The presence of anti-CV2 antibodies was shown using immunohistochemistry on sections of rat brain and on western blots of a variable fraction of newborn rat brain proteins, as previously reported (figure, B). The identity of the antibody was confirmed by immunoprecipitation of the CV2 protein. 2 However, it is not clear why the authors used an immunohistochemistry assay rather than another method. The reason could be related to the tissue specificity of the antigens used for immunohistochemistry.

Our finding is remarkable because it suggests that anti-CV2 antibodies may be involved in paraneoplastic encephalomyelitis-sensory neuropathy, a condition that is characterized by pain, sensitivity to touch, and autonomic dysfunction. The antibody was found in a patient with a history of paraneoplastic encephalitis and in another patient with a history of paraneoplastic encephalomyelitis. The authors hypothesize that anti-CV2 antibodies may be involved in the pathogenesis of these conditions.

Anti-Hu antibodies have been associated with several neurological disorders, including paraneoplastic encephalitis and sensory neuropathy. The presence of anti-Hu antibodies in the serum of the patient with paraneoplastic encephalitis suggests that the antibody may be involved in the pathogenesis of this condition.

The authors also report the presence of anti-Ri antibodies in the serum of the patient with paraneoplastic encephalitis. Anti-Ri antibodies have been associated with paraneoplastic syndromes, including opsoclonus-myoclonus syndrome, paraneoplastic encephalomyelitis, and sensory neuropathy. The presence of anti-Ri antibodies in the serum of the patient with paraneoplastic encephalitis suggests that the antibody may be involved in the pathogenesis of this condition.

In summary, the presence of anti-CV2 antibodies in the serum of the patient with paraneoplastic encephalitis and in the serum of the patient with paraneoplastic encephalomyelitis suggests that the antibody may be involved in the pathogenesis of these conditions. Further studies are needed to confirm these findings and to understand the role of anti-CV2 antibodies in paraneoplastic encephalomyelitis.
Increase of flexor reflex latency in patients with amyotrophic lateral sclerosis treated with riluzole

Recently, riluzole has been reported to increase life expectancy in patients with amyotrophic lateral sclerosis. Pharmacologically, riluzole is an inhibitor of glutamate release and a non-competitive antagonist at N-methyl-D-aspartate receptors. Glutamate antagonists are also undergoing clinical trials for several other diseases. It is known that the H reflex and flexor reflexes in experimental animals are mediated by different subtypes of ionotropic glutamate receptors; the flexor reflex by NMDA receptors and the H reflex by non-NMDA receptors. Therefore, spinal reflexes may provide an opportunity to investigate glutamatergic neurotransmission in humans in vivo. We investigated whether riluzole differentially alters the H reflex and flexor reflexes in patients with amyotrophic lateral sclerosis treated with the drug. From the natural course of the disease it is known that the density of the non-NMDA binding sites increases in the spinal cords of patients with amyotrophic lateral sclerosis. We therefore expected that treatment with riluzole would maintain the latency of the H reflex and increase the latency of the flexor reflexes.

The study was approved by the Ethikkommission of the Humboldt-University.

The H reflex (recorded from M soleus) and the flexor reflexes from M tibialis anterior were investigated in 15 controls (nine men, six women, mean age 25 years) and 10 patients with amyotrophic lateral sclerosis, at onset and after three months of treatment with 50 mg riluzole daily, twice a day (four men, six women, age range 35-75 years; less than 30 months since the onset of clinical symptoms). The H reflex was investigated in a sitting position with stimulation of the nerve in the popliteal fossa. The flexor reflex was elicited in a sitting position with the foot mildly dorsally flexed. Stimulation was performed at the plantar aspect of the foot (20 ms duration, 50 Hz, 50 mA). The stimulus was recognised as a sharp burning pain but was tolerated by all patients and controls. The differential electrode was placed 10 cm below the patella ligament, the indifferent 3 cm distal over the tibial bone. In controls, the mean latency of the H reflex was 29.2 (SD 2.0) ms and of the flexor reflex 80.1 (SD 7.1) ms. At the onset of treatment with riluzole, the mean latency for the H reflex in patients with amyotrophic lateral sclerosis was 31.0 (SD 3.3) ms. After three months of treatment with riluzole, the latency was unchanged at 30.4 (SD 3.0) ms.

The latencies of the H reflex in controls and untreated patients with amyotrophic lateral sclerosis are consistent with the medical literature. The latency and pattern of the flexor reflex is similar to the report from which the method was adapted. Our results show that the H reflex and the flexor reflex are differentially affected in patients with amyotrophic lateral sclerosis treated with riluzole. The latency of the H reflex did not change in patients treated with riluzole, whereas the latency of the flexor reflex increased. This is consistent with the known pharmacological properties of riluzole as a non-competitive antagonist at NMDA receptors.

We conclude that spinal reflexes can be used to investigate the differential modulation of glutamatergic neurotransmission in patients with amyotrophic lateral sclerosis treated with riluzole.

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