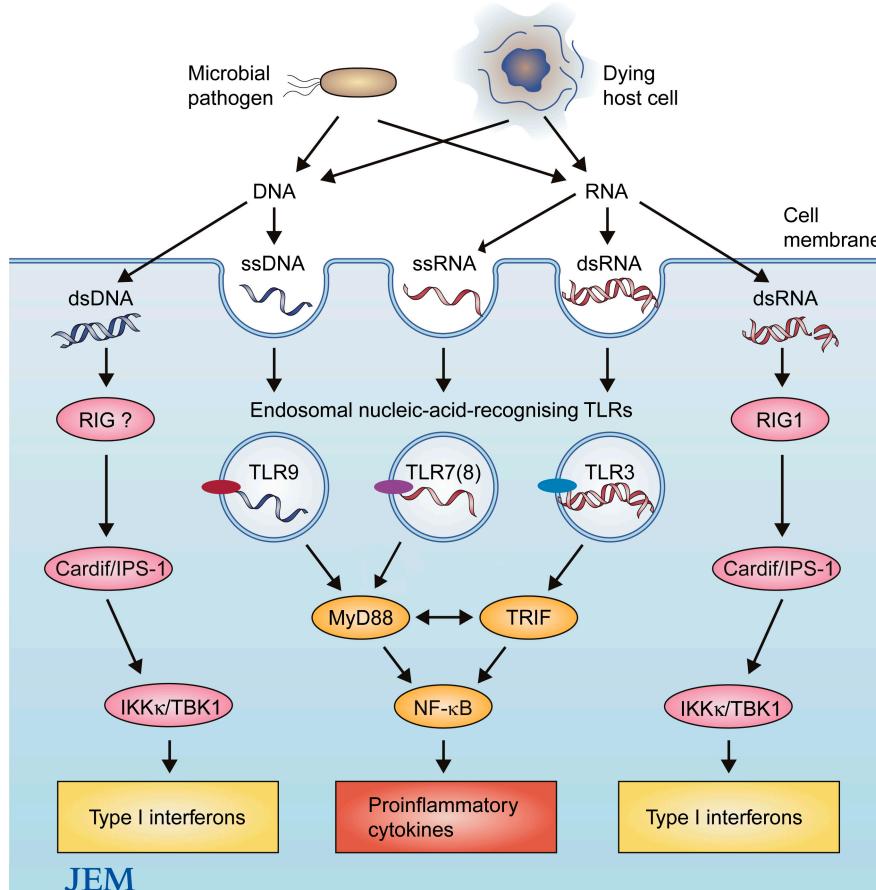


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The term interferon  $\beta$  promoter stimulator 1 (IPS) referred to in the text originates from the work of Kawai et al. (Kawai, T., K. Takahashi, S. Sato, C. Coban, H. Kumar, H. Kato, K.J. Ishii, O. Takeuchi, and S. Akira. 2005. IPS-1, an adaptor triggering RIG-I- and Mda5-mediated type I interferon induction. *Nat. Immunol.* 6:981–988). The reference was inadvertently not cited in the original text.

Please note that an error also appeared in the online early release version of this article. The final html, pdf, and print versions have been corrected. For reference, the correction appears below.

The authors regret an error in Fig. 1. The arrow from the TLR7–9-expressing endosomes (TLR7(8)) should be directed towards MyD88 (rather than towards TRIF). The corrected figure and its legend appear below.



**Figure 1. Nucleic acid recognition pathways in innate immune cells.** Both pathogen-derived RNA or DNA and host-derived mammalian RNA or DNA are sensed via TLR and TLR-independent recognition pathways. Upon endosomal translocation, viral dsRNA, microbial or mammalian ssRNA and ssDNA are recognized by endosomally expressed TLR3, TLR7, (8) and TLR9, respectively. After ligation of TLR7, TLR8, and TLR9, the adaptor molecule MyD88 is recruited and drives the production of proinflammatory cytokine genes or type I interferon genes. TLR3 ligation triggers type I interferon genes via the adaptor protein TRIF. Viral dsRNA is also sensed by RIG-1 (retinoic acid inducible gene 1), which was recently shown to recruit Cardif/IPS-1, a new CARD-containing adaptor protein. Cardif/IPS-1 in turn interacts with  $\text{Ikk}\alpha/\beta/\gamma$  kinases and thus activates IRF3. Mammalian DNA triggers type I interferon production by an ill-defined signal pathway. Whether the dsDNA recognition receptor belongs to the RIG family is not yet known.