

# Supplementary information for “Variable-Depth Simulation of Most Permissive Boolean Networks” – Simulation results

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Results of the simulation of models from literature with the following parameters:

- full asynchronous, with `depth=1` and  $W = \mathbf{10}_{n-1}$
- (general) asynchronous with `depth=1` and  $W = \mathbf{1}_n$
- random walk in full MP dynamics with `depth=n` and  $W = \mathbf{1}_n$
- full MP dynamics with random depth following negative exponential distribution: for each  $d \in \{1, \dots, n\}$ , `depth=d` with probability  $1/(2^d.M)$  where  $M = \sum_{j=1}^n 1/2^j$  and  $W = \mathbf{1}_n$ .

*Cell death model* [1] with the same set of mutations as in the original article from the configuration where components `cIAP`, `ATP`, and `TNF` are active. Results are listed in Fig. 1-3.

*Bladder model* [3] without mutation (wild type) and with PI3K constitutive activation from the configuration where components `EGFR_stimulus`, `FGFR3_stimulus`, `GrowthInhibitors` are active. Results are listed in Fig. 4.

*Tumor invasion* [2] without mutation (wild type), with p53 deletion, and with NICD constitutive activation, and from four different initial conditions where components `miR200`, `miR203`, and `miR34` are always active and for all combination of `DNADamage` and `ECMicroenv` activities. Results are listed in Fig. 5-9.

## References

1. Calzone, L., Tournier, L., Fourquet, S., Thieffry, D., Zhivotovsky, B., Barillot, E., Zinovyev, A.: Mathematical modelling of cell-fate decision in response to death receptor engagement. *PLOS Computational Biology* **6**(3), e1000702 (2010). <https://doi.org/10.1371/journal.pcbi.1000702>
2. Cohen, D.P.A., Martignetti, L., Robine, S., Barillot, E., Zinovyev, A., Calzone, L.: Mathematical modelling of molecular pathways enabling tumour cell invasion and migration. *PLOS Computational Biology* **11**(11), e1004571 (2015). <https://doi.org/10.1371/journal.pcbi.1004571>
3. Remy, E., Rebouissou, S., Chaouiya, C., Zinovyev, A., Radvanyi, F., Calzone, L.: A modeling approach to explain mutually exclusive and co-occurring genetic alterations in bladder tumorigenesis. *Cancer Research* **75**(19), 4042–4052 (2015). <https://doi.org/10.1158/0008-5472.can-15-0602>

