SUPPLEMENTARY INFORMATION

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Ion channels enable electrical communication within bacterial communities

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Mathematical Model

We use the following conductance-based model, in the spirit of the well known Hodgkin-Huxley model of neuronal excitability[1], to describe the dynamics of the membrane potential V in *B. subtilis* cells:

$$\frac{dV}{dt} = -g_K n^4 (V - V_K) - g_L (V - V_L)$$

According to this equation, the membrane potential is affected by electric current due to potassium ions crossing the membrane via a dedicated ion channel (first term on the right-hand-side), and by a generic leak current (second term on the right-hand side). The potassium channel is assumed, again analogously to the Hodgkin-Huxley model, to be formed by four subunits, which are open during a fraction of time n whose dynamics is given by the following equation:

$$\frac{dn}{dt} = \alpha(S)(1 - n) - \beta n$$

Here the opening rate α of the potassium channel is assumed to depend on metabolic stress, which we represent here by a dynamical variable, S. This variable stands for the concentration of stress-related metabolic products[2], such as excess NAD+[3,4]. We thus consider that the channel opens when S becomes large:

$$\alpha(S) = \frac{\alpha_0 S^m}{S_{th}^m + S^m}$$

Additionally, and according to our description in the main text, we assume that stress is triggered by depolarization of the cell membrane. We represent this fact by the following dynamical equation, inspired again by the Hodgkin-Huxley model and similar representations of neuronal excitability[1,5]:

$$\frac{dS}{dt} = \frac{\alpha_s (V_{th} - V)}{\exp\left(\frac{V_{th} - V}{\sigma}\right) - 1} - \gamma_s S$$

The first term of this dynamical equation is a threshold-linear function of the membrane hyperpolarization. We also consider the potassium and leak reversal (Nernst) potentials to be affected by the excess extracellular potassium concentration resulting from the channel opening. As a qualitative approximation, we assume that the reversal potentials increase linearly with the excess extracellular potassium concentration E:

$$V_K = V_{K0} + \delta_K E, \qquad V_L = V_{L0} + \delta_L E$$

In turn, E will increase when the channel opens, and will be affected by the loss of potassium to the extracellular environment. We represent the latter by a linear decay rate γ_e:

$$\frac{dE}{dt} = F g_K n^4 (V - V_K) - \gamma_e E$$

Where F is a parameter related with the capacitance of the cell membrane, which describes the relation between changes in excess extracellular potassium and membrane potential. In the experiments we monitor membrane potential via ThT, whose intracellular concentration is expected to increase with the level of hyperpolarization of the cell, since as mentioned in the main text, an increase in the negative character of the cell's interior will cause an increase in the intracellular retention of the dye. We assume the simplest possible dependence between the ThT concentration, T, and the level of membrane hyperpolarization, quantified by the difference $(V_{L0} - V)$ between the resting potential of the cell (equal to V_{L0} in the absence of stress) and the instantaneous value of the membrane potential at any given time. The fluorescence of ThT will also be assumed to decay at a rate γ_t:

$$\frac{dT}{dt} = \alpha_t (V_{L0} - V) - \gamma_t T$$

The equations above allow us to describe the local dynamics of the polarization state of the cells in response to a sudden increase in the concentration of extracellular potassium. Specifically, such a spike in potassium triggers a pulse in membrane potential, pulling it towards depolarization. The subsequent opening of the potassium channel leads then to hyperpolarization. The parameter values used in the simulations are listed in Extended Data Figure 6. To model *KCl* shock (Figure 3d), we used an instantaneous extracellular potassium perturbation of amplitude E = 200 mM.

In order to model the propagation of the potassium signal across the biofilm we extend the model to space. To that end, we assume that the equations listed above describe now the evolution of a set of mesoscopic variables, which represent local population averages on boxes of size $\Delta x = 30$ μm. These boxes are assumed to be small enough so that extracellular potassium concentration inside them can be considered homogeneous, and large enough so that its flux can be assumed to be proportional to the concentration gradient. We then define a discrete chain of $N = 41$ such boxes, and describe the dynamics of potassium concentration by:

$$\frac{dE_i}{dt} = F g_K n_i^4 (V_i - V_K) - \gamma_e E_i + \frac{D}{\Delta x^2}(E_{i+1} + E_{i-1} - 2E_i)$$

Here the subindex i labels the box along the chain, and D is the diffusion coefficient of potassium through the biofilm[6], for which we used the same value employed in the analysis of Figure 2h. Note that all variables of the model depend now on space (V_i, n_i, S_i, E_i, T_i), although only E_i is directly affected by diffusion. The linear chain is simulated with free boundary conditions. To trigger propagating waves (Figure 3e), a stress pulse of size $S = 100\ \mu M$ is applied at one end of the chain.

To model the effect of *yugO* deletion, we set the conductance of the potassium channel to $g_K = 0$. To model the effect of *yugOΔtrkA*, we consider that it leads to an increased flux of potassium toward the outside of the cell and effectively reduces its decay. This can be represented by a leaky potassium outflux in the form:

$$I_{trkA} = g_{trkA}([K]_{int} - E) = g_{trkA}(f - 1)E$$

where $f > 1$ is the normalized intracellular potassium concentration relative to the extracellular one. In effect, *trkA* deletion leads to a reduction in the decay γ_e of extracellular potassium ($\gamma_e = 9\, min^{-1}$). We also consider that the channel conductance g_K is reduced due to *trkA* deletion ($g_K = 19.785\, min^{-1}$). These assumptions are based on several considerations. First, the literature suggests that deleting the TrkA domain increases the basal conductance[2], corresponding to a more "open" channel. Also, deleting the cytoplasmic domain of KcsA, the prototypical bacterial potassium channel, though admittedly different from YugO, yielded a channel with altered gating that still exhibited ion conductance[7]. Furthermore, our own data shows that the *yugOΔtrkA* phenotype does not match the complete *yugO* knockout phenotype, suggesting that the effect of *yugOΔtrkA* is not to produce a permanently closed channel.

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