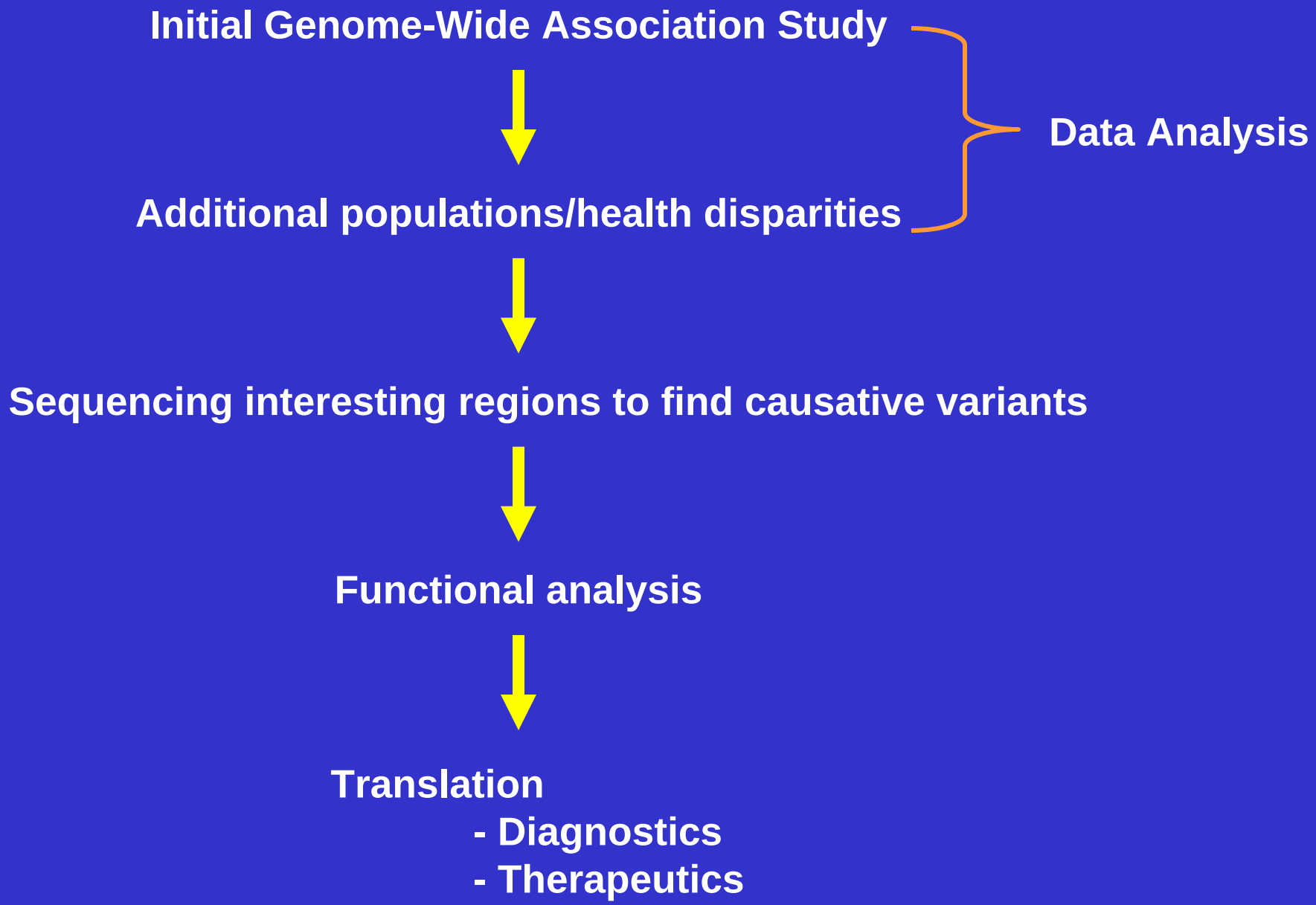
What a GWAS success tells you *if* you achieve genome-wide significance, and you've ruled out population stratification or some technical problem with the genotypes:

- There is a common variant located somewhere in a segment of 3 – 100 kb that is associated with risk of disease (or quantitative trait) in the particular population studied

What it doesn't tell you

- Whether this same variant carries the same or similar risk in other populations
- Which of the many variants in strong LD with the best SNP is actually the *causative* allele(s)
- What the contribution of this variant would be in a population-based prospective study
- How this variant interacts with the environment
- Whether this variant contributes to health disparities
- What the functional basis of the risk is
- Whether this finding will be useful clinically – in diagnosis, prediction of response to therapy, or as a new drug target


Initial Genome-Wide Association Study
Additional populations/health disparities
Data Analysis
Sequencing interesting regions to find causative variants
Functional analysis
Translation
- Diagnostics
- Therapeutics

Possible discussion topics for the panel

- Where is all the missing heredity?
- How can we find less common alleles of larger effect?
- Are we doing a good job of looking for interactions?
- Do we need a deeper database of human genetic variation?
- Could gene expression data from multiple tissues in genotyped donors help us with functional analysis?