Cellular miR-2909 can Restrict Pro-Viral Cyclophilins

Deepak Kaul*
Experimental Medicine and Biotechnology Department, Postgraduate Institute of Medical Education and Research, Chandigarh-160012, India

Editorial

Mounting evidence suggests that cyclophilin genes are encoded by the genomes of all living forms including that of a mimivirus [1]. The large cyclophilin family has not only been implicated in various diseases including cancer, diabetes, neurodegeneration and atherosclerosis [2] but also in the life cycles of diverse viruses [2] including human cytomegalovirus, vesicular somatitis virus, vaccine virus, measles virus, human papilloma virus, hepatitis B virus, and HIV-1. Cyclophilins have an evolutionary conserved trait of peptidyl-prolyl cis/trans isomerases required for the proper folding of certain proteins [2]. The first link between HIV-1 replication and host cyclophilin was established by the finding that revealed a direct interaction of cyclophilin (CyPA) with the HIV-1 Gag polyprotein [3]. Subsequently, it was established that this binding of CyPA was crucial for the HIV-1 life cycle [4,5]. Two other HIV-1 proteins, Vpr and p6, have been reported to interact with CyPA thereby asserting its role as a proviral factor in the HIV-permissive human cells [6,7]. Several evidences support the view that host AATF genome encoded miR-2909 has the inherent capacity to target HIV-1 genome encoded hiv-1-miR-H1 as well as its Vpr gene [8]. In this context, it is pertinent to note that miR-2909 has the ability to target cyclophilin family members (Table 1), which have known as well as unknown cellular functions. Keeping in view that Cyclophilins influence diverse viral life cycles (including that of HIV-1), it would be interesting to explore the antiviral role of miR-2909 through its inherent capacity to target a selected set of cyclophilin family members (Table 1).

References


*Corresponding author: Deepak Kaul, Experimental Medicine & Biotechnology Department, Postgraduate Institute of Medical Education & Research, Chandigarh-160012, India, Tel: 91-0172-2755233; E-mail: dkaul_24@hotmail.com

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