Stochastic spreading models reproduce embolism propagation dynamics in angiosperm xylem networks of vessels connected by bordered pits. Supplementary information.

Supplementary figures

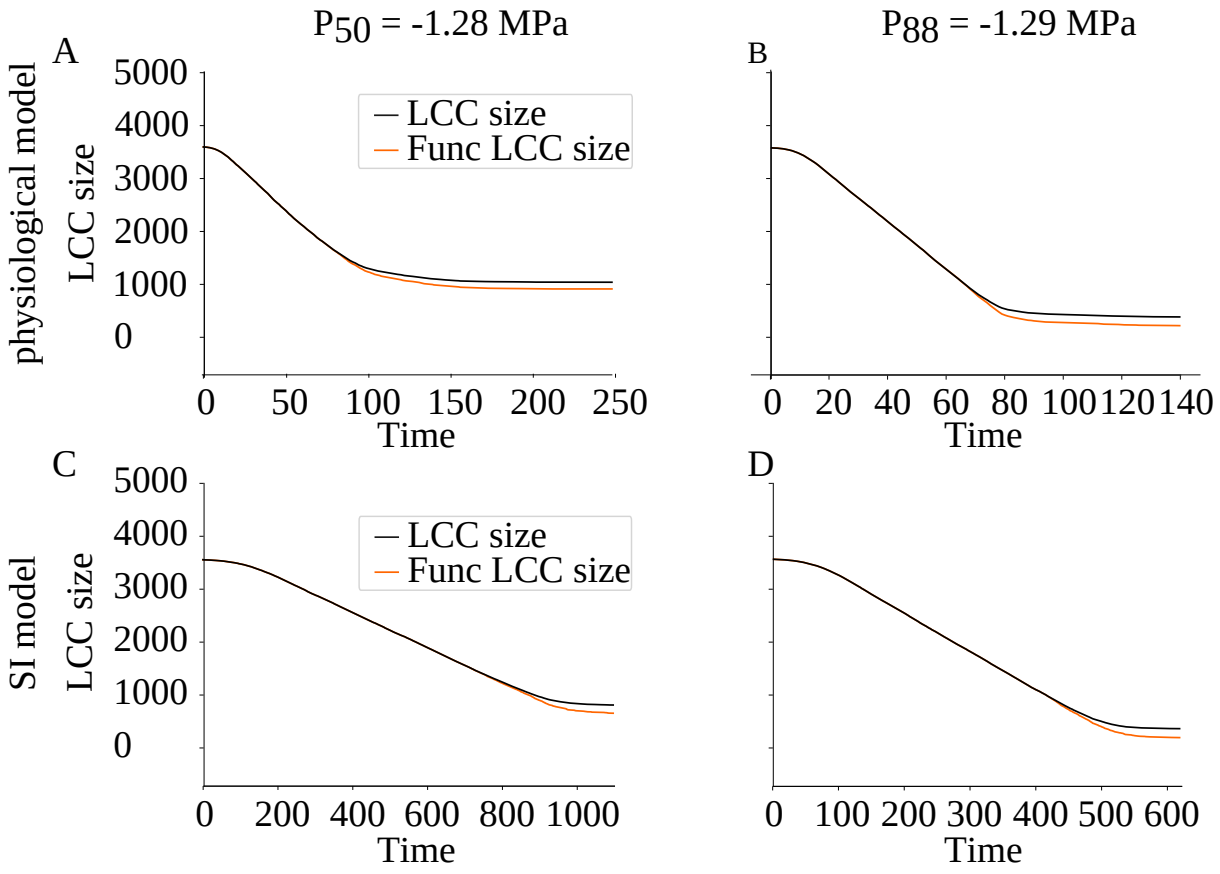

Fig. S1: Largest connected component became nonfunctional at more negative xylem pressures. Behaviour of the LCC size (black) and functional LCC size (cyan) as a function of simulation time for the physiological (A, B) and SI (C, D) models at P_{50} (A, C) and P_{88} (B, D). At P_{12}, LCC was always functional.

