Almost all of the canon of genetics, including human genetics, has been through the study of rare, highly pathogenic alleles causing rare diseases. But, it is likely that most human disease phenotypic variation arises from common, mildly pathogenic variants in transcriptional enhancers. What are the phenotype consequences of such variation and do they teach us new rules (mechanisms) of genetics? I will illustrate some of these new mechanisms by which the widespread genomic variation within enhancers are canalized into phenotypic variation.