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# Peptide IPL344, an AKT activator, treats SOD1 ALS model mice to improve neuromuscular function

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**Abstract:**
ALS in humans and mouse models features lowered levels of AKT in motor neurons and skeletal muscles (1-3). A synthetic peptide of 7 amino acids IPL344 (Stressin) was found to activate AKT and inhibit apoptosis induced in different ways in various cell cultures (4). Here we report that IPL344 administered therapeutically IV and IP to SOD1G93A transgenic mice at disease onset, detected by weight loss according to guidelines (5), led to clinical benefits: enhanced survival, arrested weight loss, and neurologic preservation (measured by limb function). Previous experiments determined dose and site of administration in a total of 100 mice. The concluding experiment was done with two groups of 21 mice each, supported statistically (6); 21 mice were vehicle-treated controls and 21 test mice were given 200 micrograms IP and 200 micrograms IV daily to individual mice at the onset of their weight loss. The results are shown in Figure 1: Median survival was longer by 9 days in the treated mice compared to controls (P=0.02); loss of neurologic function over days 107-119 was 40% in controls and 12% in treated mice (p=0.001); and weight loss on days 114-125 was 11% and 4%, respectively (p=0.01). Thus, IPL344 administered after the onset of clinical disease benefited the course of the disease. In view of these positive benefits in an ALS animal model, we have initiated daily IV treatments of human ALS patients.

**Biography:**
Ilana Cohen earned her Ph.D. in neurobiology at the Weizmann Institute of Science, Israel. She has more than 20 years of experience in scientific management of drug development, leading multidisciplinary projects from initial drug discovery stages to clinical trials. As part of Immunity Pharma (IPL) management her main goal is helping people living with neurodegenerating disorders by developing innovative therapies. She leads the development of Immunity Pharma's novel technology platform that focuses on receptor-independent activation of the pro-survival Akt pathway, a key pathway in preventing neurodegeneration. IPL's first drug candidate targets Amyotrophic Lateral sclerosis (ALS) is currently evaluated in exploratory open label Phase 2a clinical trial with ALS patients.

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