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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- κ B 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- κ B-mediated gene transcription. Preferential activation of a specific NF- κ B pathway depends upon a multitude of intracellular and extracellular factors. The PIP₂ and PIP₃-membrane phospholipids; GSK-3 β -glycogen synthase kinase-3 β ; FOXO-subgroup of the Forkhead family of transcription factors; I κ B-the inhibitory subunit of NF- κ B; IKK-I κ B kinase; TLR-Toll-like receptor; IL-1R-IL-1 β 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.