



Supplementary Fig 2. Effect of α -conotoxin MII on nicotinic responses mediated by dissociated VTA DAergic neurons. **A:** Typical traces of nicotinic responses of type ID, IID and IIID neurons showing no inhibitory effect of 100 nM α -conotoxin MII (pre-treated for 2 min). The same results were observed in 3-5 neurons tested for each DAergic neuronal type (ID, IID, IIID). **B:** Under the same conditions for whole-cell current recording, but using VTA DAergic neurons isolated by purely mechanical dissociation to preserve presynaptic boutons apposed to DAergic neuronal soma. Exposure to 100 nM α -conotoxin MII abolished the increases in frequency of spontaneous inhibitory postsynaptic currents (sIPSCs) elicited in the presence of 1 mM acetylcholine and apparently mediated by a toxin-sensitive nAChR on presynaptic boutons, but not postsynaptic whole-cell current responses mediated by somatodendritic nAChR on the DAergic neurons. Therefore, perforated-patch recordings of acutely-dissociated DAergic neurons from the VTA of Wistar rats are not reconciled with findings by Klink et al. (2001) using slice-patch recordings that α -conotoxin MII partially suppressed nicotinic responses of DAergic neurons, but we do demonstrate biological activity of α -conotoxin MII via its ability to block sIPSCs evident on DAergic neurons.