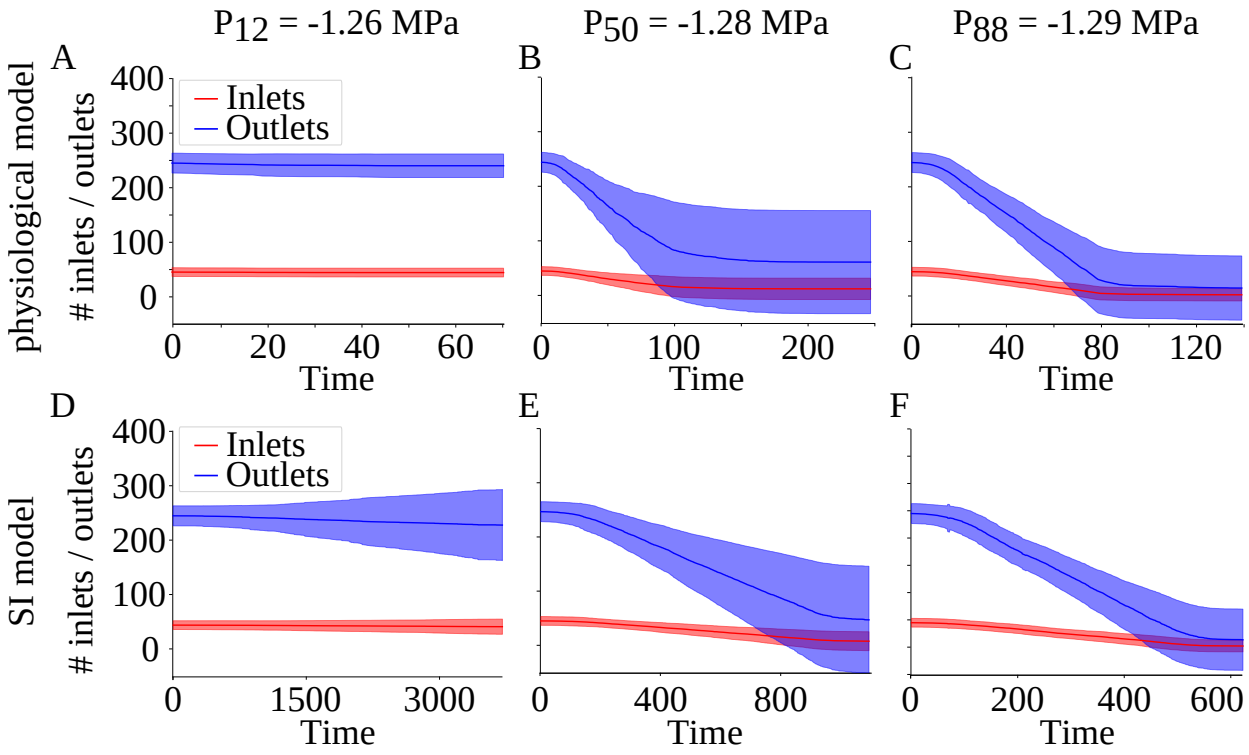

Fig. S2: Behaviour of the average number of inlet and outlet vessels per component as a function of simulation time steps for the physiological (A, B, C) and SI (D, E, F) models at P_{12} (A, D), P_{50} (B, E), and P_{88} (C, F).

