exhibit different dynamical behaviors at the gene and protein levels. Finally, *simple unstable* when all levels do not satisfy the stability condition. A potential example of this extreme case could be cancer cells, which manifest continuous transformations at the epigenetic, gene and protein levels.

We can further draw first rudimentary conclusions on the factors that influence changes between these dynamic modes based on the linear analysis of near-stable regimes. Our set-up allows us to weigh the contributions of the different cell components against each other and determine their comparative influence using the control structure given by the type-level wiring. We show that the primary factor in regulating stability is a dynamic mode involving all cell regulatory mechanisms (cycle D in Fig. 2), in particular also epigenetics. To our current knowledge there are so far no ensemble models in the literature which integrate epigenetic influence into gene expression in a systematic fashion which separates the different regulation mechanisms on the system level. In our model we can distinguish epigenetic factors from other cell components and account for the special role of epigenetic transcription regulation in a biologically sensible and accessible way.

Our approach also allows us to investigate the influence of "microscopic" parameters such as neighborhood sizes, membership sizes and Boolean function properties. We obtain that the increase on neighborhood size, at any level, push the systems towards the unstable regime. In contrast, the increase in protein complex sizes makes the system more unstable. This mathematical result has important biological implications. It tell us that if, during the course of evolution, both the number of regulatory interactions and the protein complex sizes are increased, then the cell can remain in a nearly stable