

# 20 - 9. Autism

## 9. Autism

© SPMM Course 7. Mood disorders □ A strong association between mood disorder and the number and severity of focal signal hyperintensities on T2-weighted images has been established. These white matter hyperintensities (WMH) occur particularly in the deep subcortical white matter and to a lesser extent in the basal ganglia and periventricular tissue. They are seen in excess in both bipolar and unipolar mood disorder, with an odds ratio of 3 to 7 when compared to healthy controls. □ In major depression, WMH are particularly common in elderly subjects, where they are linked to risk factors for, and the presence of, vascular disease. This finding is consistent with a robust epidemiological association between the two conditions. □ WMH confer a poor prognosis in major depression and bipolar disorder. □ Lithium treatment increases cortical grey matter volume suggesting that lithium is neurotrophic. Lithium may also enhance neurogenesis and inhibit apoptosis □ Antidepressants may affect neuronal morphology. These agents help regenerate monoaminergic axons, promote hippocampal neurogenesis and prevent the loss of dendritic spines in animal models.

8. Alcoholic brain damage □ Wernicke's encephalopathy is characterized by degenerative changes including gliosis and small hemorrhages in structures surrounding the third ventricle and aqueduct (i.e. the mamillary bodies, hypothalamus, mediodorsal thalamic nucleus, colliculi, and midbrain tegmentum), as well as cerebellar atrophy. □ Brain shrinkage can be found in uncomplicated alcoholism, which can largely be accounted for by the loss of white matter. Some of this damage appears to be reversible. □ Alcohol-related neuronal loss has been documented in specific regions of the cerebral cortex (superior frontal association cortex), the hypothalamus (supraoptic and paraventricular nuclei), and cerebellum.
9. Autism □ Hypoplasia of cerebellar vermis and to some extent the cerebellar hemispheres is documented. □ Purkinje cell count in the cerebellum is significantly lower. □ Inconsistent changes noted in the neocortex. Some suggest increased cortical volume, probably related to reduced pruning.

© SPMM Course Notes prepared using excerpts from: □ Belay & Schonberger. Variant Creutzfeldt Jakob disease and BSE. Clin Lab Med 2002;22:849-62 □ Harrison PJ. The neuropathology of primary mood disorder. Brain 2002;125:1428-49. □ Harrison PJ. The neuropathology of schizophrenia. A critical review of the data and their interpretation. Brain 1999; 122: 593-624 □ Neary, D & Snowden, J. Fronto-temporal Dementia: Nosology, Neuropsychology, and Neuropathology. Brain & Cognition 1996;31:176-87 □ Love, S. Neuropathological investigation of dementia: a guide for neurologists Journal of Neurology, Neurosurgery, and Psychiatry 2005;76(Supplement 5 ):v8-v14. □

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