Serine receptors (Tsr) desactivated
Autophosphorylation of CheA

Controlling cell Division:
We control cell division using [ppGpp] modulated by RelA and Mesh1. To validate our part BBA_K1349001 (RelA coding sequence), we used a mutant unable to synthesize RelA, and growing very slowly. When we complement this strain with BBA_K1349001, bacteria normal growth is restored. Our RelA part is functional.

Rewiring Intracellular signaling:
To modulate expression of genes, we used phosphorylation of the regulator CusR. CusR binds to CusR box sequences in response to metallic stress (Cu, Ag). To validate our part BBA_K1349002 (CusR box sequence), we grew cells in the presence of Ag. As expected, 2 or 3 CusR boxes on a plasmid sequester the endogenous CusR, preventing the normal detoxication of the cells.

Modeling:
A computer simulation of the designed system shows that CusR, and ppGpp can oscillate as the system grows and serine accumulates. We implemented our simulation in Mathlab, using a set of differential equations that we integrated numerically using a Runge-Kutta 4 algorithm.

Beyond the bench:
"Synthetic + + Biology = ...how does it sound for the public?"
Our goal: identify some key rules to communicate efficiently on Synthetic Biology (SB) to a broad audience. Our resources: bibliography + our own experiences "on field" (presentation to local media, sponsors, and during the French Science Festival “Fête de la Science”)

Increased level of serine
Overexpression of RelA

Serine production stopped
Division of bacteria

Autophosphorylation of CheA inhibited
Overexpression of Mesh1