The 2015 Zika virus (ZIKV) outbreak originating in Brazil saw an unprecedented upsurge of severe neurological phenotypes such as Guillain Barré syndrome (GBS) in adults and neurodevelopmental disorders in neonates. Zika congenital syndrome encompasses an array of conditions associated with prenatal ZIKV infection, some of which may take significantly longer post-partum to manifest. It is evident that ZIKV-mediated neurological complications remain to be fully characterised at the clinical, cellular and molecular levels. Herein we present mixed cell co-culture systems to model ZIKV-mediated neurotropism and toxicity within the central and peripheral nervous systems (CNS and PNS). Such systems have previously been utilised to study GBS (PNS) and multiple sclerosis (CNS). Co-cultures are derived from embryonic day 13, Ifnar1 knockout or wildtype mouse (background strain A129) spinal cord or dorsal root ganglia; modelling the developing CNS and PNS respectively. Cultures were infected with a Brazilian ZIKV isolate (ZIKV/H. sapiens/Brazil/PE243/2015) and subjected to immunofluorescence analysis using cell type and ZIKV specific antibodies. Total populations of each cell subtype, ZIKV infected cells, and double-positive cells were enumerated. Furthermore, the number of pyknotic nuclei present in infected versus mock infected co-cultures was assessed. Consequently, we were able to ascertain cell populations targeted during ZIKV infection and their relative susceptibility to infection in the presence or absence of type I interferon signalling. These data provide important insights into pathogenesis of ZIKV infection in the central and peripheral nervous systems.