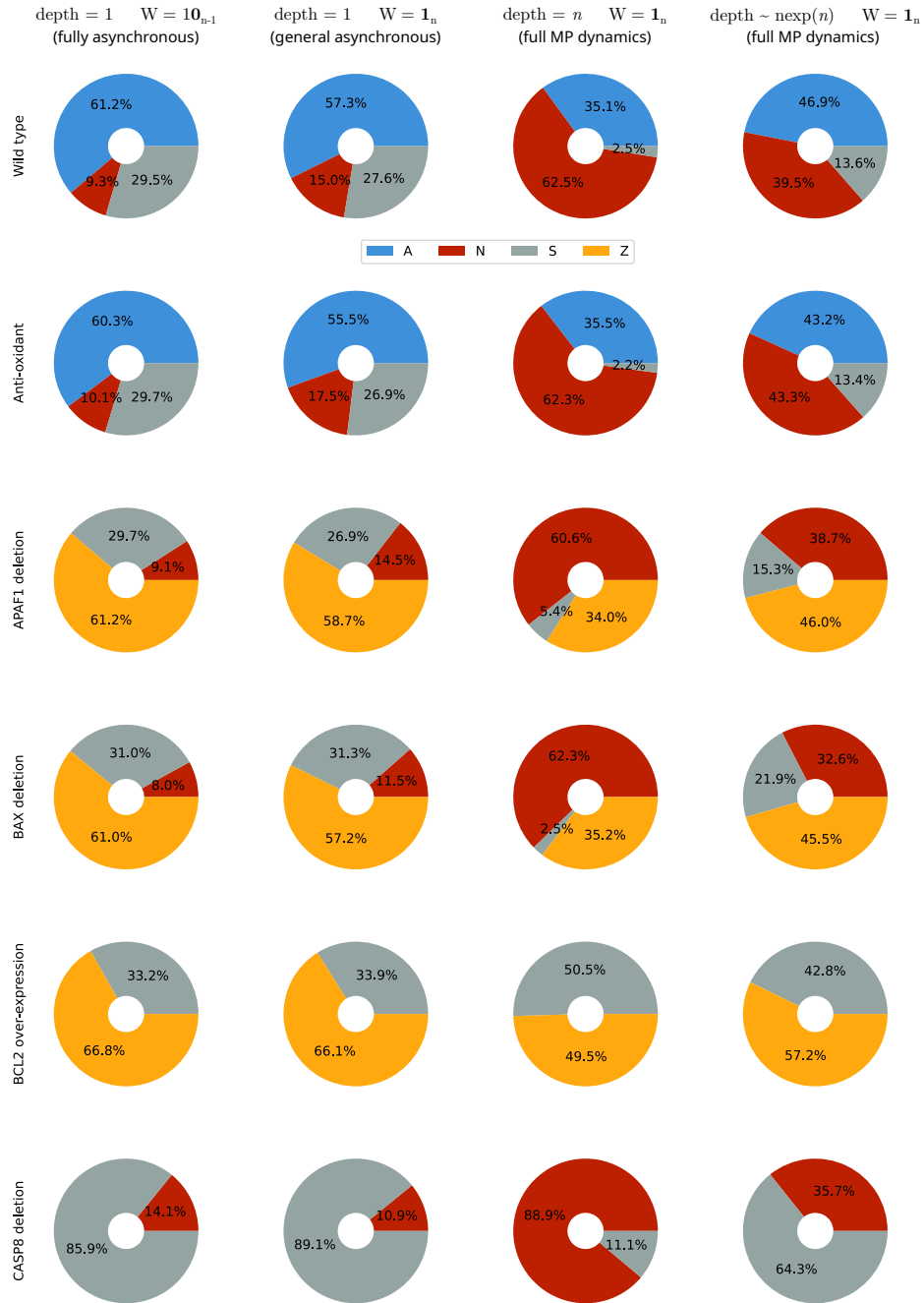


Fig. 1. Simulation results for the cell death receptor model (part 1/3)

