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Sommario/riassunto	Productive HIV infection requires completion of all the steps of the replication cycle, the success of which largely relying on the multiple interactions established by viral proteins with cellular partners. Indeed, cellular and viral fates are intertwined and this interplay may involve rerouting of cellular factors/pathways to the benefit of the viral life cycle. To gain a foothold into host cells, HIV has to take advantage of available cellular factories and overcome the numerous potential blocks opposed to its replication while ensuring cellular survival. Viral auxiliary proteins are a perfect paradigm to illustrate the complexity of the relationship between HIV and its host. Although these accessory proteins are mostly unnecessary for viral replication in permissive cells in vitro, they play a crucial role in regulating viral spread ex vivo in non-permissive cells and in vivo in hosts. Most accessory proteins are pleiotropic and instrumental in the counteraction of restriction factors and proteins involved in innate immune response.Several proteins of the “intrinsic” immune system that detect the presence of the assailant and initiate a subsequent immune response, as well as restriction factors that are directly devoted to arresting the replication cycle at precise steps have been characterized. Despite the numerous cellular

mechanisms dedicated to preventing viral replication, HIV is able to efficiently replicate in humans. Indeed, as a master regulator of cellular machineries and processes, not only has HIV evolved strategies to avoid triggering of pattern recognition receptors, but HIV has also elaborated ways to counteract host restriction factors, thereby overcoming the hurdles that oppose efficient replication.