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Double-blind, placebo-controlled, clinical study to investigate the safety and efficacy of MLC 601 in patients after stroke

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Stroke is the leading cause of severe neurological disability and results in enormous cost measured in health care and lost productivity. To date, no effective treatment has been found for reducing stroke-induced disability. MLC 601 (NeuroAid) as a Traditional Chinese Medicine has been developed to aid post-stroke recovery. This is a double-blind, placebo-controlled clinical trial study on 150 patients with a recent (less than 3 month) ischemic stroke. All patients were given either MLC 601 (100 patients) or placebo (50 patients), 4 capsules 3 times a day, as an add-on to standard medication of post stroke for 3 months. Baseline characteristics for gender, age and elapsed time from stroke onset and risk factors were not significantly different between two groups ($p>0.05$). There were no difference in Fugl-Meyer Assessment (FMA) score at baseline; 53.69±23.01 in the MLC 601 and 54.96±24.27 in the control group, $p=0.755$. FMA scores increases significantly in MLC 601 comparing to controls in 4th week (77.13±19.22 vs. 63.50±24.21; $p<0.001$), 8th week (82.51±14.27 vs. 72.06±21.41; $p=0.001$) and 12th week (86.22±12.34 vs. 82.78±14.93; $p<0.001$) after medication. Repeated measured analysis showed statistically difference in FMA during 12 months between two groups ($p<0.001$). Patients showed a good tolerability to treatment and adverse events were mild and transient. MLC 601 showed better motor recovery than placebo and was safe on top of standard ischemic stroke medication. It was more effective in motor recovery in subjects with severe and moderate than mild patients. However, still more clinical trials are needed to evaluate safety and efficacy of MLC 601 for stroke recovery.

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PERK inhibition reduces hyperphosphorylated tau and rescues neuronal function in an eIF2α-independent mechanism

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Tauopathies are a group of more than twenty known debilitating neurodegenerative disorders that affect nearly eight million people in the United States. Currently, there is no cure for tauopathies, and there are temporary and limited benefits to current therapeutic strategies. The endoplasmic reticulum (ER) stress sensor PERK (protein kinase R-like ER kinase) has been identified as a participant in the pathogenesis and progression of tauopathies. However, the mechanism by which the PERK pathway causes neuronal dysfunction is still unknown. In this study, we treated rTg4510 tau transgenic mice at a stage when tau pathology is rampant and cognitive function is impaired with a novel and potent PERK inhibitor. The treatment significantly reduced hyperphosphorylated tau species and led to improvement of neuronal function, as determined with a sensitive and innovative imaging technique called manganese-enhanced magnetic resonance imaging (MEMRI) with quantitative R1 mapping. We also found that PERK inhibition mediated these improvements via a pathway that is independent of eIF2α. Our results show a novel mechanism of PERK-mediated tau phosphorylation that potentiates pathogenesis and progression of tau pathology. Future efforts aim to delineate the mechanism ruling the tau-PERK relationship. Finally, this study suggests that PERK is a viable therapeutic target to ameliorate neuronal function in tauopathies.

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