

LIST OF FIGURES

Chapter 1

- Figure 1. Trichostatin A increases acetylation of p55 in the hippocampus
- Figure 2A. p55 and the tumor-suppressor, p53, are two different proteins
- Figure 2B. p55 and alpha tubulin resolve at the same position
- Figure 3. p55 is a non-nuclear protein
- Figure 4. p55 is associated with polymerized microtubule fraction
- Figure 5. Trichostatin A, not sodium butyrate, increases acetylation of p55
- Figure 6. p55 and alpha tubulin have identical mobility on two-dimensional gel
- Figure 7. Acp55K of insular cortex is likely alpha tubulin
- Figure 8. KCl increases acetylation of α -tubulin
- Figure 9A. NMDA increases acetylation of α -tubulin
- Figure 9B. APV blocks NMDA-induced increase in α -tubulin acetylation
- Figure 10A. Endogenous glutamate release increases acetylation of α -tubulin
- Figure 10B. APV blocks BNG-induced increase in α -tubulin acetylation
- Figure 11A. Roscovitine blocks KCl-induced increase in α -tubulin acetylation
- Figure 11B. Olomucine blocks KCl-induced increase in α -tubulin acetylation
- Figure 12. Roscovitine blocks NMDA-induced increase in α -tubulin acetylation
- Figure 13. KCl reduces sirtuin2 activity
- Figure 14. Roscovitine inhibits KCl-induced reduction in sirtuin2 activity
- Figure 15. Habituation in the arena
- Figure 16. Interaction time with objects used for novel object recognition task
- Figure 17A. Equal exploration of the objects during training
- Figure 17B. Short-term memory (STM) for the object

- Figure 17C. Long-term memory (LTM) for the object
- Figure 18. Memory training enhances acetylation of alpha tubulin in the hippocampus
- Figure 19. Memory training does not affect acetylation of alpha tubulin in the insular cortex
- Figure 20. A schematic depiction of regulation of alpha tubulin acetylation, and its possible role in LTP and memory

Chapter 2

- Figure 1A. Trichostatin A increases surface expression of GluR1
- Figure 1B. Trichostatin A-induced increase in surface GluR1 level in neurites
- Figure 1C. Trichostatin A increases surface expression of GluR1 without affecting total GluR1 level
- Figure 2A. Trichostatin A increases surface expression of GluR1 at synaptic sites
- Figure 3. Trichostatin A increases phosphorylation of GluR1
- Figure 4. Trichostatin A increases phosphorylation of CaMKII
- Figure 5A. Tubacin increases acetylation of alpha tubulin and surface GluR1 expression
- Figure 5B. Tubacin-induced increase in surface GluR1 expression in neurites
- Figure 5C. Tubacin increases surface level of GluR1 without affecting total GluR1 level
- Figure 6. Acetylation of alpha tubulin increases its association with GluR1
- Figure 7A. KCl increases surface GluR1 expression in the neurites
- Figure 7B. KCl increases GluR1 on the membrane surface without affecting total GluR1 level
- Figure 8A. Roscovitine blocks KCl-induced increase in surface GluR1 level
- Figure 8B. Roscovitine blocks KCl-induced increase in surface GluR1 level, but does not affect total GluR1 level

Chapter 3

- Figure 1. Schematic depiction of the 5-day and 1-day spaced training paradigms in Morris water maze task.
- Figure 2A. Performance of 1-day spaced-trained and 5-day spaced-trained animals in the spatial memory task
- Figure 2B. Both 5-day and 1-day spaced-trained animals show long-term memory (LTM) for the platform
- Figure 3. Schematic depiction of single-day spaced and massed training paradigms
- Figure 4A. Performance of spaced and massed trained rats in the spatial memory task
- Figure 4B. Spaced-trained, but not massed-trained, animals preferentially explore the platform quadrant during the long-term memory test
- Figure 5. Similar swim speed of spaced- and massed-trained animals
- Figure 6. Intraperitoneally injected sodium butyrate increases acetylation of histones in the hippocampus
- Figure 7A. Performance of sodium butyrate-injected animals in the spatial memory task
- Figure 7B. Sodium butyrate-injected animals given massed training preferentially explore the platform quadrant during the long-term memory test
- Figure 8. Similar swim speed of sodium butyrate-injected massed-trained and saline-injected massed-trained animals
- Figure 9. Sodium butyrate does not affect body weight
- Figure 10. Sodium butyrate does not affect thigmotaxic activity

Chapter 4

- Figure 1. Schematic depiction of the experiment designed to study the effect of training on poly(ADP)-ribosylation of histone H1
- Figure 2. Memory training increases poly(ADP)-ribosylation of histone H1

- Figure 3. Histone deacetylase inhibition increases poly(ADP)-ribosylation of histone H1 in the hippocampus
- Figure 4. Sodium butyrate-induced poly(ADP)- ribosylation of histone H1 requires PARP activity
- Figure 5. PARP inhibitor blocks LTM formation
- Figure 6A. Schematic depiction of the experiment designed to study the effect of sodium butyrate on weak training
- Figure 6B. Sodium butyrate causes weak training to form LTM
- Figure 7. Schematic depiction of the experiment designed to study the effect of PARP inhibitor on deacetylase inhibitor-induced facilitation of long-term memory
- Figure 8. PARP activity is required for sodium butyrate-induced LTM formation after weak training