The egress of enveloped viruses, particularly RNA viruses (retroviruses..), is well characterized ([*1-4*](#_ENREF_1)). Nascent capsids become engulfed in a plasma membrane-derived lipid envelope by hijacking the multivesicular body (MVB) biogenesis machinery ([*5*](#_ENREF_5)). Current models propose a sequential, virus-induced recruitment of class E vacuolar protein sorting (VPS) proteins which act in three complexes, referred to as ESCRT I-III (endosomal sorting complex required for transport) ([*6*](#_ENREF_6)). Retroviruses directly interact with the ESCRT components via proline-rich late domains (L-domains) which are present in their structural proteins ([*7*](#_ENREF_7)). ESCRT components or viral sequences in the proximity of the L-domains become ubiquitinated by ubiquitin binding proteins, such as tumor-susceptibility gene 101 (TSG101) or Nedd4-like ubiquitin ligases. Ubiquitination results in the biogenesis and fission of MVBs and thus, budding of the virions ([*8*](#_ENREF_8)).

In contrast, the release of non-enveloped viruses from mammalian host-cells is generally associated with cellular lysis, thus considered a passive process ([*9-13*](#_ENREF_9)). However, there is rising evidence that an active egress of non-enveloped viruses precedes virus-induced cell lysis. For instance bluetongue virus (BTV), ~~a member of the genus~~ *~~Orbivirus~~* ~~within the~~ *~~Reoviridae~~* ~~family~~, has been demonstrated to usurp the ESCRT machinery for egress ([*14*](#_ENREF_14)). Comparable to enveloped viruses, BTV interacts with TSG101 via its L-domain in order to exploit the cellular MVB sorting pathway ([*15*](#_ENREF_15)). Similarly, Hepatitis A virus (HAV) uses host membrane hijacking to egress in a TSG101-independent manner. Thus, HAV release involves ESCRT-associated proteins but seems to be independent on the early ESCRT complexes required for initial cargo recruitment ([*16*](#_ENREF_16)). Poliovirus, another picornavirus, constitutes a further example for non-lytic egress of viruses lacking an envelope. Drug-induced stimulation of the autophagy pathway increased non-lytic spread of the virus ([*17*](#_ENREF_17)). Additionally, progeny virions were shown to accumulate unilaterally on the apical surface of polarized and productively infected epithelial cells ([*18*](#_ENREF_18)). Equally, simian vacuolating virus 40 (SV40) was almost exclusively recovered from the apical culture fluid of polarized epithelial cells. Moreover, the egress of SV40 occurred prior to cell lysis. The appearance of SV40 in smooth membrane reticular structures argues for a vesicle-associated release of SV40 virions ([*19*](#_ENREF_19)). Finally, simian rotavirus (RRV) was demonstrated to egress from the apical pole of epithelial cells before any cell lysis was detected. Electron microscopy studies and specific inhibition of vesicular transport pathways indicate a vesicle-associated release of progeny virions that is irrespective of the golgi-dependent secretory pathway ([*20*](#_ENREF_20)).

***Polio-paper…***

…Documentation that such events are truly nonlytic, however, requires rigorous demonstration that no cell lysis occurred. However, it has been difficult to test this and other hypotheses concerning unconventional secretion because the use of cell populations makes it nearly impossible to exclude the possibility that lysis of a few cells is responsible for the release of cytoplasmic constituents. …

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