FOR IMMEDIATE RELEASE

Press Contact:
5W Public Relations
Jennifer Fiorenza
Amy Mitchell
Tel: 212.999.5585

Antioxidant Shows Promise in Supporting Treatment for Lou Gehrig's Disease

(Montevideo, Uruguay) – Researchers at The Center of Free Radical and Biomedical Research at Facultad de Medicina, Universidad de la República in Uruguay today announced the discovery that the antioxidant MitoQ prolonged the lifespan and health-span of mice suffering from amyotrophic lateral sclerosis-like (ALS) disease, and successfully improved functional parameters related to muscular strength and reversed mitochondrial damage in nervous and muscle tissue. The study was published in the journal *Free Radical Biology and Medicine* and the findings of this study point to a positive direction for those suffering from ALS.

MitoQ is a mitochondria-targeted antioxidant created over a decade ago that reduces mitochondrial oxidative stress and, when administered to a mouse model of ALS, delayed the progression of the fatal neurodegenerative disease. Mitochondria are also known as cellular batteries and provide energy to cells.

Commonly known as Lou Gehrig's or Motor Neuron disease, ALS is a disorder within the nerve cells of the brain and spinal cord that control voluntary muscle movement, and ultimately results in progressive paralysis and death. The results of this study show a promise that mitochondria-targeted antioxidants could potentially be of use for the treatment of ALS, a condition for which there is currently no cure.

"This study supports the role of mitochondrial dysfunction in the development and progression of ALS, which may allow for the development of more mitochondria-targeted therapies to fight this disease," said Rafael Radi, MD, PhD, Director of the Center and a key member of the study's research team. "We also found that MitoQ has beneficial effects in the murine model of ALS, which will likely lead to clinical trials using MitoQ with ALS patients and hopefully lead to extend the survival and improve the quality of life of ALS patients."

Researchers sought to decrease oxidative stress in mitochondria as a way to slow the death of motor neurons and disease progression within the ALS-induced mice. MitoQ works by accumulating inside the mitochondria and performing antioxidant activity, while allowing the molecule to act specifically at the site of free radical formation in the cells and efficiently reduce oxidative stress.

To conduct their study, scientists administered MitoQ to the mice's drinking water from a time when early symptoms of neurodegeneration had become evident. After 20 days of

administration, MitoQ was detected in all tested tissues and the treatment had slowed the decline of mitochondrial function in both the spinal cord and the quadriceps muscles.

"MitoQ increased approximately 6% the survival of the treated mice," noted Patricia Cassina, MD, PhD, another key senior member of the research team. "This might seem small, but it's in the range of the best that other drugs have been able to achieve with this model in which the disease progression is extremely fast and there is a small window of opportunity to improve symptoms and survival." Radi noted that MitoQ administration also resulted in improved muscular strength as evaluated by functional tests performed in the animals.

An advantage of MitoQ is that it has been tested for safety in a variety of clinical human trials since its inception in the late 1990s. The Center of Free Radical and Biomedical Research is now evaluating the possibility of conducting clinical human trials specifically related to MitoQ and its effects on ALS patients.

###