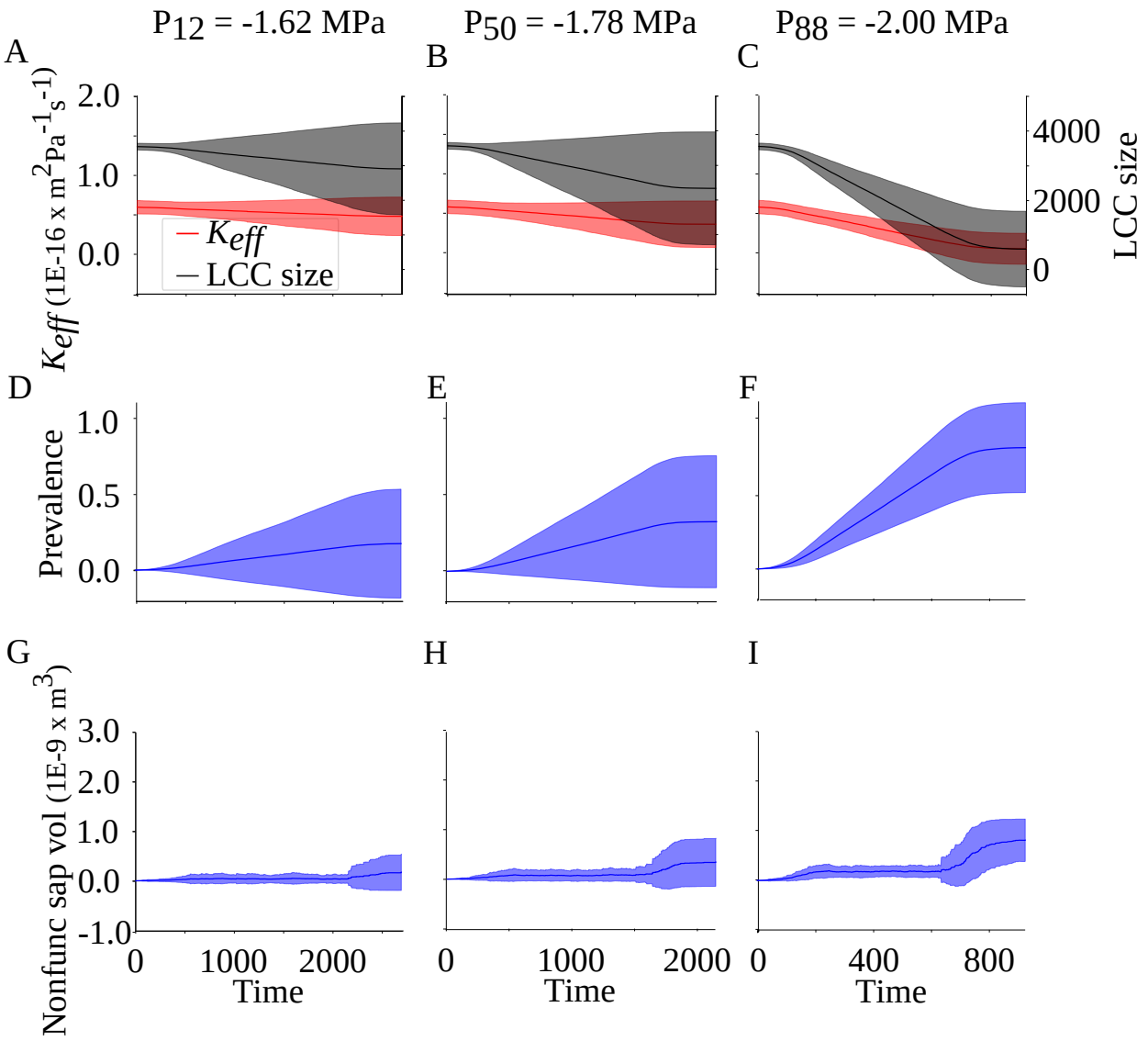

Fig. S3: The SI spreading model fitted to empirical data allowed estimating the behaviour of network properties during embolism spreading based on an empirical vulnerability curve. K_{eff} and the largest connected component (LCC) size (A, B, C), prevalence (D, E, F), and nonfunctional sap volume (G, H, I) are monitored for P_{12} (A, D, G), P_{50} (B, E, H), and P_{88} (C, F, I). The full lines and shadowed areas show mean and standard deviation across simulations, and the x axis shows the number of simulation time steps.