Sir,

We read with great interest the letter by Dr. Martinez-Lavin in response to our article (Üçeyler et al., 2013). Sympathetic dysfunction has indeed been widely discussed in fibromyalgia syndrome. During a systematic review of the literature undertaken in 2008 for the first German S3-guideline on fibromyalgia syndrome we already identified 29 original articles on this subject, including work by the author; the number has constantly increased in the meantime. The differentiation between diseased autonomic and somatic nerve fibres in the skin of patients with fibromyalgia syndrome should be an important task for future studies.

We would like to point out that our study did not reveal a small fibre neuropathy in patients with fibromyalgia syndrome, but small fibre pathology (Üçeyler et al., 2013). It is essential to make this distinction. The term ‘small fibre neuropathies’ is reserved for a distinct subgroup of sensory neuropathies that has a substantially different clinical presentation from that in fibromyalgia syndrome. Whereas patients with fibromyalgia syndrome suffer from deep and generalized musculoskeletal pain that is regularly associated with additional symptoms like sleep disturbance and fatigue (Wolfe et al., 1990), patients with small fibre neuropathies report superficial acral burning pain that is typically located at toes and feet. Symptom spread over the entire body is possible, but is an exception, and additional symptoms are usually missing (Devigili et al., 2008). Small calibre nerve fibres also have autonomic functions and it is intriguing that autonomic nervous system dysfunction has repeatedly been reported in patients with fibromyalgia syndrome (Martinez-Lavin, 2004). However, dysautonomic symptoms in patients with fibromyalgia syndrome (mostly orthostatic dysregulation, loss of heart rate variability) are also different from those usually reported by patients with small fibre neuropathy (mostly abnormal acral sweating or thermal dysregulation). Thus, this obvious difference in clinical presentation underscores that small fibre pathology in patients with small fibre neuropathies and in patients with fibromyalgia syndrome may emerge from different pathophysiological backgrounds.

In light of the current knowledge, an analogy between findings from recent reports on sodium channel abnormalities in subgroups of patients with small fibre neuropathies (Faber et al., 2012) and patients with fibromyalgia syndrome appears speculative. The hope is that future translational research using robust animal models will decipher the underlying differential pathophysiology of small fibre pathology. This will also help to clarify the currently most burning questions about the classification of pain in fibromyalgia syndrome as neuropathic and of fibromyalgia syndrome as a disease.

References