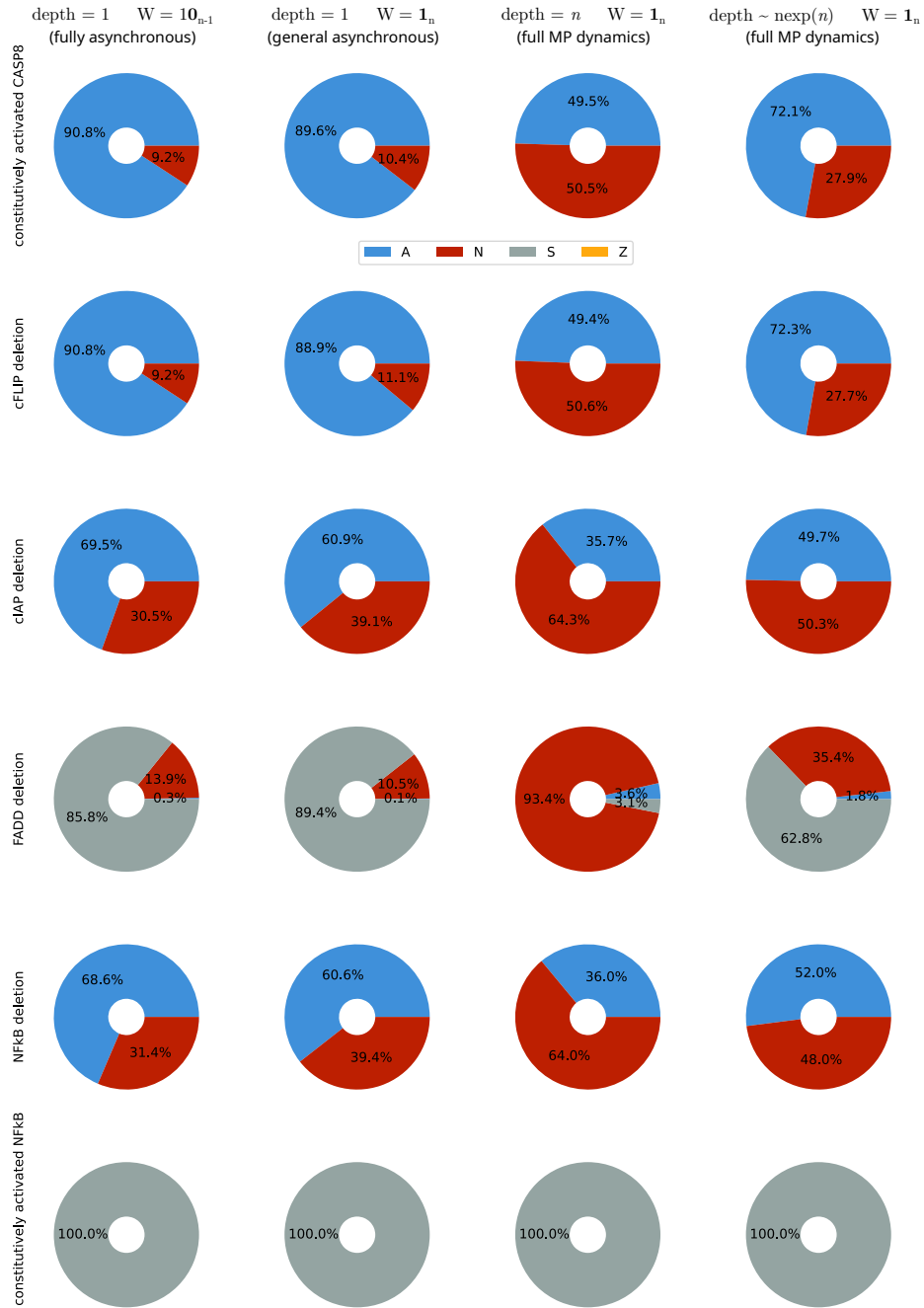


Fig. 2. Simulation results for the cell death receptor model (part 2/3)

