# Introduction

## Background on cellular aging and calorie restriction (TOR)

## Role of Mitochondria in CR and cellular aging

## CR and ROS (H2O2 and superoxide)

### See bio233 reading assignment

## Ecoli as cellular aging model, its signficantce on CR mechanims

# Material and Methods

## Ecoli

## M9 buffer

## Life span measured by CFU

# Results

## CR extended CLS

## Flow cytomer comparisoin of CR and nonCR E coli

# Discussion

## Rapamycin

## Mitochondira role on CR and aging

# Figures and Tables

## Fig 1,

### 1A comparison of E coli and yeast as models of cellular aging

### 1B, role of mitochondria and TOR on cellular aging

## List of known aging genes in yeast and their E coli orthologs by BLAST

### TOR

### SIR2

### Sch9

### MSN2/4 ?

## Fig 2, suvival cuves and CR effect

## Fig x, conclusion diagram??

# Refrences

# Acknowleges

# abstact

**Lifespan extension by calorie restriction in E. coli cells**

Morgan Maite, Hong Qin

Calorie restriction (CR) is an effective method for lifespan extension in eukaryotes. TOR mediated nutrient sensing pathway and mitochondria play key roles in the lifespan extension effect of CR in eukaryotes. E. coli is a prokaryotic bacterium without mitochondria, and no otholog for TOR has been found in E. coli. Here, we investigated whether CR can extend the lifespan of E. coli cells. We found that E. coli grown in low concentration of glucose live longer than those grown in higher concentration of glucose. We also studied the lifespan of E. coli cells treated with osmolarity shock and rapamycin. Rapamycin is an antibiotics that targets TOR complex in eukartyotic cells. Our study can address whether TOR-independent pathways can also play a role in lifespan extension effect of CR, and whether mitochondria is indeed an essential factor of CR.

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