

## **New Advances in Amyotrophic Lateral Sclerosis**

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New Therapies in Amyotrophic Lateral Sclerosis can be considered a progressive, degenerative and inexorable disease, which progresses with the depletion of neurons in the anterior tip of the spinal cord and the pyramidal bundle. It is undoubtedly one of the neurological diseases with the worst prognosis still lacking therapies with "significant" modifying potential in natural history in clinical evolution. In addition to muscle weakness (paresis) and amyotrophy, patients have dyspnea (shortness of breath), dysphagia (difficulties in swallowing), dysarthria (difficulties in articulating words) and dysphonia (changes in the timbre and intensity of the voice).

After riluzole, numerous drugs have been used based on changes in the pathophysiological framework of the disease, such as: methylcobalamin (intra-muscular), l-serine, edaravone (venous use), tauroursodeoxycholic acid, naltrexone, among others. In some patients who have one of the familiar forms of the disease, described as SOD1, new studies have already identified ways to reverse or prevent the formation of aggregates (Trimer Study). Unfortunately, the results do not apply to other mutations.

Many patients question us about stem cell therapy. The recognition of pluripotent cells, those that allow the genome of injured patients to be captured at the cellular stage, seems to be a good signal. Such cell lines are already being used *in vitro* and obviously, gene therapy in the testing phase. We hope that the "injection" of these cells will lead to improvements in the remaining motor neurons and, consequently, promote some protection to the surrounding neuronal population.

Another condition is gene therapy. I highlight three types of therapy: the insertion of naked DNA, liposomes or viral vectors; the latter being the most debated and with the best potential for ALS patients. Experimental research has signaled that viruses of the "lentivirus" class can carry sequences from human SOD1 (intramuscular injection) to neurons in the spinal cord and the pyramidal pathway through retrograde transport. Some steps still need to be understood to direct the gene treatment exclusively to the population of motor neurons. It is not a risk-free form of treatment, therefore, we must act with parsimony.

The transdisciplinary team of doctors, physiotherapists, nurses, speech therapists, occupational therapists, psychologists and other health professionals is imperative. Together and exchanging knowledge, we can improve the quality of life of these patients.

## **Conflicts of Interest**

The authors declare no conflict of interest.

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