Mouse mammary tumor virus (MMTV) is a prototypical member of the Betaretrovirus genus characterized by the ability to preassemble viral particles in the cytoplasm of the host cells. Intracellularly preassembled particles are subsequently transported to the plasma membrane being enveloped by a lipid bilayer and released from the cell in the process referred to as budding. Retrovirus particle assembly is driven by the Gag polyprotein precursor, which is cleaved in the maturation process by virus-encoded protease to liberate multiple structural proteins. The matrix (MA), capsid (CA) and nucleocapsid (NC) protein domains that are common to all retroviruses and in the case of MMTV, also the noncanonical domains, pp21, p3, p8 and „n“, located between MA and CA domain are present. The role of these specific domains remains undefined.

The retroviral budding is stimulated by short peptide motifs, so-called late (L) domains, located within Gag sequence. These L domains mediate interactions with cellular proteins normally involved in the biogenesis of the multivesicular bodies and protein sorting. Three types of the L domains have been identified to date, with the consensus of the amino acid sequences (i) P(T/S)AP, (ii) YP(x)nL (where x represents any amino acid and $n \leq 3$) and (iii) PPxY. Disruption of the L domain sequence prevents viral particle release at the late stages. No L domain has been described for MMTV yet.

The aim of this study was to characterize the process of the mouse mammary tumor virus particle assembly and release. Therefore this thesis focused on the noncanonical domains presented within Gag polyprotein, as well as on selected amino acid sequences located within Gag molecule. Results described here provide the first glimpse into the role of unique protein domains of MMTV Gag polyprotein precursor and the importance of specific amino acid residues.