Optimal responsiveness and information flow in networks of heterogeneous neurons

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Cerebral cortex is characterized by a strong neuron-to-neuron heterogeneity, but it is unclear what consequences this may have for cortical computations, while most computational models consider networks of identical units. Here, we study network models of spiking neurons endowed with heterogeneity, that we treat independently for excitatory and inhibitory neurons. We find that heterogeneous networks are generally more responsive, with an optimal responsiveness occurring for levels of heterogeneity found experimentally in different published datasets, for both excitatory and inhibitory neurons. To investigate the underlying mechanisms, we introduce a mean-field model of heterogeneous networks. This mean-field model captures optimal responsiveness and suggests that it is related to the stability of the spontaneous asynchronous state. The mean-field model also predicts that new dynamical states can emerge from heterogeneity, a prediction which is confirmed by network simulations. Finally we show that heterogeneous networks maximise the information flow in large-scale networks, through recurrent connections. We conclude that neuronal heterogeneity confers different responsiveness to neural networks, which should be taken into account to investigate their information processing capabilities.

Results
We first investigate the responsiveness of heterogeneous networks of excitatory and inhibitory neurons numerically, and next we use a theoretical approach to understand the mechanisms underlying responsiveness.
Optimal responsiveness for heterogeneous networks. We studied networks of sparsely connected excitatory and inhibitory spiking neurons (see "Methods" for details). Each neuron receives afferent excitatory spike trains at a frequency $v_{ext}(t)$. To characterise the responsivity of the network to external stimulation, we considered $v_{ext}(t)$ composed by a time constant baseline value $v_0$ and a time variation of amplitude $A$ (see "Methods", Input in Fig. 1a). The network responds to the stimulation by increasing its population spiking activity (see Output in Fig. 1a). The network responsiveness $R$ can be estimated as the total amount of evoked spikes by the whole network (see the blue area in the Output of Fig. 1a). We compared homogeneous networks to networks constructed from measurements in the adult human and mouse brain from the Allen Brain Atlas$^{32}$. In Fig. 1b, we report the histogram of the resting membrane potential $E_L$ measured experimentally from excitatory (blue, top panels) and inhibitory neurons (red, lower panels) in human and mouse cortical layers. By calling $E_L^E$ the average value of $E_L$, one can see a heterogeneous distribution of the re-scaled resting potential $E_L = E_L/E_L^E$. The distribu-
Figure 2. Excitatory and inhibitory heterogeneity determines network responsiveness. (a) Responsiveness \( R \) as a function of heterogeneity of excitatory (\( \sigma_E \)) and inhibitory (\( \sigma_I \)) neurons for networks displaying relatively high levels of spontaneous activity (input baseline \( V_0 = 1.5 \) Hz and input amplitude \( A = 1 \) Hz). (b) Responsiveness \( R \) for networks with a lower level of spontaneous activity (input baseline \( V_0 = 0.4 \) Hz and input amplitude \( A = 0.6 \) Hz). Different markers correspond to heterogeneity values estimated from Human cortex layer 2, 3 (white circle), Human cortex layer 5, 6 (grey circle), mouse cortex layer 2/3 (grey star) and mouse cortex layer 6 (white diamond) estimated from Allen Brain database\(^{12}\), see Fig. 1.

Responsiveness depends on heterogeneity in excitatory and inhibitory neurons. To explore the combined effect of heterogeneity in both excitatory and inhibitory populations we calculated the responsiveness \( R \) as a function of both \( \sigma_E \) and \( \sigma_I \) (see Fig. 2a). Apart from fluctuations due to the endogenous noise in network dynamics, we observe a region of optimal responsiveness for heterogeneous networks (warm colors in Fig. 2).

