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Sommario/riassunto	Excessive alcohol drinking represents a major social and public health problem for several countries. Alcohol abuse during pregnancy leads to a complex clinical disorder referred to as fetal alcohol spectrum disorder (FASD), chiefly characterized by mental retardation (MR). The effects of early exposure to ethanol can be reproduced in laboratory animals and this helped to answer several key questions concerning the human pathology. The interest of experimental models of FASD is twofold. Firstly, they increase our knowledge about the dose and modality of alcohol consumption able to induce damaging effects on the developing brain (see Valenzuela et al., TINS 35: 284-292, 2012). Therefore, laboratory research can help to refine health policy strategies aimed at the prevention of FASD. Second, experimental models of FASD can provide useful hints to elucidate the basic mechanisms leading to MR. In fact, experimental exposure to alcohol can be carried out during discrete, often very restricted time windows. As a consequence, FASD models, though depending on the multifaceted interference of alcohol with several molecular pathways, can nonetheless provide valuable information about which specific developmental periods and brain areas are critically involved in the genesis of MR. On the contrary, experimental models of genetically

determined MR are ideally suited to study the involvement of single molecules (e.g, the fragile X mental retardation protein).Putting together the rich ensemble of data obtained through the various experimental paradigms of alcohol exposure, as well as those deriving from other genetic and non-genetic models, one can figure out to what extent different types of MR share common pathogenetic mechanisms, regardless of whether the aetiological factors intervene during different phases of neural development, or affect different brain structures. The present Research Topic is aimed at establishing the state of the art of the current research on experimental FASD, focusing on differences and homologies with respect to other types of MR. The ultimate goal is to find out a common roadmap in view of future therapeutical approaches to MR. Particular attention will be devoted to: a) structural and functional anomalies of dendrites b) derangement and rewiring of cortical and hippocampal microcircuits c) involvement of non-cortical brain structures d) apoptosis and/or altered neurogenesis e) anomalies of ion channels, neurotransmitters, and neurotrophic factors f) comparison between experimental studies and imaging studies performed on humans affected by FASD.