To cover our cell-cell mutual support system, we develop a series of equations to try our best to simulate its behavior. All of them are showing below.

$$\frac{dc_1}{dt} = r_1 c_1 (1 - \frac{c_1 + c_2}{c_{\max}}) - \mathrm{d}_{c1} c_1 - T_1 c_1 - t_1 c_1$$

$$\frac{dc_2}{dt} = r_2 c_2 (1 - \frac{c_1 + c_2}{c_{\max}}) - \mathrm{d}_{c2} c_2 - T_2 c_2 - t_2 c_2$$

$$\frac{dc_{12\,AHL}}{dt} = v_{12\,AHL} c_1 + v_{leak} c_1 - d_{12\,AHL} c_{12\,AHL} - k_{AHL\&R} c_{12\,AHL} c_2$$

$$\frac{dc_{6\,AHL}}{dt} = v_{6\,AHL} c_2 + v_{leak} c_2 - d_{6\,AHL} c_{6\,AHL} - k_{AHL\&R} c_{6\,AHL} c_1$$

$$\frac{dtetA_1}{dt} = (k_{vtetA1} v_{12\,AHL} - d_{tetA} tetA_1) / 2$$

$$\frac{dtetA_2}{dt} = (k_{vtetA2} v_{6\,AHL} - d_{tetA} tetA_2) / 2$$

$$\frac{dZeoR_1}{dt} = v_{ZeoR_m} \frac{c_{6\,AHL}^{\beta_1}}{c_{6\,AHL}^{\beta_1} + m_{C6\,AHL}^{\beta_1}} - d_{ZeoR} c_{ZeoR_1} - m * k_{Zeo\&ZeoR} ZeoR_1^{\,m} c_{Zeo}$$

$$\frac{dZeoR_2}{dt} = v_{ZeoR_m} \frac{c_{12\,AHL}^{\beta_2}}{c_{12\,AHL}^{\beta_2} + m_{C12\,AHL}^{\beta_2}} - d_{ZeoR} c_{ZeoR_2} - m * k_{Zeo\&ZeoR} ZeoR_2^{\,m} c_{Zeo}$$

$$\frac{dT_1}{dt} = T_m c_{Zeo} \frac{m_{T_1}^{\beta_3}}{m_{T_1}^{\beta_3} + ZeoR_1^{\beta_3}}$$

$$\frac{dT_2}{dt} = T_m c_{Zeo} \frac{m_{T_2}^{\beta_4}}{m_{T_2}^{\beta_4} + ZeoR_2^{\beta_4}}.$$

$$\frac{dt_1}{dt} = k_{Ni} c_{Ni} c_{tetA1}$$

$$\frac{dt_2}{dt} = k_{Ni} c_{Ni} c_{tetA2}$$

Variables	Description
c_1	The concentration of cell 1
c_2	The concentration of cell 2
T_1	Describe the pressure on cell 1 comes from the environment
T_2	Describe the pressure on cell 2 comes from the environment
t_1	Describe the harmfulness to cell 1 comes from itself choice
t_2	Describe the harmfulness to cell 2 comes from itself choice
c_{12AHL}	Describe the concentration of 12AHL in the system
c_{6AHL}	Describe the concentration of 6AHL in the system
$tetA_1$	Describe the amount of tetA in cell 1
$tetA_2$	Describe the amount of tetA in cell 2

$ZeoR_1$	Describe the amount of ZeoR in cell 1
$ZeoR_2$	Describe the amount of ZeoR in cell 2

Parameter	Description
r_1	Growth constant of cell 1
r_2	Growth constant of cell 2
c_{max}	The carrying capacity of the system
d_{c1}	The death rate of the cell 1
d_{c2}	The death rate of the cell 2
v_{12AHL}	The max expressing rate of 12AHL
v_{6AHL}	The max expressing rate of 6AHL
V_{leak}	The leak rate of AHL
d_{12AHL}	The degradation rate of 12AHL
d_{6AHL}	The degradation rate of 6AHL
$k_{AHL\&R}$	A coefficient stands for the efficient of the combination of AHL and its receptor
k_{vtetA1}	A coefficient describes the efficient of the translation of tetA which accompanies with the expressing of AHL in cell 1
k_{vtetA2}	A coefficient describes the efficient of the translation of tetA which accompanies with the expressing of AHL in cell 2
d_{tetA}	The degradation rate of tetA
v_{ZeoRm}	The max expressing rate of ZeoR
m_{c12AHL}	12AHL concentration at which the ZeoR expressing is half of the max
m_{c6AHL}	6AHL concentration at which the ZeoR expressing is half of the max
β_1	Hill coefficient of ZeoR expressing in cell 1
β_2	Hill coefficient of ZeoR expressing in cell 2
d_{ZeoR}	The degradation rate of ZeoR
$k_{Zeo\&ZeoR}$	A coefficient stands for the efficient of the combination of Zeocin and its ZeoR
c_{Zeo}	The concentration of Zeocin in the system
m_{T1}	$ZeoR_1$ concentration at which the Zeocin expressing its half harmfulness in cell 1
m_{T2}	$ZeoR_2$ concentration at which the Zeocin expressing its half harmfulness in cell 2
β_3	The Hill coefficient of the exposed Zeocin in cell 1
β_4	The Hill coefficient of the exposed Zeocin in cell 2
k_{Ni}	The coefficient describe the sensibility of the two cells to Ni
c_{Ni}	The concentration of Ni in the system

In our model, we use the growth rate and the quantity of the two cells to represent the punishment of the two prisoners, and they can help each other to resist the environmental stress (Zeocin) by express AHL, but this help may also does harm to itself via producing tetA to bring in Ni.
Here some words describe how we develop these equations.

