**Supplementary file 3.** **List of newly described biochemical parameters in the Systems Model *v*1.0**

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| --- | --- | --- | --- |
| **Biochemical reaction** | **Reaction no. as in Table 2** | **Corresponding modeled reaction** | **Justification for the parameter value** |
| constitutive transcription of NFkB1 mRNA | 5 | tNFkB1 | tNFkB1 is a composite species encoding RelA:p50 dimer. The reaction rate is fitted to obtain a peak values of ~150nM RelA:p50 dimer at 30min during canonical TNF signaling, as also modeled earlier (Basak et al. al., 2007). |
| inducible transcription of IBs and p100/*Nfkb2* by RelA:p52 dimer | 10 | tIkBa | Assumed to be identical with that of RelA:p50. This assumption is further supported by our experimental data presented in appendix figure 4B and 4C. |
| 11 | tIkBb |
| 12 | tIkBe |
| 13 | tp100 |
| degradation rate of NFkB1 mRNA | 18 | tNFkB1  | Assumed to be similar to that of p100/*Nfkb2* mRNA degradation rates. Along with the constitutive synthesis rate of tNFkB1, this degradation rate provides for nuclear induction of ~150nM RelA:p50 dimer during TNF signaling. |
| constitutive degradation of RelA:p52 | 30 | RelA:p52  | Assumed to be identical with that of RelA:p50. |
| nuclear import export rates of RelA:p52 dimer | 40 | RelA:p52  RelA:p52n | Assumed to be identical with RelA:p50. Note, nuclear import/exports are largely determined by the nuclear localization sequence present in RelA. |
| 46 | RelA:p52n  RelA:p52 |
| association and dissociation rates between NFkB and IkB | 51 | RelA:p52 + IkBa IkBa:RelA:p52 | Relative to RelA:p50 dimer, RelA:p52 binding to IBs were weak as demonstrated in Appendix-1, appendix figure 4D. Accordingly, slower association rates for RelA:p52 binding to IBs were used to reflect the dissimilar binding affinities. However, dissociation rates were assumed to be identical with that of RelA:p50-IB. |
| 52 | RelA:p52 + IkBb IkBb:RelA:p52 |
| 53 | RelA:p52 + IkBe IkBe:RelA:p52 |
| 54 | RelA:p52 + IkBd IkBd:RelA:p52 |
| 59 | IkBa:RelA:p52 RelA:p52 + IkBa |
| 60 | IkBb:RelA:p52 RelA:p52 + IkBb |
| 61 | IkBe:RelA:p52 RelA:p52 + IkBe |
| 62 | IkBd:RelA:p52 RelA:p52 + IkBd |
| constitutive degradation of IkBs and NFkB dimers within the NFkB:IkB complexes | 67 | RelA:p52:IkBa RelA:p52 | Assumed to be identical with that of the respective RelA:p50-IB complexes. |
| 68 | RelA:p52:IkBb RelA:p52 |
| 69 | RelA:p52:IkBe RelA:p52 |
|  | 70 | RelA:p52:IkBd RelA:p52 |  |
| 75 | RelA:p52:IkBa IkBa |
| 76 | RelA:p52:IkBb IkBb |
| 77 | RelA:p52:IkBe IkBe |
| 78 | RelA:p52:IkBd IkBd |
| kinase mediated degradation of IkBs bound to RelA:p52:IkB complex | 83 | NEMO+ IkBa:RelA:p52 RelA:p52 | Assumed to be identical with those of RelA:p50 dimer. Our assumption is supported by the experimental data in Appendix-1, appendix figure 4A. |
| 84 | NEMO+ IkBb:RelA:p52 RelA:p52 |
| 85 | NEMO+ IkBe:RelA:p52 RelA:p52 |
| nuclear Import Export rates of NFkB:IkB | 91 | IkBa:RelA:p52 IkBa:RelA:p52n | Assumed to be identical with RelA:p50:IB molecules. Note, nuclear import of NF-B:IB complexes are largely determined by the nuclear localization signal present in RelA, while nuclear export is controlled through IB-derived nuclear export signal. |
| 92 | IkBb:RelA:p52 IkBb:RelA:p52n |
| 93 | IkBe:RelA:p52 IkBe:RelA:p52n |
| 94 | IkBd:RelA:p52 IkBd:RelA:p52n |
| 99 | IkBa:RelA:p52n IkBa:RelA:p52 |
| 100 | IkBb:RelA:p52n IkBb:RelA:p52 |
| 101 | IkBe:RelA:p52n IkBe:RelA:p52 |
| 102 | IkBd:RelA:p52n IkBd:RelA:p52 |
| Association of p100 and dissociation of IkBd | 103 | p100+p100IkBd | association/dissociation rates were kept similar to that of NF-B-IB association/dissociation rates those captured experimentally observed 3-5 fold induction of RelA/NF-B during LTR signaling (compare appendix figure 5 and Figure-1 Figure Supplement 1). |
| 104 | IkBdp100+p100 |
| NIK-IKK1 mediated processing of p100 | 105 | NIK+p100NFkB2 | fitted based on experimentally observed time kinetics of LTR stimulated NIK induced IB/p100 degradation (Appendix-1 appendix figure 3C) and RelA/NF-B activation (Figure 1 - figure supplement 1). |