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| Sommario/riassunto | Strong evidence continues to accumulate indicating that amyloid-beta (A) is a central part of Alzheimer's disease (AD) pathogenesis in spite of the negative evidence coming from failed clinical trials. Therefore, mechanisms of clearance of A are of great interest in understanding AD pathogenesis and the development of effective treatments. This topic focuses on the issues related to A clearance in AD. The topics covered include proteases that degrade A and their localization, regulation, and functions. This topic also covers issues related to clearance through uptake by glia and through low-density lipoprotein (LDL) receptor mediated mechanisms. Signal transduction related to AD pathology and clearance is also addressed. Finally, immunotherapy and other novel therapeutic approaches are discussed. |