1. **12AHL/6AHL expression & degradation**

Since the two kinds of AHL are enzymatically synthesized by LuxI/LasI proteins from some constructional genes, we assume that the expression rate of them are both constant as v_{12AHL} and v_{12AHL} there also exists a leak expression denoted by v_{leak}, then the diffusion speed of them are very quickly that we regard the concentration in and out of the cell as union, also AHl can be both naturally degradation and consumed by combination with its receptor. So we get

$$\frac{dc_{12\,AHL}}{dt} = v_{12\,AHL}c_1 \quad \cancel{v_{leak}}c_1 \quad d_{12\,\overline{AHL}}c_{12\,AHL} \quad k_{\overline{AHL\&R}}c_{12\,AHL}c_2$$

$$\frac{dc_{6\,AHL}}{dt} = v_{6\,AHL}c_2 \quad \cancel{v_{leak}}c_2 \quad d_{6\,\overline{AHL}}c_{6\,AHL} \quad k_{\overline{AHL\&R}}c_{6\,AHL}c_1$$

Where v_{12AHL}, v_{12AHL}, v_{leak}, d_{12AHL}, d_{6AHL}, $k_{AHL\&R}$ are all constant.

2. **ZeoR expressing & degradation**

We also assume that receptor of AHL are enough that the effect of this signal path only depend on the concentration of these signal molecules.
When cells experience the enough amount of AHL they will express ZeoR as respond, we use a Hill faction to simulate this process. While considering the ZeoR will bond with Zeocin reduce its effect on cells, we insert the last part of the equation to stand for the consumption of this process.

$$\frac{dZeoR_1}{dt} = v_{ZeoR_m} \frac{c_{6AHL}^{\beta_1}}{c_{6AHL}^{\beta_1} + m_{C16AHL}^{\beta}} - d_{ZeoR} c_{ZeoR_1} - m * k_{Zeo\&ZeoR} ZeoR_1^{m} c_{Zeo}$$

$$\frac{dZeoR_2}{dt} = v_{ZeoR_m} \frac{c_{12AHL}^{\beta_2}}{c_{12AHL}^{\beta_2} + m_{C212AHL}^{\beta}} - d_{ZeoR} c_{ZeoR_2} - m * k_{Zeo\&ZeoR} ZeoR_2^{m} c_{Zeo}$$

Where $k_{Zeo\&ZeoR}$ d_{ZeoR} β_2 β_1 m_{c6AHL} m_{c12AHL} v_{ZeoRm} m are all constant

3. The threat from Zeocin

In our system we regard the concentrate of Zeocin in our system retain a constant level and the harmfulness our cells will experienced is denoted by a Hill faction

$$\frac{dT_1}{dt} = T_m c_{Zeo} \frac{m_{T_1}^{\beta_3}}{m_{T_1}^{\beta_3} + ZeoR_1^{3}} \beta$$

$$\frac{dT_2}{dt} = T_m c_{Zeo} \frac{m_{T_2}^{\beta_4}}{m_{T_2}^{\beta_4} + ZeoR_2^{4}} l$$

Where T_m stands for the max toxin cells will suffer, m_{T1} m_{T2} β_3 β_4 are all constant.

4. tetA expression & degradation

While cells expressing AHL to help each other, they will also translate the sequence right behind luxI/lasI, that is to say, they will express tetA protein as a risk of lies at the same time. So the rate of tetA expression is related to the AHL expression rate we regard their relationship as linear, use a k_{vtetA} to convert v_{AHL} to $v_{tetA.}$ We also assume the dt will represent the time of a whole cell circle, considering the cell division, we divide our equation by 2.

$$\frac{dtetA_1}{dt} = (k_{vtetAl} v_{12AHL} - d_{tetA} tetA_1) / 2$$

$$\frac{dtetA_2}{dt} = (k_{vtetA2} v_{6AHL} - d_{tetA} tetA_2) / 2$$

Where $k_{vtetA1,}$ $k_{vtetA2,}$ $v_{12AHL,}$ $v_{6AHL,}$ d_{tetA} are all constant.

5. The punishment from tetA

We regard the tetA transport Ni into cells as first order reaction. So we get this

$$\frac{dt_1}{dt} = k_{Ni} c_{Ni} c_{tetAl}$$

$$\frac{dt_2}{dt} = k_{Ni} c_{Ni} c_{tetA2}$$

k_{Ni} is a constant describe the sensibility of the cells to Ni.

6. the growth of the two cells

when we add up these equations together and put them into a foundational logistic growth we will get our final equation. But here for simplicity we also supposed that these both toxin subjects have neither constructive nor destructive relationship between each, so we get:

$$\frac{dc_1}{dt} = r_1 c_1 (1 - \frac{c_1 + c_2}{c_{max}}) - d_{c1} c_1 - T_1 c_1 - t_1 c_1$$

$$\frac{dc_2}{dt} = r_2 c_2 (1 - \frac{c_1 + c_2}{c_{max}}) - d_{c2} c_2 - T_2 c_2 - t_2 c_2$$