Rift Valley Fever Virus is a mosquito-borne zoonotic pathogen, which causes hemorrhagic fever, neurological disorder or blindness in humans and a high rate of abortion in ruminant. NSs is a major virulence factor of RVFV and induces 1) host transcription shutoff and 2) the degradation of PKR. MP-12 strain is a candidate live-attenuated vaccine for Rift Valley fever. MP-12 retains residual virulence in the S-segment, which encodes functional NSs proteins. To further attenuate MP-12, and maintain the strong immunogenicity, we aimed to replace MP-12 NSs with NSs derived from other phleboviruses. We have generated recombinant MP-12 encoding Punta Toro Virus Adames (rMP12-PTANSs) or Balliet strain NSs (rMP12-PTBNSs), Sandfly Fever Sicilian virus NSs (rMP12-SFSNSs) or Frijoles Virus NSs (rMP12-FRINSs) in place of MP-12 NSs by using reverse genetics system. None of mutants promoted degradation of PKR in VeroE6 cells or mouse Hepa1-6 cells, while all, except for rMP12-FRINSs, did not increase the abundance of IFN-β mRNA upon infection. rMP12-SFSNSs or rMP12-PTANSs replicated more efficiently than rMP12-PTBNSs or rMP12-FRINSs in Hepa1-6 cells. Outbred CD1 mice were immunized with those mutants (1x10⁵ pfu, s.c.), and challenged with wt RVFV ZH501 (1x10³ pfu, i.p.) at 44 dpi. Viremia was detected in mice immunized with MP-12 (10%) or rMP12-PTANSs (11%) at 3 dpi, while others did not develop it. Neutralizing antibodies were detected in 60, 78, 70, 67 or 100% in mice immunized with MP-12, rMP12-PTANSs, rMP12-PTBNSs, rMP12-SFSNSs or rMP12-FRINSs, and the mean titers were 1:834, 1:1512, 1:712, 1:534 or 1:154, respectively, while 67, 78, 60, 89 or 90% of them survived after wt RVFV challenge, respectively. Our data suggested that all the MP-12 mutants were similar or better efficacy than parental MP-12 in mice. We are also testing the attenuation of wt RVFV encoding other phlebovirus NSs.