Pain Mechanisms and Therapeutics Conference, held in Verona (Italy) from the 29th of May to the 3rd of June 2022. The main goal of the conference was to present the most up-to-date basic science evidence, from some of the main research groups involved in the study of pain. Although at a first glance the six-day programme seemed too engrained into basic science to fit the professional scope of a clinical physiotherapist, it offered some extremely enlightening insights to reflect upon, with the potential for immediate clinical translation.

The two main themes; the presence of low-level inflammation in different persistent pain states and its mediation by the immune system, are of significant interest to the whole physiotherapists community. Although it is out of the scope of this report to mention all the relevant pieces of research presented, three are particularly worthy of comment and are discussed in turn. First, a research group from King's College London (UK) led by Prof. Andersson demonstrated how fibromyalgia-related symptoms can be passively transferred from patients to mice by simply injecting rodents with patients' immunoglobulin G. Mice presented features of peripheral nociceptive afferent hypersensitivity, reduced muscular strength and locomotor activity, as well as a loss of intraepidermal innervation. Second, Prof. Loggia from Harvard (USA) presented the work his group has conducted in recent years regarding the presence of neuro-inflammation in different persistent pain states. Interestingly, they have imaged a specific inflammatory biomarker in patients affected by chronic low back pain (CLBP), migraine, lumbar radicular pain, and knee osteoarthritis, showing how its spatial location and density correlates well with symptom location and intensity. Third, and perhaps counterintuitively, Prof. Diatchenko from McGill University (Canada) presented a study on the protective role of an increased inflammatory response for the transition from acute to chronic pain in humans. Interestingly, they crossed this finding with pain trajectories of human subjects reporting acute back pain from the UK Biobank, showing how those taking non-steroidal anti-inflammatory drugs have a seven-fold risk of developing chronic pain compared to individuals taking paracetamol or antidepressants.

I believe all the evidence above is certainly something that we as a profession should reflect upon. It now suggests that discarding and dismissing one's pain experience - as sometimes happens in practice - is wrong not only from a psychological and therapeutic perspective, but also from a biological one. It also poses a big question regarding the role of inflammation in pain. On one

hand, we should become more familiar with the concept that chronic pain in general might be associated with a persistent low level inflammatory response, and perhaps start to think how to encompass this concept into the clinical narrative, bearing in mind that little advances have been made for its treatment. On the other hand, we must be careful not to demonise it and remind ourselves that inflammation has a specific reparative and protective role, aimed at preserving the integrity of the human being.