work model Kauffman (1969, 1993). Thus, our approach contains the standard Boolean network model as a special case.

Regulation at the protein level: In the next example we study regulation on the transcription factor level Fig. 3b. As opposed to the first example, here we have proteins and protein compounds regulating transcription; this includes the assembly rule, i.e. the regime change on transcription factor level. The only non-zero matrix elements are those corresponding to arrows in Fig. 3b and the only relevant dynamic modes are the cycles $\mathrm{A}_{\mathcal{P}}$ and B in Fig. 2. The characteristic polynomial (3) now reduces to $P(\lambda) = -\lambda^2(\lambda^3 - A_{\mathcal{P}}\lambda^2 - B)$. While finding the roots of this cubic polynomial can be cumbersome, finding the stability condition is in this case straigthforward. At $\lambda = 1$ we obtain the stability condition $B + A_{\mathcal{P}} = 1$. Furthermore, since the largest eigenvalue Λ of A, $A_{\mathcal{P}}$ and B are all continuous increasing functions of the associated matrix elements of A, then $\Lambda < 1$ for $B + A_{\mathcal{P}} < 1$ and $\Lambda > 1$ when $B + A_{\mathcal{P}} > 1$. So in this case, the effective control parameter for stability is given by $\theta = B + A_{\mathcal{P}}$. In particular, assuming constant neighborhood and membership sizes and synchronous updates, from (4) and (5) we obtain

$$\theta = s_{\mathcal{G}} K_1^2 \rho_{\mathcal{P}}^{+} M_1 \rho_{\mathcal{G}}^{M_1 - 1} + s_{\mathcal{P}} K_2^2 \rho_{\mathcal{G}}^{M_1} \tag{8}$$

Notice that this formula consistently solves the following subtlety: instead of speaking of gene-gene regulation networks as in the previous example, one could actually distinguish between the gene and its products (proteins), and construct a network where single gene products regulate gene transcription. This would be a more accurate description of the biological reality, should, however, lead to the same results, since de facto we do not change any inter-