



Mathematical Modelling of the beta-TrCP-dependent Regulation of Canonical NF-kappaB and Wnt/beta-catenin Signalling

Uwe Benary

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Cells gain information about their environment through signals that are transduced from specific signal receptors via signalling pathways into the cell's nucleus to regulate gene expression. This enables cells to adequately react to environmental changes. Aberrant signal transduction can result in inappropriate cellular responses causing diseases such as cancer. Signalling pathways are built of complex interactions between many signalling molecules creating regulatory feedbacks and mutual interaction mechanisms (crosstalk). Mathematical modelling approaches provide sophisticated methods to investigate how signals propagate through these complex signalling networks and to predict interference strategies to correct for aberrant signal transduction.

Here, signal transduction through the canonical NF-kappaB and the Wnt/beta-catenin signalling pathway is investigated under wild-type and cancerous conditions. Signal transduction in both pathways depends on ubiquitination and proteasomal degradation of central pathway components mediated by beta-transducin repeat-containing proteins (beta-TrCP). Hence, conditions are explored that enable or prevent potential crosstalk by competitive beta-TrCP sequestration. The analyses offer mechanistic explanations to account for conflicting experimental observations concerning the mutual impact of NF-kappaB and Wnt/beta-catenin signalling. Since expression of the two mammalian beta-TrCP paralogues FWD1/beta-TrCP1 and HOS/beta-TrCP2 is regulated by Wnt/beta-catenin signalling, two transcriptional feedback mechanisms are established in the signalling network adding to its complexity. The specific impact of each feedback is thoroughly dissected casting doubts on the current notion of functional redundancy of FWD1 and HOS.

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