Silent synapses in cocaine-associated memory strength

ASSIGN BUSTER

Recent reports demonstrate that repeated cocaine exposure generate silentglutamatergic synapses which contribute to metaplasticity (Huang et al, 2009) and thedevelopment of cocaine-induced locomotor sensitization (Brown et al, 2011). Silentsynapses are glutamatergic synapses which express NMDAR-mediated currents in theabsence of AMPAR-mediated currents (Isaac et al, 1995; Liao et al, 1995). Interestingly, in vitro evidence supports the idea that silent glutamatergic synapses contain higherlevels of NR2B-containing NMDARs compared to neighboring active synapses.

Moreover, the authors showed that elevated levels of NR2B at silenced synapsesincreased calcium entry into neurons and lowered the threshold for LTP induction (Lee etal, 2010). This suggests that silent synapses are capable of undergoing rapidmetaplasticity to strengthen synaptic connections. Given these observations, it isreasonable to assume that my cocaine administration schedules generated silent synapsesillustrated by the increases in NR2B protein.

Furthermore, it is plausible that the extent towhich silent synapses were generated contributed to the differential salience of cocainereward elicited by Fix-C and Esc-C. Thus, increased numbers of silent synapses throughenhanced expression of NR2B provide greater potential for metaplasticity and subsequentmemory strengthening. Further research should use electrophysiological tools toinvestigate the extent to which silent synapses were generated and how they may havecontributed to the observed behavioral effects.