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LETTERS TO THE EDITORS

The mirror neuron: thirty years since its discovery

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It is of great importance that we do not let 2022 pass without remembering the 30th anniversary of the discovery of the mirror neuron. It was in 1992 that Giacomo Rizolatti[1] and his team discovered this special type of cell while studying neuronal activation in certain tasks, more specifically, while observing that some kind of neurons were activated when monkeys observed movement. Unfortunately, the study, rejected by *Nature* but published by *Experimental Brain Research*, did not provoke the excitement it warranted in the scientific community.

Fast forwarding 18 years, Mukamel et al.[2] published a study on electrophysiology in deep-brain epilepsy patients, the only one to successfully identify similar properties in those same neurons in human brains. Until then, other studies had focused only on behavioral associations, such as movement imitation, as had been done with animals.[3] A survey of PubMed (Figure 1) shows that until Mukamel et al.,[2] few studies had been published on the same theme.

However, these specific neurons remained relatively unknown to the public. It was only Rizzolatti and Ramachandran (whose work praised the discovery in an Edge.org editorial years after Rizzolati's publication[4]) who defended the crucial importance of mirror neurons in many human social skills. Certain theories were proposed, such as the "broken mirror" hypothesis, that ascribed the difficulty autistic patients have in understanding the thoughts of others to mirror neuron deficiency.[4] Nevertheless, other studies only described behavioral associations, such as the imitation or observation of movement, or used indirect brain visualization techniques, which cannot conclusively demonstrate that mirror neurons exist in humans, which is why so many scientists contest these theories.[4]

However, recent technology has allowed for better visualization of the brain,[5] leading to a shift in opinion among skeptics. Although older studies used electroencephalography to the identify activation of these neurons, with the advent of neuroimaging, specifically transcranial magnetic stimulation and functional magnetic resonance imaging,[3,6] it is now possible to explore other means of visualizing mirror neurons in action. However, the new evidence is still insufficient to resolve dispute about the "broken mirror" hypothesis.[4]

Thus, although the importance of mirror neurons has been considered less than what was initially assumed, and despite recent criticism, they do play an important role in many behaviors. Due to their discovery, the day-to-day lives of many neurological and psychiatric patients have been affected, e.g., the use of virtual reality to activate the mirror neuron system, their use in post-stroke[6] and aphasia treatment,[7] as well as in hand laterality recognition.[8]

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The authors report no conflicts of interest.

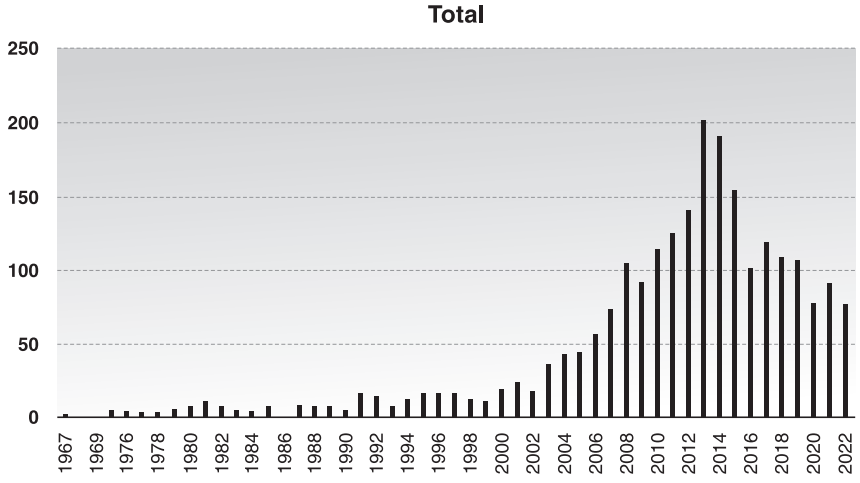

Figure 1 Number of articles on phenomena related to mirror neurons published in PubMed each year until 2022.

