ORAL PRESENTATION





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 (Pr65gag) 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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