Using Tn-seq we found a Salmonella locus required for metabolism of a novel nutrient, fructose-asparagine. Mutants that cannot utilize fructose-asparagine have fitness defects of up to 10,000-fold in mice experiencing inflammation suggesting that this locus encodes potential drug targets. The fitness defect requires inflammation suggesting that competing microbes that normally consume fructose-asparagine may be eliminated by inflammation. The discovery of this locus, the sources of fructose-asparagine in our diet, effect on microbial communities, and our drug discovery efforts will be discussed.