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Targeting of MuLV Gag to the plasma membrane is mediated by PI(4,5)P_2 and PhosphatidylSerine

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Membrane targeting by the modern human immunodeficiency viruses is dependent on the plasma membrane-located phospholipid PI(4,5)P_2. In order to determine if evolutionarily distant retroviruses are targeted by a similar mechanism, we generated mutant Gag constructs in the matrix (MA) domain of the Murine Leukemia Virus (MuLV) and examined their binding to membrane models and phenotypes in cell culture. Mutations in the MA polybasic region altered Gag localization, membrane binding and virion production. In addition, we show that MA binds with good affinity to all the phosphatidylinositol phosphates but displays a strong specificity for PI(4,5)P_2 only if enhanced by phophatidylserine. Virus production was strongly impaired by PI(4,5)P_2 depletion under 5ptaseIV overexpression. Our results suggest that the N-terminal polybasic region of MA is essential for Gag targeting to the plasma membrane and Gag cellular trafficking. The binding of the MA domain to PI(4,5)P_2 appears to be a conserved feature among retroviruses, despite the fact that the MuLV-MA domain is structurally different from that of HIV-1 and -2 and lacks a readily identifiable PI(4,5)P2 binding cleft.