A growing body of experimental evidence indicates that the plasminogen activating system plays a pivotal role in neuronal function and dysfunction. Our recent work indicates that the interaction of urokinase-type plasminogen activator with its receptor (uPAR) promotes neurorepair and functional improvement following an ischemic stroke. We show that uPA binding to uPAR activates an intracellular cell signaling pathway by a mechanism independent of its ability to catalyze the conversion of plasminogen into plasmin, and instead mediated by the low-density lipoprotein receptor-related protein 1 (LRP-1). More importantly, we found that treatment with recombinant uPA (ruPA) may be an effective therapeutic tool to promote neurological recovery in ischemic stroke survivors.

Walters Life Sciences Bldg M311
3:15 refreshments
3:35-4:55 seminar