Domanda N1

1) Descrivi la procedura dell'iniezione cranica stereotassica in topo
2) Descrivi un test comportamentale utilizzato per testare la memoria spaziale in modelli murini di malattie neurodegenerative
3) Descrivi la procedura di preparazione di fettine cerebrali per esperimenti di imaging del calcio tramite microscopia 2 fotoni

Informatica: Programmi di analisi statistica

Abstract

Impairments of the dialog between excitation and inhibition (E/I) is commonly associated to neuropsychiatric disorders like autism, bipolar disorders and epilepsy. Moderate levels of hyperexcitability can lead to mild alterations of the EEG and are often associated with cognitive deficits even in the absence of overt seizures. Indeed, various testing paradigms have shown degraded performances in presence of acute or chronic non-ictal epileptiform activity. Evidences from both animal models and the clinics suggest that anomalous activity can cause cognitive deficits by transiently disrupting cortical processing, independently from the underlying etiology of the disease. Here, we will review our understanding of the influence of an abnormal EEG activity on brain computation in the context of the available clinical data and in genetic or pharmacological animal models.

Domanda N2

1) Descrivi un test comportamentale utilizzato per testare la memoria non spaziale in modelli murini

2) Descrivi la tecnica di immunoistochimica su fettine cerebrali

3) Descrivi la tecnica della perfusione intracardiaca

Informatica: Descrivere come viene eseguita l'analisi di immagini delle dinamiche calcio acquisite tramite sonde fluorescenti in fettine cerebrali al microscopio a due fotoni

Abstract

Intellectual disability (ID) is a common and highly heterogeneous paediatric disorder with a very severe social impact. Intellectual disability can be caused by environmental and/or genetic factors. Although in the last two decades a number of genes have been discovered whose mutations cause mental retardation, we are still far from identifying the impact of these mutations on brain functions. Many of the genes mutated in ID code for several proteins with a variety of functions: chromatin remodelling, pre-/post-synaptic activity, and intracellular trafficking. The prevailing hypothesis suggests that the ID phenotype could emerge from abnormal cellular processing leading to pre- and/or post-synaptic dysfunction. In this chapter, we focus on the role of small GTPases and adhesion molecules, and we discuss the mechanisms through which they lead to synaptic network dysfunction.