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Autore	Luisa Pinto
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Sommario/riassunto	Major depression is a highly prevalent disorder that poses a significant social burden in society nowadays. The pathophysiology of this disease is still poorly understood but growing evidence suggests that impaired neuron and glial plasticity may be a key underlying mechanism for the precipitation of the disorder. One of the most surprising findings in this field was the involvement of glial cells in the pathophysiology of major depression and in the action of antidepressants, namely in mechanisms related with adult neurogenesis imbalances or dendritic arborization impairments. In particular, several works refer to alterations in the morphology and numbers of astrocytes, microglia and oligodendrocytes in the context of depression in human patients or animal models of depression. These observations were linked to functional evidences and suggested to underlie the pathophysiology of depression. Among others, these include impairments in the cross-talk between glia and neurons, changes in the level of neurotransmitter or immunoactive substances, myelination status, synapse formation, maintenance, or elimination. In addition to the implication of glia in the pathophysiology of depression, a number of studies is ascribing glia pathways to classically accepted antidepressant mechanisms. Therefore, it is noteworthy to elucidate the role of glia in the effect provided by antidepressant treatment in order to better understand secondary effects and elucidate alternative targets for treatment.