Importantly, the experimental values of heterogeneity (Fig. 1b) fall close to the predicted optimal region of network responsiveness (see symbols in Fig. 2a). More specifically, we observe that, if heterogeneity in inhibitory neurons is very high (and thus responsiveness very low), increasing heterogeneity in excitatory neurons permits to increase responsiveness \( R \). In another way, both heterogeneity cooperate to increase network responsiveness.
Figure 3. Enhanced responsiveness corresponds to regions closer to instability. (a) The largest stability Lyapunov exponent \( \lambda \) (real part) of asynchronous dynamics as a function of the heterogeneity of inhibitory neurons (\( \sigma_I \)). Different colors indicate different parameters of the baseline external drive and the strength of excitatory-excitatory quantal conductance (\( \nu_0, Q_{EE} \)), i.e. black (1.5 Hz, 1.5 nS), red (3 Hz, 1.5 nS), blue (2 Hz, 1.5 nS) and orange (3 Hz, 1.65 nS). Symbols are located at the value of \( \sigma_I \) for which the responsiveness \( R \) is maximum (same color code as continuous line). Different symbols indicate different amplitudes \( A \) of the input, diamond (\( A = 0.1 \) Hz), star (\( A = 0.5 \) Hz) and dot (\( A = 1 \) Hz). (b) The larger stability Lyapunov exponent \( \lambda \) of asynchronous dynamics as a function of the heterogeneity of inhibitory neurons (\( \sigma_I \)) and the strength of excitatory-excitatory quantal conductance \( Q_{EE} \) for a baseline external drive \( \nu_0 = 1.5 \) Hz. The dotted (diamond) line is the responsiveness \( R \) for an input amplitude \( A = 0.1 \) Hz (\( A = 1 \) Hz) and \( Q_{EE} = 1.5 \) nS (as in Figs. 1 and 2). Such responsiveness has been properly rescaled on the y-axes (i.e. multiplied by an ad-hoc factor) in order to fit in the image.

Optimal responsiveness comes from pushing the network at the edge of a dynamical transition. To determine the mechanism at the origin of the enhanced responsiveness due to neuronal heterogeneity, we developed a mean-field approach explicitly taking into account diversity. We started from a mean-field model previously introduced for homogeneous neural populations. We extend this approach to heterogeneous systems by employing a technique, called heterogeneous mean field (HMF), successfully applied previously to model networks with heterogeneous connectivity and extended here to networks with heterogeneous cell properties (see "Methods" section). This procedure describes the collective dynamics of large sparsely connected networks of heterogeneous neurons with a relatively simple three dimensional model. This HMF model predicts the time evolution of excitatory neurons population firing rate \( r_E(t) \) and of inhibitory neurons population firing rate \( r_I(t) \). By comparing the prediction of the HMF model to estimations from direct numerical simulations of a large network of neurons, we observed a very good agreement, that can be appreciated from the prediction of the response \( R \) for different parameter setups (other parameters are also studied in next sections), an heterogeneous network corresponds to an optimal responsiveness. Moreover, the optimal responsiveness appears for values of heterogeneity close to experimental amounts. In order to test the robustness of this observation, we have verified that changing network realization and initial conditions the results are the same (see Supplementary Information).
region where the asynchronous state becomes marginally stable (red region, $\lambda$ close to zero). By superimposing the responsiveness $R$ from Fig. 1d (dotted line, in this case for fixed $Q_{EE} = 1.5$ nS), we can see that the value of $\sigma_I$ for which the responsiveness is maximum corresponds to a network that is closer to the transition point where the asynchronous regime becomes unstable.

**Heterogeneity can induce new dynamical regimes.** Finally, to determine what the unstable regions correspond to, we investigated heterogeneous networks both numerically and theoretically using the mean-field model. Figure 4a shows that, for intermediate amount of heterogeneity the asynchronous state can be unstable (blue dashed line in panel a). This dynamical phase corresponds to an oscillatory regime at the level of the whole network, arising from a super-critical Hopf bifurcation where the amplitude of the limit cycle (red lines in panel a) smoothly increases in function $\sigma_I$. In panel b of Fig. 4 we observe that this oscillating synchronous regime (present whenever the asynchronous state is unstable, $\lambda > 0$) appears only for heterogeneous networks ($\sigma_I > 0$) but the location of the synchronous region depends on the average value of neurons’ resting potential $E^*_L$ (notice that in panel a of Fig. 4 we used $E^*_L = -70$ mV and in Figs. 1, 2 and 3 we used $E^*_L = -65$ mV). In order to verify these theoretical predictions, in panel c we finally perform numerical simulation of the neuronal network where we observe that the limit cycle corresponds indeed to collective network oscillations (middle panel) that disappear in homogeneous or too heterogeneous setups (higher and lower panels). This shows that not only heterogeneity can induce higher responsiveness, but it can also induce new dynamical regimes. The dynamics of heterogeneous networks is therefore richer.

