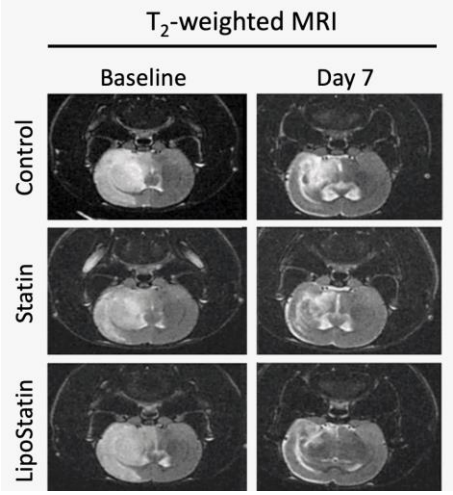
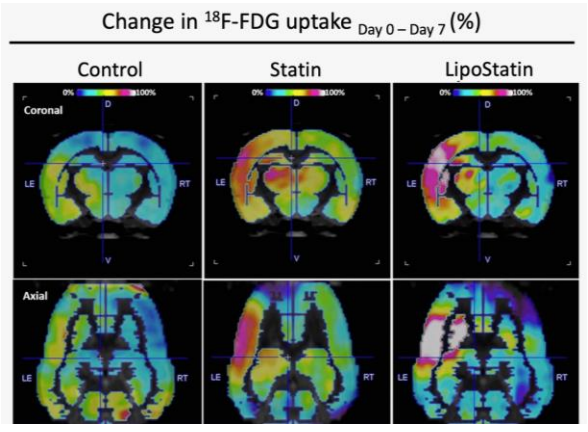
Treatment of Ischemic Stroke by Atorvastatin-loaded PEGylated Liposome

Stroke is a leading cause of death and disability worldwide. According to the World Health Organization, approximately 15 million people suffer from a stroke each year, and of those, 5 million die and another 5 million are left permanently disabled. Stroke is the second leading cause of death globally, and a leading cause of adult disability. Additionally, the incidence of stroke is projected to increase in the coming years due to aging populations and increasing rates of risk factors such as hypertension and obesity.

Despite advances in medical management and neurosurgical techniques, there are currently no effective neuroprotective agents for improving outcomes after ischemic stroke. The use of nanoparticles, specifically liposomes, as a neuroprotective strategy for treating ischemia-reperfusion injury has been proposed as a potential solution. Liposomes, which are biocompatible and biodegradable, can be used to deliver therapeutics across the blood-brain barrier to increase drug accumulation at diseased sites. Statins, specifically atorvastatin, have been found to be effective in treating cardiovascular diseases and have potential as a neuroprotective agent.

In a recent paper entitled "Treatment of Ischemic Stroke by Atorvastatin-loaded PEGylated Liposome" researchers at Chonnam National University Medical School and Hwasun Hospital demonstrated that PEGylated liposomes loaded with atorvastatin (LipoStatin) efficiently accumulated at the site of cerebral ischemic injury. Images obtained using the Aspect Imaging M7 preclinical MRI, and the Sedecal SuperArgus preclinical PET/CT showed reduced infarct volume (anatomical MRI), improved neurological function recovery, and improved brain metabolism, as demonstrated by a significantly increased uptake of the clinically-used radiotracer ^{18}F-Fluoro-deoxyglucose (FDG). *Ex vivo* examination of the rate brain at study endpoint showed that treatment with lipoStatin led to significant anti-inflammatory effects and recovery of blood-brain barrier breakdown and endothelial dysfunction, as demonstrated by reduced extravasation of evans blue measured by *ex vivo* fluorescence imaging system, like the Vilber Newton bioluminescence and fluorescence system.





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