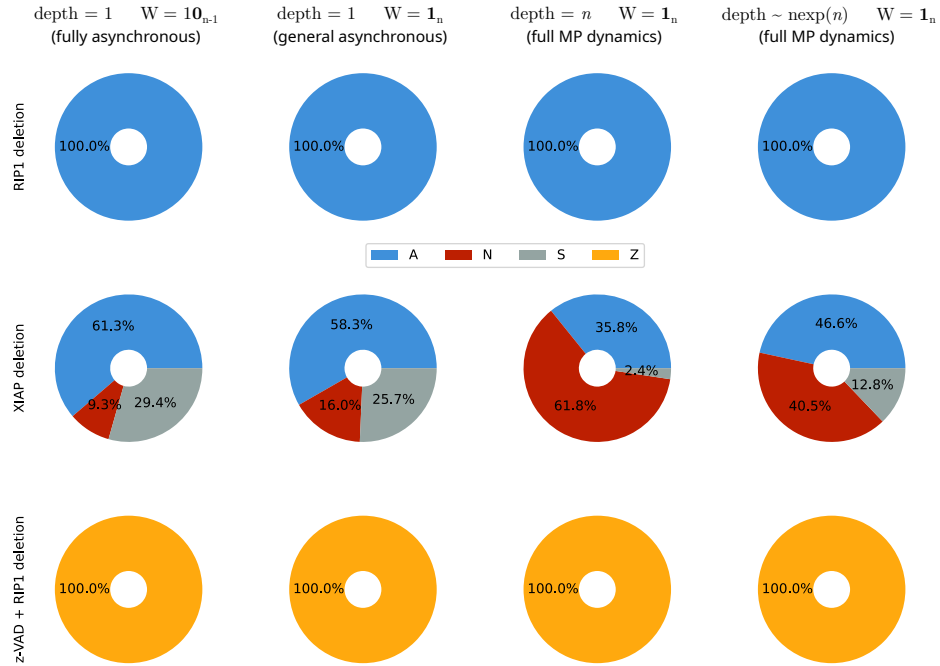


Fig. 3. Simulation results for the cell death receptor model (part 3/3)

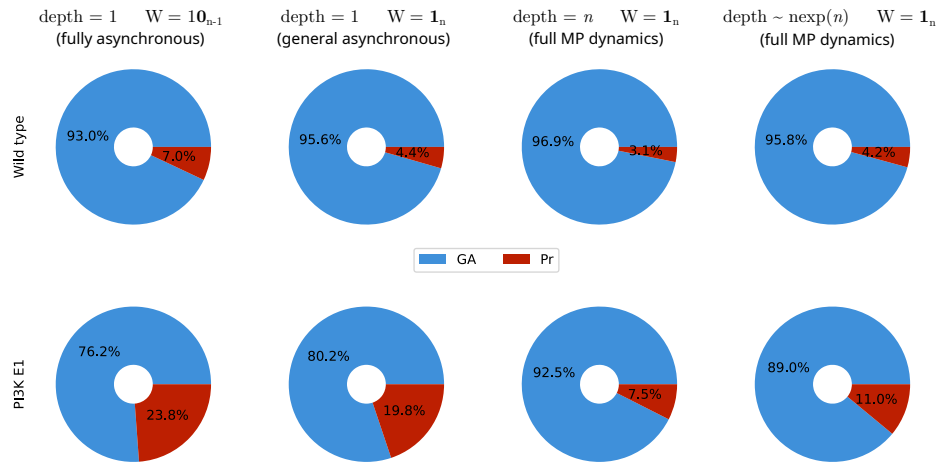
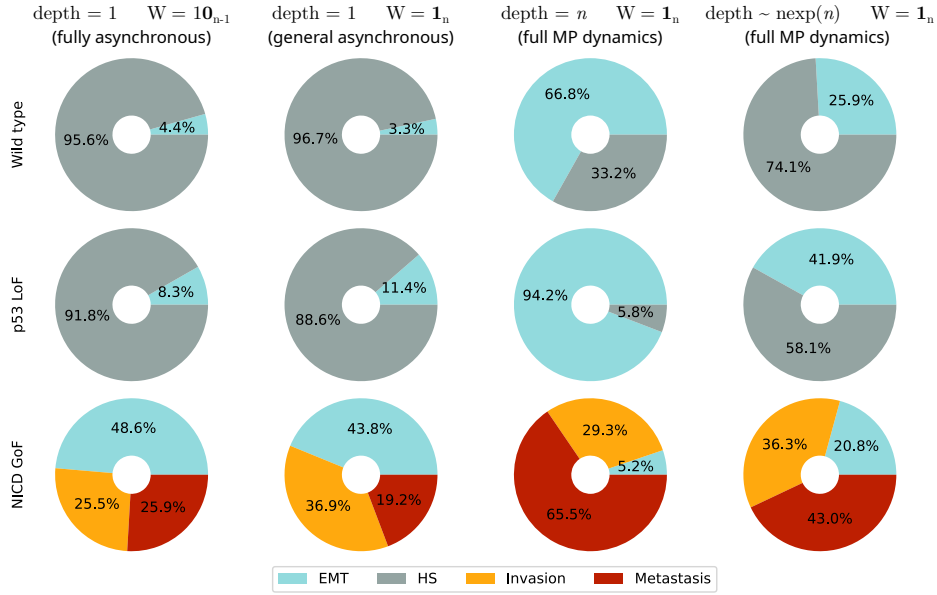
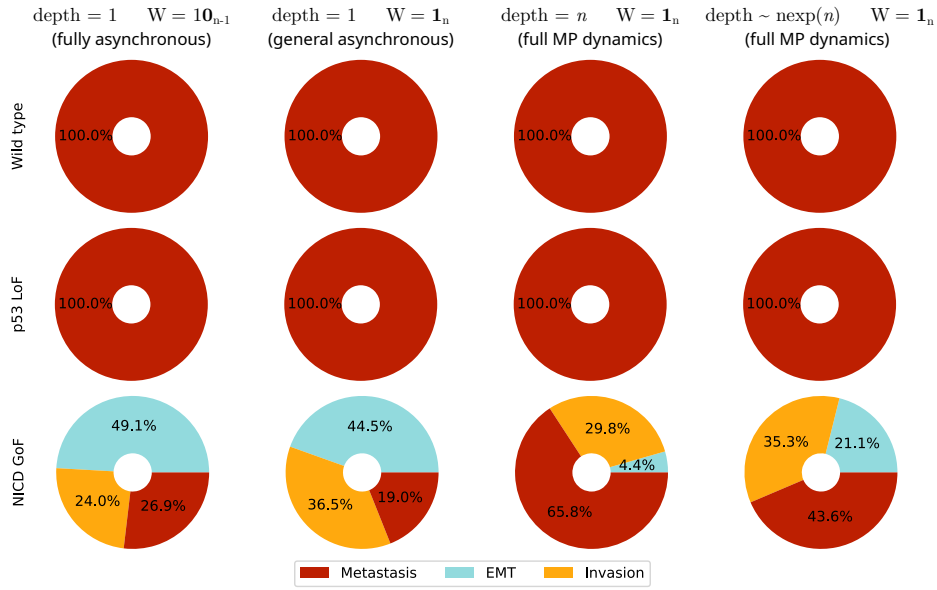