**Heterogeneity boosts information flow in large-scale networks.** To determine if the enhanced responsiveness applies not only to external inputs but also to the flow of information through recurrent connectivity, we considered large-scale networks where each unit is a population of heterogeneous neurons. We considered a two dimensional lattice of mean-field models, that are interconnected to each other via Gaussian connectivity profiles (see Fig. 5a and “Methods” section). According to anatomical connectivity estimates, inhibitory neurons have a short-range connectivity (red curves in Fig. 5a) at variance with excitatory connectivity (blue curves in Fig. 5a) \cite{Vogels2005}. Importantly, we integrate distance-dependent propagation delays due to the finite velocity of axonal conduction of action potentials (we considered here an axonal conduction velocity of 0.3 m/s, see “Methods” section).

We stimulated this large-scale model by an external input (see “Methods” section) and we compared a model with locally heterogeneous neurons (each node is modeled with an heterogeneous mean field with $\sigma_I = 0.15$) versus the same model with locally homogeneous neurons (each node is modeled with an homogeneous mean field with $\sigma_I = 0$). As it can be appreciated from Fig. 5b, both the heterogeneous and the homogenous model respond with an activation that propagates in space as a wave. We observed that the intensity of the wave is much larger in the model with locally heterogeneous networks. This is a direct consequence of the enhanced responsiveness of each node. But does local heterogeneity also impacts the spatial extent of the wave through recurrent connections? To answer this question we compared the homogeneous and the heterogeneous system...
by normalising the firing rate activity with its maximum in space and time, in order to highlight the spatio-temporal profile of the response. As it can be observed from panels c and d in Fig. 5, the homogeneous and the heterogeneous systems have different spatio-temporal profiles, indicating that local heterogeneity influences not only the intensity of the response to the stimulus, but also its spatio-temporal pattern through recurrent interactions. Remarkably the heterogeneous system has much longer-range spatial propagation of activity. More specifically, for an optimal amount of heterogeneity we observed a much enhanced propagation of the activity through the network (red curve in Fig. 5e), compared with homogeneous and highly heterogeneous networks.

These results show that an optimal amount of local heterogeneity has a double effect at large scale. First, it amplifies the intensity of the response to the external input (Fig. 5b, a consequence of local enhanced responsiveness) and, second, enhanced responsiveness also applies to recurrent inputs, which results in a long range propagation of activity across the network (Fig. 5e).

Discussion
In conclusion, we report here four findings. First, we have found that the heterogeneity of inhibitory neurons, which has been well documented experimentally13-16, optimises the responsiveness of spontaneously active networks to external stimuli. There appears a resonance peak as a function of the level of heterogeneity. We have here studied the heterogeneity in neurons’ resting potential, that is experimentally quantified (see Fig. 1b and Supplementary Information) and determines neurons’ proximity to firing threshold (and thus neurons’ excitability). On the other side our results do not limit to only this cellular parameter. We have indeed found, both theoretically and by performing network simulations, that an optimal responsiveness appears also for intermediate heterogeneity in neurons’ leakage conductance, while almost no effect is observed for heterogeneity in neurons’ membrane capacitance (see Supplementary Information).

A similar effect of diversity-induced resonance was previously observed in excitable or bistable systems41–45, where heterogeneity creates active excitation clusters which were absent in the quiescent homogeneous system. We showed here a different type of heterogeneity, that of inhibitory neurons, can induce optimal population responses. More generally, we found that the heterogeneity of both excitatory and inhibitory populations induces optimal responsiveness in spontaneously active sparse networks with irregular firing activity. Importantly, we found that the level of heterogeneity measured experimentally across different cortical layers and species (Human...
and Mouse) corresponds to the resonance peak, which suggests that cortical networks may have naturally evolved towards optimal responsiveness by adjusting their heterogeneity. Moreover, while several studies reported that heterogeneity can enhance coding in uncoupled networks and decrease neuronal correlations, we report here that a higher input–output population response is linked to an increased tendency to synchronization in heterogeneous networks. The coding capabilities of neural networks will therefore be largely affected by neuronal heterogeneity, which opens interesting perspectives for future studies.

Second, we found that the enhanced responsiveness of heterogeneous networks is paralleled with a decreased stability of the spontaneous activity regime. To obtain this result, we designed a mean-field model that explicitly includes heterogeneity, and which can capture this diversity-induced resonance. This new mean-field formulation keeps track of microscopic complexity, compared to traditional mean-field approaches which implicitly assume homogeneous systems and would not predict the correct responsiveness. This also shows that responsiveness must be understood using the knowledge of the spontaneous activity of the network—and in particular its level of stability. The relation between instability and responsiveness also constitutes a promising subject to further explore in the future.

