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The role of murine leukemia virus glycosated Gag protein in virus replication

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Murine leukemia viruses (MuLVs) encode an alternate form of Gag protein in addition to the standard Gag precursor polyprotein, glycosylated Gag (glyco-gag or $gPr80^{gag}$). $gPr80^{gag}$ contains additional amino-terminal peptides, and it is glycosylated. Until recently the function of $gPr80^{gag}$ was unclear, but we have recently shown that $gPr80^{gag}$ facilitates viral budding and release through lipid rafts. Glyco-gag mutant virus is less efficiently released from fibroblasts, mutant virus has lower cholesterol content (and higher buoyant density), and less Gag precursor polyprotein ($Pr65^{gag}$) is associated with detergent-resistant membranes in infected cells. The mechanism by which $gPr80^{gag}$ facilitates virus release is under investigation. The N-terminal 88 amino acids of $gPr80^{gag}$ are sufficient to enhance virus release, indicating that the Gag sequences on this protein are not required. Cellular La protein is involved in $gPr80^{gag}$ function, since overexpression of La phenocopies $gPr80^{gag}$ in facilitating virus release, and knockdown of La abrogates $gPr80^{gag}$-enhancement of virus release. MuLV glyco-gag can also facilitate release of HIV-1 particles from transfected cells, which suggests that these two viruses may share mechanisms for directing virus release through lipid rafts. $gPr80^{gag}$also counteracts the murine APOBEC3 restriction factor (mA3) since $gPr80^{gag}$mutant virus shows a defect for establishment of infection in wild-type mice, but not in mA3 knockout animals.

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