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Injury, Inflammation, and Sepsis: Laboratory and Clinical Approaches

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COVER: Schematic diagram of the PI3K signaling pathway and its involvement in NF-κB signaling during sepsis. The PI3K pathway is activated by a variety of ligands and receptors. The means by which activation occurs through TNF-α and complement C5-9 has not been fully elucidated and may include additional mechanisms. The downstream protein kinase, Akt, serves as a common convergent point to facilitate cellular survival and modulate NF-κB-mediated gene transcription. Preferential activation of a specific NF-κB pathway depends upon a multitude of intracellular and extracellular factors. The PIP_2 and PIP_3-membrane phospholipids; GSK-3β-glycogen synthase kinase-3β; FOXO-subgroup of the Forkhead family of transcription factors; IκB-the inhibitory subunit of NF-κB; IKK-IκB kinase; TLR-Toll-like receptor; IL-1R-IL-1β receptor; TNFR-TNF-α receptor; TNFR-TNF-α receptor; IRAK-IL-1R-associated kinase; TRAF-TNFR-associated factor; TRADD-TNFR-associated death domain protein; RIP-receptor interacting protein. See Manukyan et al., pages 442–449, 2010.