In this work we have studied heterogeneity in the intrinsic proprieties of neurons, but it is known that heterogeneity can be found also in the structure of connections (say for example in neurons’ in-degrees). Structural Heterogeneity (i.e. in the topology of connections) can have a strong impact in network dynamics. Our mean field approach is general and can be employed in the future to study the impact of heterogeneity in neurons’ connectivity.

Third, we have shown that neuronal heterogeneity is not only important for responsiveness, but also can induce new dynamical regimes. Using the mean-field models, we could predict a transition to a sparsely synchronous collective oscillation regime, which was confirmed by network simulations. This type of diversity-induced oscillations reminds some aspects found in noise-induced transitions in dynamical systems. Whether the effects of heterogeneity could be considered as analogous to the effect of noise (‘quenched noise’) in neural networks is also an interesting direction for future studies.

Finally, we have found that the enhanced responsiveness enables activity to propagate much easier in large-scale heterogeneous networks. The enhanced response not only applies to the stimulus, but also it applies to information flow through recurrent excitatory inputs. As a result, comparing heterogeneous to homogeneous networks, a given stimulus produces a larger local response, and in addition, this response also propagates to a larger spatial extent because the effect of recurrent excitatory inputs is also amplified by heterogeneity. Thus, we conclude that heterogeneous networks can provide activity propagation at a level much superior compared to a homogeneous system, and thus will necessarily better propagate information and make it available to larger brain areas.

Methods

Network model. We examined networks of excitatory and inhibitory neurons connected through conductance-based synapses. We used networks of $N = 10,000$ neurons, 80% of excitatory ($N_E = 0.8 N$) and 20% of inhibitory ($N_I = 0.27 N$) neurons. The membrane potential $V_i$ of each neuron evolves according to the Adaptive Exponential integrate and fire model (AdExp):

$$C_m \dot{V}_i = g_L (E_L - V_i) + g_L e^{rac{V_i - v_{r}}{\Delta}} + I_i^s - w_i$$

(1)

$$\tau_w \dot{w}_i = -w_i + b \sum_{j} \delta(t - t_i^{sp})$$

(2)

where $C_m = 200 \text{pF}$ is the membrane capacitance, $g_L = 15 \text{nS}$ the leakage conductance, $v_{r} = 50 \text{mV}$ the effective threshold and $\Delta$ defines the action potential rise ($\Delta = 0.5 \text{mV}$ for inhibitory neurons and $\Delta = 2 \text{mV}$ for excitatory neurons). The adaptation current $w_i$ increases of an amount $b = 60 \text{nS}$ at each spike emitted by neuron $i$ at times $t_i^{sp}$ and has an exponential decay with time scale $\tau_w = 500 \text{ms}$. Only excitatory neurons have spike frequency adaptation, while for inhibitory neurons $b = 0$. The current $I_i^s$ is the current received by neuron $i$ from other neurons in the network. We consider a random graph where each couple of neurons is connected with probability $p = 0.05$. By calling $t_i^{sp}$ the ensemble of spiking times of neuron $i$ we have, for an excitatory post-synaptic neuron $i$:

$$I_i^s = g_{EE}^E (V_i - E_E) + g_{EI}^E (V_i - E_I)$$

(3)

$$\tau_s g_{EE}^E = -g_{EE}^E + Q_{EE} \sum_{t_j^{sp} \in (E)} \delta(t - t_j^{sp})$$

(4)

$$\tau_s g_{EI}^E = -g_{EI}^E + Q_{EI} \sum_{t_j^{sp} \in (I)} \delta(t - t_j^{sp})$$

(5)

where $E_{EE}$ is the reversal for excitatory ($E_E = 0 \text{mV}$) and inhibitory synapses ($E_I = -80 \text{mV}$), $\tau_s = 5 \text{ms}$ the synaptic decay time and $Q_{EE}$ ($Q_{EI}$) is the interaction strength of excitatory (inhibitory) synapses to excitatory neurons. The same equations (with $g_{EE}^E \rightarrow g_{EE}^I$, $g_{EI}^E \rightarrow g_{EI}^I$, $Q_{EE} \rightarrow Q_{EE}$ and $Q_{EI} \rightarrow Q_{EI}$) apply for inhibitory
post-synaptic neurons. We fix $Q_{EI} = Q_{IE} = 5 \, \text{nS}$ and $Q_{EE} = Q_{EE} = 1.5 \, \text{nS}$ (we employed different values of $Q_{EE}$ in Fig. 3 and in Fig. 4, see the relative caption).

