Carbon monoxide (CO) poisoning is one of the most harmful types of air poisoning in the world. CO gas is mostly released from the internal combustion of engines as well as the use of fuels such as wood and coal. Since carbon monoxide is colorless and odorless it is very hard to be detected without using technological means. However, an enzyme called Carbon Monoxide Dehydrogenase (CODH), found in Mycobacterium tuberculosis, is able to convert CO gas into CO$_2$ gas chemically. In this project, we tried to build a system which can act as a CO filter which can be used to reduce the amount of CO gas in areas where it is elevated. We also attempted to introduce two systems that will help for safer and more controlled use of our "eCO Filter".

**KILL SWITCH:**
We built a kill switch by modifying the T4 lysis device designed by Berkeley 2008 (Bba_K112808). Our kill switch is designed to enhance safety for biological machinery to be used by many, outside the laboratory. The design allows the growth of bacteria only when there is IPTG available. When the bacteria used if at all escapes from its designated area (which contains IPTG) It will not be able to have excess to IPTG in the system via diffusion. When the free Holin molecules reach 3000 cell lysis will occur.

**CARBON MONOXIDE CONVERTER:**
CODH enzyme that we used in our system is isolated from Mycobacterium tuberculosis. This enzyme is able to convert both CO into CO$_2$ and CO$_2$ into CO depending on their concentrations.

The action mechanism of the enzyme is not yet very well studied so it is not possible to gather very good data on its mechanism, yet the procedure is as in the drawing on the right. By using this enzyme the amount of CO can be reduced.

**CELL LIMITER:**
Cell population density changes as the bacteria grow and divide in the biofilm and QS can be defined as the regulation of gene expression during this change. As the cell density increases bacteria starts to release molecules named as autoinducers and according to the concentration of autoinducers gene expression is altered. Eventually bacterial growth will be controlled. In our project, we aimed to modify E.coli to synthesize its own QS signals, detect the cell density and prevent the cell division. When 3OC6HSL is produced, it will form a complex with LuxR which is synthesized according to the activity of LuxPL promoter. After that point we aimed LuxR-3OC6HSL complex to increase the activity of LuxPR promoter. Finally LuxPR activity determines the amount of LasR and when PlasR promoter is activated MinC, a cell division inhibitor can be synthesized. Eventually, cell division stops after MinC reaches its critical concentration.