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Tourette syndrome and multiple sclerosis: a case report

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Tourettism and tics have rarely been reported in multiple sclerosis (MS).[1-5] Tourette syndrome (TS) is a complex neuropsychiatric disease of uncertain etiology not previously reported in MS.[1-5] Here, we present a patient with TS who was later diagnosed with MS.

In 2013, a 29-year-old male was referred for possible motor neuron disease after developing left arm fasciculations, numbness, and weakness for 5 years. The patient agreed to the discussion and publication of this case, and his anonymity was guaranteed. Electromyography revealed restricted fasciculations and re-innervation in the left arm, with normal nerve conduction studies. He was diagnosed with TS: multiple motor and vocal tics (head turning, blinking, throat clearing, humming), coprolalia, and obsessive compulsive disorder. His neurological exam revealed left proximal arm paresis (4/5) with proximal fasciculations, decreased left biceps and triceps reflexes, atrophy, and no sensory deficits. He was started on risperidone for tics and obsessive compulsive disorder, with good response. Tics began at 10 years of age and progressed over the years. His maternal grandfather had tics and a history suggestive of TS.

Cervical spine magnetic resonance imaging revealed a cervical demyelinating lesion (Figure 1A).

The fasciculations and left arm weakness improved over the next months. In 2016, he experienced bilateral leg weakness. Brain magnetic resonance imaging revealed slowly progressive subcortical white matter lesions (including brainstem plaques), characterizing dissemination in time and space (Figures 1B and C). The cerebrospinal fluid protein and cell count were normal, with no oligoclonal bands. Anti-aquaporin 4, ANA, P and C-ANCA, RF, anti-SSA and SSB, anti-cardiolipin, and lupus anticoagulant were negative. The patient was diagnosed with MS and started on glatiramer acetate (Copaxone©) sc 20 mg/day in December 2017. Risperidone (1 mg/day) and clonazepam (1 mg/day) were continued to control tics and obsessive compulsive disorder. In 2019, he developed insomnia, worsening tics, irritability, and depression, and was started on sertraline 25 mg/day and an increased dose of risperidone (1 mg BID). As of his last outpatient visit to our clinic (January 2023), no further MS relapses have been reported after glatiramer treatment began. Glatiramer had no effect on TS symptoms, which were well controlled with risperidone, clonazepam, and sertraline.

To our knowledge, this is the first case to simultaneously fulfill all diagnostic criteria for TS in the DSM 5/ICD-10 and the latest McDonald MS criteria. Tics have been rarely reported in MS and, among the 5 reported cases of tics and/or tourettism (features of TS that do not meet all the criteria), the movement disorders were considered secondary to MS disease activity, i.e., MS plaques affecting the basal ganglia.[1-5] Our patient had no basal ganglia demyelinating plaques. Tics and tourettism in MS have been attributed to involvement of the cortico-striatal-thalamocortical circuit and/or basal ganglia.[6]

The phenomenology underlying the 5 other reported MS cases with tics was quite variable, but only one mention of tourettism was made, coincidentally in a Brazilian patient.[4] Both cases of MS patients with tics were reported in the USA,[2,3] and these patients had serious behavioral disorders. One of them was diagnosed with Asperger syndrome, pedophilia, and seizures[3]; the tics were the least striking symptom. The other had serious issues with criminal behavior and substance abuse.[2] The 2 patients from Italy had secondary progressive MS.[1,5] Finally, the Brazilian patient with Tourettism had phonic tics following diagnosis of MS.[4]

In summary, our patient is the first report of an association between TS and MS. No basal ganglia plaques were identified. Further studies are necessary to determine whether this association is coincidental or due to a common genetic and/or (less likely) immunological basis. In classic autoimmune disorders, there is ample evidence of genetic susceptibility – at least in part – related to human leukocyte antigen subtypes. However, in TS, there is evidence that both autoimmune and genetic factors may play a significant role, indicating the need for more elaborate and collaborative studies in this field.