Dear Sir,

We read with interest the article by Ling and Bhidayasiri [1] demonstrating the decrease of serum ceruloplasmin (Cp) level in neurodegenerative movement disorders, especially in Parkinson’s disease. In their analysis, the Cp level was normal in 7 patients with idiopathic focal dystonia.

They did not refer to our article [2] published in 2000, where the serum Cp and copper levels were significantly lower in 51 patients with cervical dystonia than those in 39 disease controls. Previous studies have repeatedly demonstrated disturbances of copper metabolism in primary dystonia by transcranial ultrasound [3–5], leukocyte analysis [6], and neurochemical analysis of trace metals and proteins in the brain tissue [7, 8]. The findings were also reproduced in post-surgical secondary dystonia [9], implying the presence of subjects susceptible to dystonia, although the copper gene might be irrelevant by itself to the pathogenesis of dystonia [10].

We agree with the authors’ inference that Cp might be associated with the cascade of neurotoxicity in neurodegenerative movement disorders, and current evidence strongly indicates that this is also the case in dystonia.

Disclosure Statement

Dr. Mezaki has worked as a consultant of GlaxoSmithKline K.K. Dr. Kaji has nothing to disclose and has no conflicts of interest.

References