Writing QE

Rationale:

Why are we and what will we get?

Hypothesis:

Experiment:

Expected Results:

Anticipated challenges and alternative methods:

OVERALL HYPOTHESIS

There is a bloom of uropathogenic *E. coli* in the gut that coincides with the onset of a UTI.

1. Metagenomic guts and glory
   1. Rationale
      1. Gut, skin, and vaginal microbiome is generally stable generally stable through time {Consortium:2012iz, Schloissnig:2012hx}.
      2. Changes in the gut microbiota have been observed in pregnant women{Koren:2012ji}, a population of women who are prone to UTIs{Law:2012jr}.
      3. Changes in the periurethral prevalence of *E. coli* indicate that changes in human microbiota accompany the onset of UTI.
   2. Hypothesis
      1. There is a greater representation of *E. coli* in the gut microbiota of a patient at the onset of a UTI than at times when the patient is healthy.
   3. Experimental methods
      1. Genomic DNA from the fecal samples collected from cohort will be extracted following the protocols of Methe *et al.* (2012). These extracts will be used as template in the amplification of the V3-V5 regions of the 16S rRNA gene using 454 pyrosequencing. Sequence data from this reaction will be then used to estimate the community structure of the gut microbiota. This method has been used previously to analyze the human microbiomes of several body habitats{Consortium:2012iz}.
      2. Binning of the amplicons into phylotypes using the QIIME software package will allow for the representation of those phylotypes in the gut microbiota to be measured. The stringency of the binning can be set to cluster 16S sequences into families, such as Enterobacteriaceae, of which *E. coli* is a member.
      3. Statistical analysis will include the Shannon diversity index to identify intra- and inter-host differences in community structure, Mann-Whitney non-parametric test of means to identify changes in the representation of Enterobacteriaceae, and prinicipal component analysis to measure the tendency of the samples to cluster to their treatment groups (before treatment, after treatment, and after recovery). As an additional control, data on the gut community structure of healthy adults available from the Human Microbiome Project will be included in these analyses in order to minimize biases that might arise due to antibiotic therapy or sampling errors.
   4. Anticipated results
      1. These results will enable discrimination between the treatment groups

What the hell am I writing? Below this line

There is a chance that there is an increase in the representation of UPEC strains in the gut microbiota that are not the same as the strain causing the UTI. This could possibly occur through environmental and behavioral shifts, such as changes in frequency and type of sexual intercourse, which has been found to affect the risk ratio for developing a UTI