Each neuron receives an external Poissonian train of excitatory spikes at a rate $v_{ext}$. The value of $v_{ext}$ determines the amount of ongoing spontaneous activity in the network. In order to study the response to external stimuli we considered a time varying $v_{ext}(t)$ of the form $v_{ext}(t) = v_0 + Ae^{-\frac{(t-t_0)^2}{\sigma_{E,I}^2}}$, where $A$ is the input amplitude, $t_0$ the time when input is maximum and $T = 50 \, \text{ms}$ measures the duration of the input. We have employed $v_0 = 1.5 \, \text{Hz}$ in all the numerical simulations, apart from Figs. 2b and 3 (see Figure caption for details). In Fig. 1 we employed $t_0 = 6 \, \text{s}, v_0 = 1.5 \, \text{Hz}$ and $A = 1 \, \text{Hz}$. The responsiveness $R$ is estimated by computing the amount of spikes of excitatory neurons while the input is on (i.e. between $t = t_0 - 3T$ and $t = t_0 - 3T$) minus the baseline activity (average of excitatory spike rate for $A = 0$). Responsivity $R$ is estimated by averaging over 20 different repetitions of this procedure.

To model heterogeneity, we considered a Gaussian distribution of the resting potential $E_I$, of inhibitory (excitatory) population $N(\tilde{E}_I, \sigma_{E,I})$ with average $\tilde{E}_E$ and standard deviation $\sigma_{E,I}$. We considered $\tilde{E}_E = E_E = -65 \, \text{mV}$ if not stated otherwise (e.g. we used different values of $E_E$ in the inset of Fig. 1d and in Fig. 4). The re-scaled standard deviation $\sigma_{E,I}/\tilde{E}_E$ is the main parameter to quantify heterogeneity. The same definitions apply for heterogeneity in other parameters (see Supplementary Information for $g_L$ and $C_m$).

**Mean field model.** In the homogeneous case a mean field model for this network has been recently developed\(^\text{37}\). By employing a Markovian approximation over a time scale $\tau = 15 \, \text{ms}$ and by considering a sufficiently slow time scale for the dynamics of adaptation $\tau_w$, mean field equations read:

\[
\tau \dot{r}_I = F_I(r_E + v_{ext}, r_I) - r_I \tag{6}
\]

\[
\tau \dot{r}_E = F_E(r_E + v_{ext}, r_I, W) - r_E \tag{7}
\]

\[
\tau_w \dot{W} = -W + br_E, \tag{8}
\]

where $r_E$ ($r_I$) is excitatory (inhibitory) neurons population firing rate, $W$ is excitatory neurons average spike frequency adaptation, $F_I(v_E, v_I)$ and $F_E(v_E, v_I, W)$ are the transfer functions of inhibitory and excitatory neurons, respectively. They measure the stationary firing rate of one neuron when receiving an excitatory (inhibitory) Poissonian spike train at a rate $v_E$ ($v_I$).

In the case of heterogeneous inhibitory neurons, a parameter $x$, say neurons reversal potential $E_L$, is distributed according to a probability density function $P(x)$. In the limit of large networks we can decompose inhibitory neurons in classes, each one characterized by a parameter $x$ and by its own transfer function $F_I(x, r_E + v_{ext}, r_I)$. We indicate with $r^x_I$ the firing rate of the class of neurons with parameter $x$. The whole population rate is $r_I = \int dx P(x)r^x_I$. The equations are closed by a self-consistency equation for the mean input received by one neuron and we need to replace Eq. (6) with

\[
\tau \dot{r}_I = \int dx P(x)F_I(x, r_E + v_{ext}, r_I) - r_I. \tag{9}
\]

Notice that the model still stays three dimensional but keeps track of the distribution $P(x)$ of heterogeneity. In the case $P(x) = \delta(x - x_0)$ we recover the homogeneous model.

