ADDENDUM

Cell death: RIPK1 protects epithelial cells

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Nature Reviews Molecular Cell Biology 15, 629 (2014)

It has come to our attention that similar findings were reported earlier in studies published by Dillon $\it et al.$, Rickard $\it et al.$ and Kaiser $\it et al.$, which identified RIPK1 as an inhibitor of apoptosis and necroptosis, and by Newton $\it et al.$, Berger $\it et al.$ and Polykratis $\it et al.$, which highlighted the importance of the kinase activity of RIPK1 in these processes. These papers all provide valuable insight into the function of RIPK1.

ORIGINAL RESEARCH PAPERS Dillon, C. P. et al. RIPK1 blocks early postnatal lethality mediated by caspase-8 and RIPK3. Cell 157, 1189–1202 (2014) | Rickard, J. A. et al. RIPK1 regulates RIPK3–MLKL-driven systemic inflammation and emergency hematopoiesis. Cell 157, 1175–1188 (2014) | Kaiser W. J. et al. RIP1 suppresses innate immune necrotic as well as apoptotic cell death during mammalian parturition. Proc. Natl Acad. Sci. USA 111, 7753–7758 (2014) | Newton, K. et al. Activity of protein kinase RIPK3 determines whether cells die by necroptosis or apoptosis. Science 343, 1357–1360 (2014) | Berger S. B. et al. Cutting edge: RIP1 kinase activity is dispensable for normal development but is a key regulator of inflammation in SHARPIN-deficient mice. J. Immunol. 192, 5476–5480 (2014) | Polykratis et al. Cutting Edge: RIPK1 kinase inactive mice are viable and protected from TNF-Induced necroptosis in vivo. J. Immunol. 193, 1539–1543 (2014)