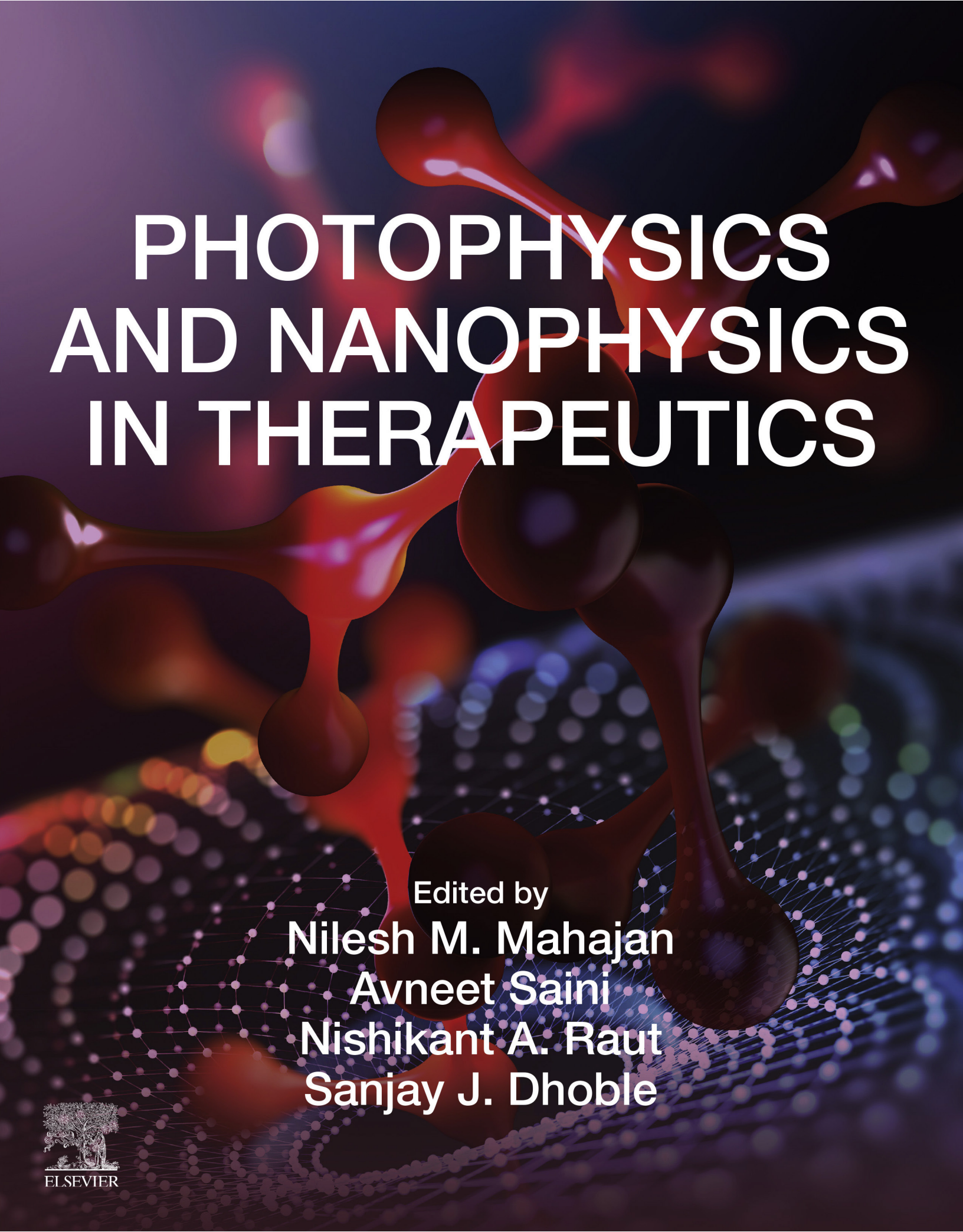
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Photophysics and Nanophysics in Therapeutics

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Chapter 15

Role of nanocarriers for the effective delivery of anti-HIV drugs

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15.1 Introduction

The human immunodeficiency virus (HIV) is an enveloped retrovirus belonging to family *lentivirus* with single-stranded RNA molecules (approximately 9 kb). Since its isolation and identification are divided into major two types, human T cell leukemia viruses (HTLV), and human immunodeficiency viruses (HIV). HTLV is subdivided into HTLV-1 and HTLV-2 belongs to the subclass *oncovirinae*, causes adult T cell leukemia and spastic paraparesis (Saura, 1998; Shuh and Beilke, 2005). HIV is subdivided into HIV-1 and HIV-2 from the subclass *lentivirinae* which are responsible for acquired immunodeficiency syndrome (AIDS). From the vast majority of viruses in the developed world, HIV-1 is mainly responsible for global AIDS pandemic and generally called HIV (Wilen et al., 2012). HIV infects CD4+ receptor-bearing helper T cells and ultimately loss of CD4 cells leads to immune deficiency and developing an opportunistic infection. AIDS transmitted by body fluid transfer including blood transfusion, organ transplant, sexual contact, and perinatally from mother to offspring. HIV-1 exist in two predominant forms such as syncytium inducing strain (SI or T-cell tropic) and nonsyncytium inducing (NSI or macrophage-tropic, M-tropic) strain. In SI pathology, HIV infects T-cell by using CXCR4 co-receptor, whereas NSI pathology is associated with CCR5 co-receptor with slower disease progression. Infection with NSI-tropic strain of HIV takes 7.12 years for progression of AIDS however T-tropic strain of the virus may take only 2–3 years (Fauci, 1993; Gardner et al., 2004).

HIV-1 stores its genetic information in RNA instead of DNA; therefore they require DNA when entering into the human cell to make replicate themself. The outer shell of the virus is called envelope which is covered by spike glycoprotein including gp 120 and gp 41which help to enter and lock the virus into the CD4+ receptor. The core of the virus is held a cone-shaped structure called capsid which contains two enzymes, the reverse transcriptase and integrase which is essential for replication of HIV (German Advisory Committee Blood, 2016). Capsid also contains two strands of RNA containing nine genes which hold genetic material and provide instruction to make new viruses. Out of these nine genes, three genes known as *gag*, *pol*, and *env* provide the instructions to make structural and nonstructural proteins for new virus particles (Fig. 15.1). The other six genes such as *tat, rev, nef, vif, vpr,* and *vpu* provide code for proteins which able to make HIV, infects the host cell, produce new virus and release them from infected cells (Margolis, 2010; Palmer et al., 2011).

15.1.1 HIV life cycle and pathogenesis

The life cycle of HIV is complex and roughly divided into the early and late phase of replication (Fig. 15.2). The early phase starts with the attachment of the virion at the host cell surface and ends with the integration of proviral DNA into the host cell genome. However late phase of replication starts with the transcription of proviral DNA and ends with the release of fully infectious progeny virions (Zinkernagel, 1996; Panova, 2020).

15.1.1.1 Viral attachment and binding

In highly activated CD4+ T cells, the life cycle of HIV last in only 1–2 days in association with the planned death of both virally infected and uninfected bystander CD4+ T cell.

Host cell-free HIV virions have 20–30 min half-life thus, the virus must find and infect a new target host cell within a short time. As described above, CD4+ is the primary receptor, and chemokine CCR5 and CXCR4 are important co-receptor for HIV entry into the host cell. Several other receptors, such as poly-glycans, lectins and others can also bind HIV virions

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