Abstract.
Understanding the pathway of HIV infectivity is crucial for designing any treatment or vaccination to prevent the immunodeficiency that is caused by the virus. HIV-1 is known to infect the CD4 T cells and it also infects macrophages[1]. However, it does not cause cell death in macrophages, while the CD4 T cells are depleted by productive viral infection.[1] Delineating the mechanisms that cause the distinction in cell fate between the different target cells of HIV-1 infection would be an important piece of the puzzle. Up until recently autophagy was mainly viewed as a fundamental anti-viral mechanism, but recent discoveries have shown that the cellular process of autophagy can be sabotaged by HIV-1 and used to its advantage during productive infection[2]. HIV-1 infected cells as well as the HIV-1 particles are coated with the viral envelope glycoproteins (gp120 and gp41, Env). The virus uses these Env proteins for fusion with the cell membrane and subsequent entry into cells[3]. New research in the field shows that the Env proteins from HIV-1 (both, X4 and R5 strains) can trigger autophagy induced cell death in the uninfected CD4 cells, and could possibly be central to the development of immunodeficiency pathology[1,3]. However autophagy is inhibited in the HIV-1(X4 and R5 strain) infected CD4 cells[3]. In contrast uninfected macrophages do not undergo Env-mediated autophagy[3]. Yet another contradiction is seen in the infected macrophages that show induction of autophagy. They demonstrate a requirement for autophagy induction to carry out a productive infection. Blockage of final stages of autophagy in the infected macrophages greatly increases the productive infection, indicating that autophagy plays a dual role in viral replication in macrophages[3]. These discrepancies display that there is a bias in the HIV-1 infectivity and suggests that HIV-induced autophagy is a cell-type dependent process[3]. Further studies are required to better understand the contribution of autophagy to HIV-1 pathogenesis.

References.