The expression of genes involved in the inflammatory response is controlled both transcriptionally and post-transcriptionally. Primary inflammatory stimuli, such as microbial products and the cytokines interleukin-1 (IL-1) and tumour necrosis factor alpha (TNF alpha), act through receptors of either the Toll and IL-1 receptor (TIR) family or the TNF receptor family. These cause changes in gene expression by activating four major intracellular signalling pathways that are cascades of protein kinases: namely the three mitogen-activated protein kinase (MAPK) pathways, and the pathway leading to activation of the transcription factor nuclear factor kappa B (NF kappa B). The pathways directly activate and induce the expression of a limited set of transcription factors which promote the transcription of inflammatory response genes. Many of the mRNAs are unstable, and are stabilized by the p38 MAPK pathway. Instability is mediated by clusters of the AUUUA motif in the 3' untranslated regions of the mRNAs. Control of mRNA stability provides a means of increasing the amplitude of a response and allows rapid adjustment of mRNA levels. Not all mRNAs stabilized by p38 contain AUUUA clusters; for example, matrix metalloproteinase-1 and -3 mRNAs lack these clusters, but are stabilized. Inflammatory gene expression is inhibited by glucocorticoids. These suppress MAPK signalling by inducing a MAPK phosphatase. This may be a significant mechanism additional to that by which the glucocorticoid receptor interferes with transcription factors [1)].

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Saklatvala J, Dean J, Clark A. Control of the expression of inflammatory response genes. Biochem Soc Symp. 2003;(70):95-106. Review. PubMed PMID: 14587285.