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## Modelling a signalling module

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The NF- $\kappa$ B transcription factor plays a critical role in regulating cell growth, cell survival and the response to stress. The activity of NF- $\kappa$ B is controlled by three inhibitory I $\kappa$ B isoforms (I $\kappa$ B $\alpha$ , - $\beta$ , and - $\epsilon$ ) that regulate NF- $\kappa$ B cellular localization. In the November 8 *Science*, Hoffmann *et al.* describe a computational modelling approach to understanding NF- $\kappa$ B regulation (*Science* 2002, **298**:1241-1245). They constructed a computational model that incorporates multiple control parameters including the rate of synthesis of each I $\kappa$ B isoform, the formation and stability of binary and tertiary protein complexes, cellular localization and transport rates. The model predicts an oscillatory NF- $\kappa$ B activation profile created by negative feedback regulation loops. Analysis of mouse fibroblasts lacking combinations of I $\kappa$ B genes revealed that the different isoforms function to modulate fast and slow responses to inflammatory stimuli and are responsible for distinct gene expression programs.

## References

1. NF- $\kappa$ B: a pleiotropic mediator of inducible and tissue-specific gene control.
2. NF- $\kappa$ B and Rel proteins: evolutionarily conserved mediators of immune responses.
3. *Science*, [<http://www.sciencemag.org>]