The same procedure applies for excitatory neurons. Nevertheless, in this case each class $x$ is characterised by its own adaptation variable $w_x$. For each class with parameter $x$ we get:

\[
\tau \dot{w}_x = F_E(w_x, r_I, w_x, r_E) - r_E \tag{10}
\]

\[
\tau_w \dot{w}_x = -w_x + br_E. \tag{11}
\]

In this case the population quantities can be written as $W = \int P(x)w_x$ and $r_E = \int P(x)r^x_E$. These equations can be solved by sampling $P(x)$ and we found that a sampling of around 50 points gives an accurate precision. In our work we employ the mean field for heterogeneous excitatory neurons only in Fig. 1d, for which we employed a sampling of 50 points.

Nevertheless, it is possible to reduce the dimensionality of this model by making the hypothesis that $w_x$ is slow enough. The stationary solution is $w_x = \bar{w}_x F_E$. In order to follow the dynamics of adaptation we evolve the population variable $W$ as $\tau_w W = -W + \bar{w}_x F_E$ and estimate the adaptation of each class from the equation $w_x = W F_E/W = (W/R) \bar{w}_x F_E(r_E + v_{ext}, r_I, w_x)$. In this way the model stays three dimensional. Preliminary results indicate that this is a good approximation for the population dynamics in the heterogeneous case.

Finally, the estimation of neuron transfer functions for the AdExp model $F_I(x, r_E, r_I, W)$ is done through a semi-analytical fitting procedure\(^\text{37}\) (see Supplementary Information).

**Spatially extended model.** To model large-scale networks, we considered a square lattice of length $L = 20 \, \text{mm}$ composed of $M \times M$ nodes, we employed $M = 100$ in the simulations of Fig. 5. Each node is modeled as a mean field model. In the limit of large $M$ the dynamics of each node at a location $(x, y)$ follows:
\[
\tau \frac{\partial r_E(x, y)}{\partial t} = -r_E(x, y) + F_E(v_E(x, y) + v_{\text{inp}}(x, y, t), v_I(x, y), W(x, y)) \\
\tau \frac{\partial r_I(x, y)}{\partial t} = -r_I(x, y) + \int dzP(z)F_I(v_E(x, y) + v_{\text{inp}}(x, y, t), v_I(x, y))
\]

\[
\tau \frac{\partial W(x, y)}{\partial t} = -W(x, y) + br_E(x, y),
\]

where \(P(z)\) is the distribution of heterogeneity, i.e., a Gaussian distribution with average \(\bar{E}_I\) and rescaled standard deviation \(\sigma_I\), and \(v_E(x, y) (v_I(x, y))\) is the excitatory (inhibitory) input incoming in \((x, y)\) from the other lattice locations, i.e.

\[
v_E(x, y) = \int dx_1 \int dy_1 G_E(x - x_1, y - y_1)r_E(x_1, y_1, t - \frac{d}{v_c})
\]

\[
v_I(x, y) = \int dx_1 \int dy_1 G_I(x - x_1, y - y_1)r_I(x_1, y_1, t - \frac{d}{v_c}),
\]

where \(d = \sqrt{(x-x_1)^2 + (y-y_1)^2}\) is the distance between two points in the lattice and \(G_E (G_I)\) is the excitatory (inhibitory) connectivity in space. We consider a Gaussian connectivity in both directions with standard deviation \(\Delta_E = 2 \text{ mm}\) (\(\Delta_I = 1 \text{ mm}\)) for excitatory (inhibitory) connections. We considered an axonal conduction velocity \(v_c = 0.3 \text{ m/s}\). We considered an external stimulation as a stationary input \(v_{\text{inp}}(x, y, t)\):

\[
v_{\text{inp}}(x, y, t) = \frac{A}{2\pi \Delta_{\text{inp}}^2} e^{-\frac{(x-b_0)^2}{2\Delta_{\text{inp}}^2}} e^{-\frac{(y-b_0)^2}{2\Delta_{\text{inp}}^2}} e^{-\frac{(z-z_0)^2}{2\Delta_{\text{inp}}^2}},
\]

with \(T = 50 \text{ ms}, \Delta_{\text{inp}} = 1.5 \text{ mm}, A = 1 \text{ Hz}, b_0 = 100 \text{ ms}\) and \(x_0 = y_0 = 10 \text{ mm}\).

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M.V. performed numerical simulations, analysed data and prepared figures. M.V. and A.D. wrote the manuscript.

Author contributions

M.V. performed numerical simulations, analysed data and prepared figures. M.V. and A.D. wrote the manuscript and conceived the work.

Competing interests

The authors declare no competing interests.

Additional information

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