Viral control of apoptosis can be a double-edged sword. In some viral infections, immune cell apoptosis will contribute to pathogenesis and determine disease evolution either by direct infection of immune cells or as a result of bystander cell apoptosis caused by viral proteins or mediators secreted from infected cells. The pathogenesis of viral infections involves dynamic interactions between viruses and hosts, which can result in different outcomes including cell death, elimination of the virus or latent infection. Viruses deliver genomes and proteins with signaling potential into target cells, resulting in growth, proliferation and apoptosis. Viral infection modifies key cell regulatory elements involved in apoptotic pathways to successfully accomplish viral. Apoptosis is usually beneficial for the host, since apoptotic clearance of infected cells can inhibit microbial replication and propagation (Lamkanfi and Dixit, 2010). In addition, tissue injury is reduced with this type of cell death. The up-regulated genes shown in Figure 1 are part of an ostensible core of the cell response to bacterial and viral pathogens which is expanded by other genes included in Supplementary Files S2, S3. There are two essential components in these host-pathogen interactions shared by respiratory viruses and bacteria: (i) recognition of the invading pathogens by collective PRRs and (ii) induction of apoptosis. Both components are intimately integrated to ensure a better control of the infecting microorganisms. Apoptosis is a form of programmed cell death that occurs in multicellular organisms. Biochemical events lead to characteristic cell changes (morphology) and death. These changes include blebbing, cell shrinkage, nuclear fragmentation, chromatin condensation, chromosomal DNA fragmentation, and global mRNA decay. The average adult human loses between 50 and 70 billion cells each day due to apoptosis. For an average human child between the ages of 8 and 14, approximately 200–300 billion cells die per day. 

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