

## Cellular miR-2909 can Restrict Pro-Viral Cyclophilins

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### 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 stomatitis 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].

Gene	Localization	Size	Host Cellular Function
CyPA (PPIA) NM_021130	Cytoplasm; Nucleus; Secreted	18kDa	Inflammation; Tumor Progression
CyPNK (NKTR) NM_005385	Membrane	150kDa	Tumor recognition in NK Cells
CyP60 (PPIL2) NM_014337	Nucleus; Golgi	59kDa	Cell surface expression of CD147
PPIL4 (PPIL4) NM_139126	Nucleus	57kDa	Not Known
SDCCAG-10 (CWC27) NM_005869	Nucleus	54kDa	Not Known

Table 1: Cyclophilins Targeted by miR-2909.

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).

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