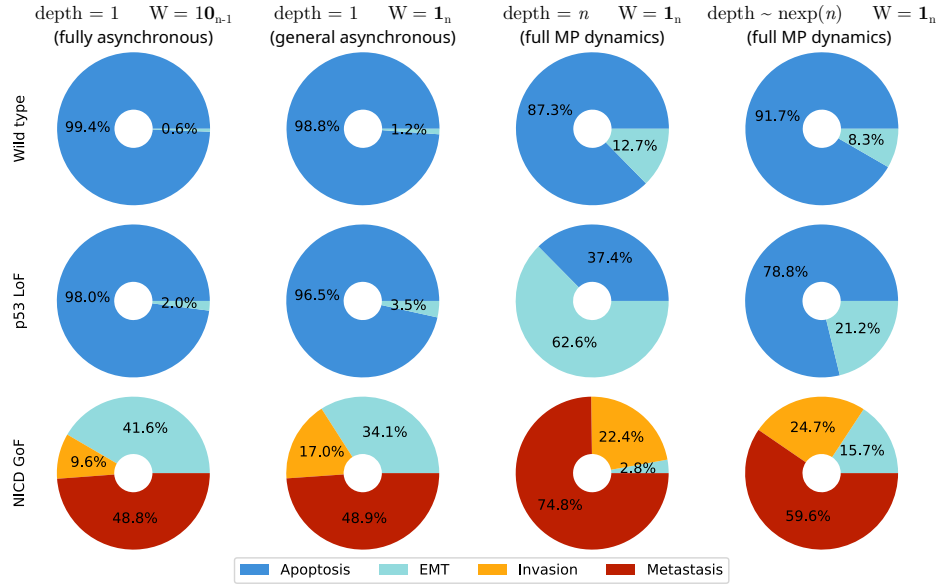
Fig. 4. Simulation results for the bladder model



