Future directions overview

My findings opened up several avenues for additional research. First, aninvestigation of the role of silent synapses (see below) in cocaine-associated memorystrength will help to identify a mechanism through which NR2B-containing NMDARscontribute to Fix-C and Esc-C memory. Second, site-specific inhibition of signalingtargets in the hippocampus and other brain regions associated with drug addiction willhelp to further define the roles of different signaling pathway in Fix-C and Esc-Cmemory acquisition, reconsolidation and reinstatement. Third, since NR2B antagonismwas effective against acquisition and reconsolidation of Fix-C and Esc-C memory butineffective against stress-induced reinstatement, future investigations into thephysiological changes that occur during withdrawal with respect to NR2B expressioncould shed light on inherent differences between the processes of acquisition andreconsolidation versus stress-induced reinstatement.

Fourth, since my work has notidentified a signaling pathway that effectively attenuates stress-induced reinstatement ofEsc-C CPP, future studies should probe for the contribution of alternate signalingpathways such as metabotropic glutamate receptor signaling. Fifth, my studies haveshown that varying the stimulus salience of cocaine reward through changes in thepattern of drug administration results in differential drug-memory strength and therecruitment of different signaling pathways. Future studies should investigate whethervarying the stimulus salience of an aversive memory such as fear conditioning willsimilarly engage different signaling molecules.