Biological membranes are featured by a remarkable ability to modulate a wide range of physiological and pathological processes. Of these, protein aggregation is currently receiving the greatest attention, as one type of the ordered protein aggregates, amyloid fibrils, proved to be involved in molecular etiology of a number of fatal diseases. It has been hypothesized that nucleation of amyloid fibrils and toxic action of their precursors is mediated by lipid–protein interactions. Lipid bilayer provides a variety of environments in which aggregated state of polypeptide chain appears to be more thermodynamically favorable than its monomeric form. The major factors responsible for the enhanced self-association propensity of membrane-bound proteins include (i) structural transition of polypeptide chain into aggregation-prone conformation; (ii) protein crowding in a lipid phase; (iii) particular aggregation-favoring orientation and bilayer embedment of the protein molecules. All these factors are considered in the present review with an emphasis being put on the role of electrostatic, hydrophobic, and hydrogen-bonding phenomena in initiating and modulating the protein aggregation on a membrane template. Likewise, we survey the advanced experimental techniques employed for detection and structural characterization of the aggregated species in membrane systems.