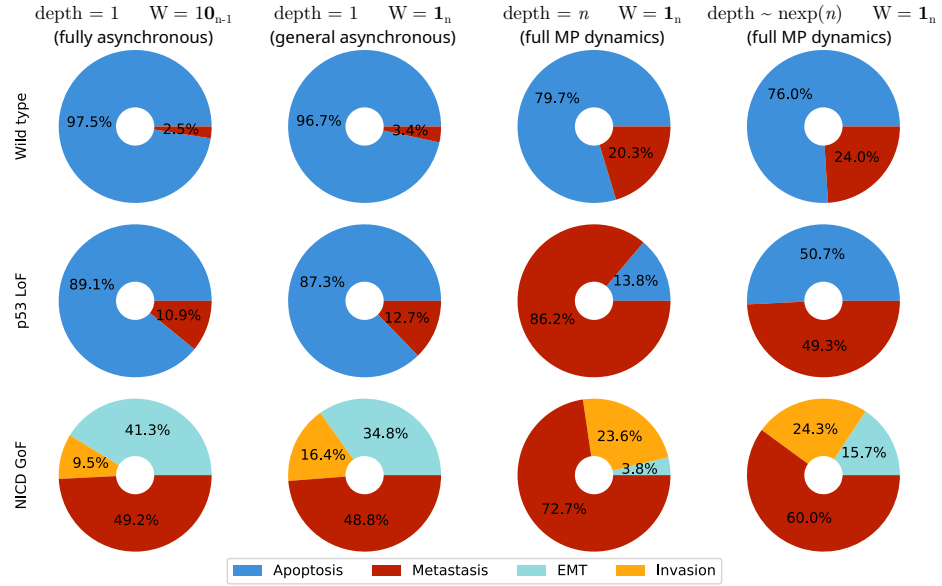
**Fig. 5.** Simulation results for the tumor invasion model from the configuration where DNadamage and ECMicroenv are inactive



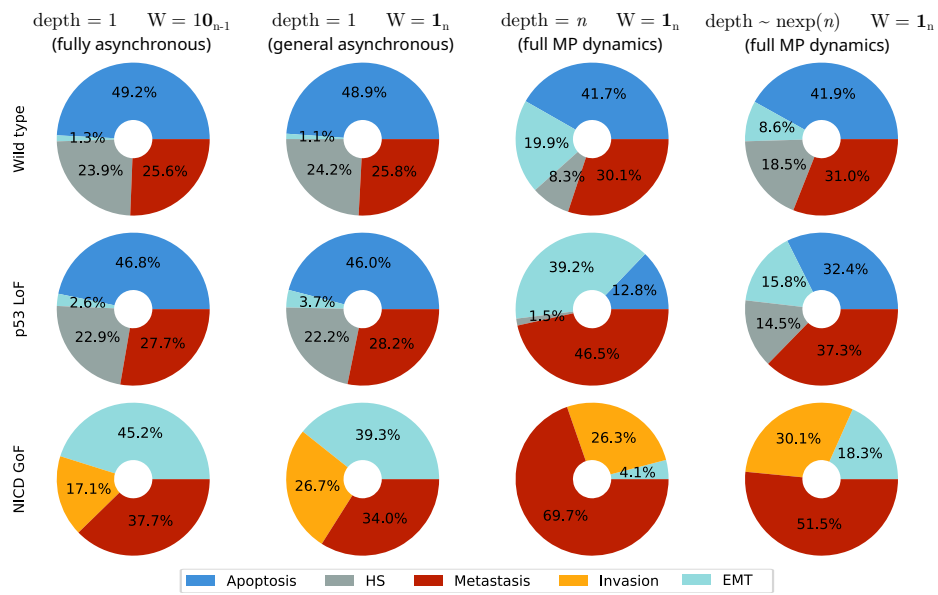
**Fig. 6.** Simulation results for the tumor invasion model from the configuration where DNadamage is inactive and ECMicroenv active



**Fig. 7.** Simulation results for the tumor invasion model from the configuration where DNAdamage is active and ECMicroenv inactive



**Fig. 8.** Simulation results for the tumor invasion model from the configuration where DNAdamage and ECMicroenv are active



**Fig. 9.** Mean of the simulation results for the tumor invasion model from the four initial configurations