Intraneuronal amyloid β (Aβ) accumulation is one of the initial events in Alzheimer’s disease (AD). Aβ deposits are formed by the 40 and 42 amino acid isoforms as well as different N- and C-truncated Aβ species of varying length. The following thesis is divided into two main parts that correspond to two N-truncated peptides: Aβ_{4-x} and Aβ_{5-x}. The Aβ_{4-x} variants are highly abundant, have a high aggregation propensity and may appear before other Aβ isoforms in the disease. In contrast, the Aβ_{5-x} isoforms have not received much attention, despite the central role suggested for the N-truncated species in AD etiology. The first part of this work uses the Tg4-42 transgenic mouse model to study the neurotoxic effects of Aβ_{4-42} overproduction as well as the possible propagation mechanisms of these peptides through interconnected areas. Regions with no intraneuronal Aβ_{4-42} deposits showed no signs of neurodegeneration. The piriform cortex was severely affected by intracellular Aβ_{4-42} and nearly 50 % of neuronal loss. This results support previous evidence showing that intraneuronal Aβ aggregation trigger neurodegeneration. In addition, dentate gyrus and CA3 lack intraneuronal Aβ and remained intact compared to wild-type (WT) animals. These regions belong the trisynaptic hippocampal loop and were not affected by the Aβ_{4-42} deposited in other areas of this network. This results suggest that Aβ_{4-42} neurotoxicity does not spread in a prion-like manner to axonally connected areas that do not express the transgene. However, further experiments are needed to exclude a prion behaviour of Aβ. On the other hand, the second part of this thesis showed the presence and distribution of Aβ_{5-x} peptides in brains affected by familial AD (FAD) and three transgenic mouse models. The AB5-3 antibody was produced and characterized as a tool to detect Aβ_{5-x} immunoreactivity. Remarkably, Aβ peptides starting at position five were found in amyloid plaques and cerebral amyloid angiopathy (CAA) of all FAD cases and 80 % of the sporadic AD (SAD) ones. The FAD cases included subjects with a mutation in the amyloid precursor protein (APP) or the presenilin1 (PS1) gene. CAA showed a stronger Aβ_{5-x} signal than the amyloid plaques in the SAD cases, while both were equally abundant in sections obtained from FAD patients. Similarly, Aβ_{5-x} was observed in plaques affecting different brain regions of the three mouse models. As no intracelluler Aβ_{5-x} was found in humans or in transgenic AD mice, these isoforms probably do not participate in the early stages of AD.