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Our description of TNF dynamics in 3T3 cells closely matched the results of the conventional 2D-model by DeLeo et al. [52], who used slightly different system of equations and made their own transformation to the variables from the paper by Chen et al. [51]. We conclude that our present model, with the exception of the TNF autocrine regulation, closely reflects the system reported by DeLeo et al. [52]. Since both models are based on the same source [51], the only discrepancy, if any, seems to be a difference in the initial conditions. The DeLeo et al. paper considered NF-B mRNA oscillations to be the fundamental trigger for the TNF oscillations, whereas the Bajar paper considers cell-to-cell TNF gradient to be the primary cause. To show the effect of autocrine regulation on the dynamics, we simulated the system with and without this mechanism. Not surprisingly, it increases the TNF oscillation period but does not affect the oscillation amplitude. On the other hand, the effect of repressing NF-B (RelA) is expected to damp both TNF and NF-B oscillations. This is verified by the results of our 2D numerical model. The steady state analysis of these equations show that at the single cell level the system is bistable for regardless of the number of underlying reactions and the total protein synthesis rate. However, the bistability disappears for larger cell population due to the system's multistability (e.g., in Fig. 6, left panels). The deterministic phase portrait and the simulated distributions of the dynamical variables for the single cell oscillation are shown in Fig. 7. The rest of the analyses were performed for one particular parameter set (shown in Table S2 in Text S1) to illustrate how the bistable behavior could be over-ridden by the stochastic effects. We will discuss results for different parameter sets for the stochastic model in the next section.

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The TNF-related activator protein (TRAF) family plays a central role in TNFR1-mediated signal transduction [28]. TRAF proteins are protein interaction modules that contain a RING domain to mediate E3 ligase activity (e.g. for ubiquitination) and a coiled-coil domain that mediates protein-protein interactions. The TRAF proteins include five members: TRAF1, TRAF2, TRAF3, TRAF5 and TRAF6. TRAF4 does not interact with TNFR1 directly [16]. TRAF2-6 proteins contain a C-terminal homology domain that directly interacts with the TNF receptor family member. TRAF1, TRAF2, TRAF3, and TRAF6 also bind to the RIP1 kinase that is required for the activation of the transcription factor NF-κB and NF-κB-dependent gene transcription [7]. TRAF6 specifically binds the non-proteolytic fragment of the TNF receptor and plays a role in TNF signalling, whereas TRAF2, TRAF3 and TRAF5 act as scaffolds, recruiting other proteins to form signalling complexes [7]. TRAF-mediated polyubiquitination of the TRAF protein is a key step in transducing signals from TNFR1 to the nucleus [7]. Up-regulation of TRAF1 and TRAF2 expression is observed in TNF-stimulated cells [14]. The TRAF1/2 knockout is highly protected against LPS-induced lethality [34]. TRAF3 mediates constitutive NF-κB signalling, which promotes inflammatory responses. TRAF4 is abundantly expressed in a number of different cell types, suggesting that it plays a role in signalling processes unrelated to TNF [34]. TRAF6 displays limited non-redundant functions in LPS-induced NF-κB activation [34]. To date, the only known ubiquitination substrates of TRAF6 are RIP1 and the p50 and p65 subunits of NF-κB [34]. RIP1 is responsible for the induction of cell death through the formation of the necrosome and the inhibition of NF-κB signalling [30, 34]. TRAF1, TRAF2, TRAF4 and TRAF6 are required for NF-κB activation [34]. 5ec8ef588b