LETTER TO THE EDITOR

New gene of spinocerebellar ataxia

Hélio A. G. Teive,1 Renato P. Munhoz1 and Tetsuo Ashizawa2

1 Movement Disorder Unit, Neurology Service, Internal Medicine Department, Hospital de Clínicas, Federal University of Paraná, Curitiba, Brazil
2 Department of Neurology, University of Florida, Gainsville, FL, USA

Correspondence to: Hélio A. G. Teive,
Rua General Carneiro 1103/102
Centro Curitiba
Pr. 80060-150, Brazil
E-mail: hagteive@mps.com.br

Sir, We read with great interest the paper published by Wang et al. (2010). In the introduction the authors stated that ‘So far, 31 different genetic loci causative genes have been identified, including trinucleotide and polynucleotide repeat expansions, such as SCA 1-3, 6-8, 12, 17, 31, and DRPLA; and non-repeat mutations, such as SCA5, 11, 13-16, 27, 28 and 16q-linked autosomal dominant cerebellar ataxia’ (Wang et al., 2010).

In fact, although Wang et al. (2010) carefully reviewed the extensive literature regarding spinocerebellar ataxias (SCAs), they did not mention SCA type 10, another autosomal dominant cerebellar ataxia caused by an expanded ATTCT pentanucleotide in intron 9 of the ATXN10 gene, on chromosome 22.q13.3. SCA 10 represents a rare form of SCA, where it accounts for ~42% of autosomal dominant cerebellar ataxia families. Currently 16q-ADCA is termed SCA type 31, which is characterized by adult-onset, pure cerebellar ataxia, due to a pentanucleotide repeat (TAGAA) expansion on the puratrophin-1 (PLEKHG4) gene on chromosome 16q-22.1 (Sakai et al., 2010).

References