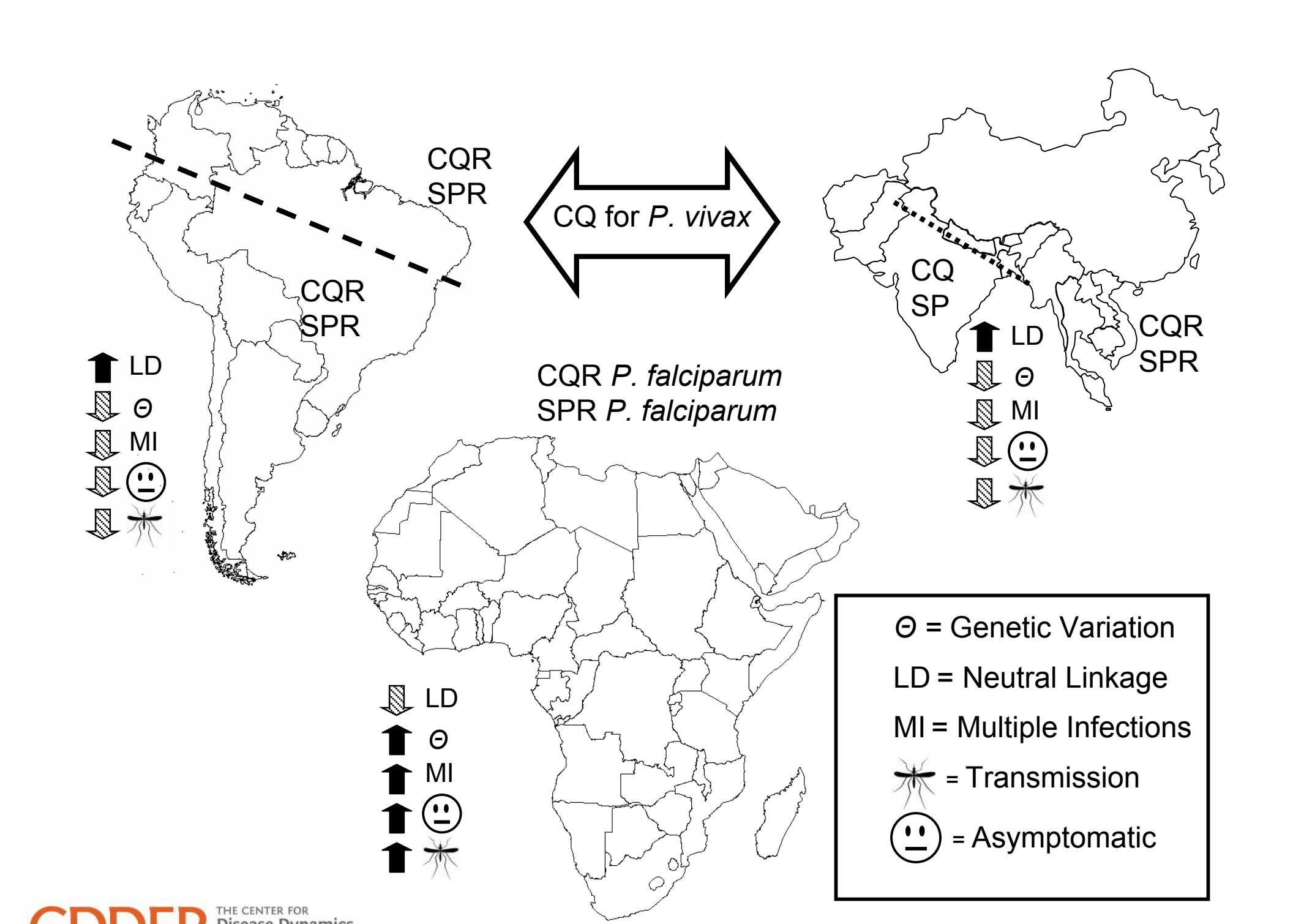

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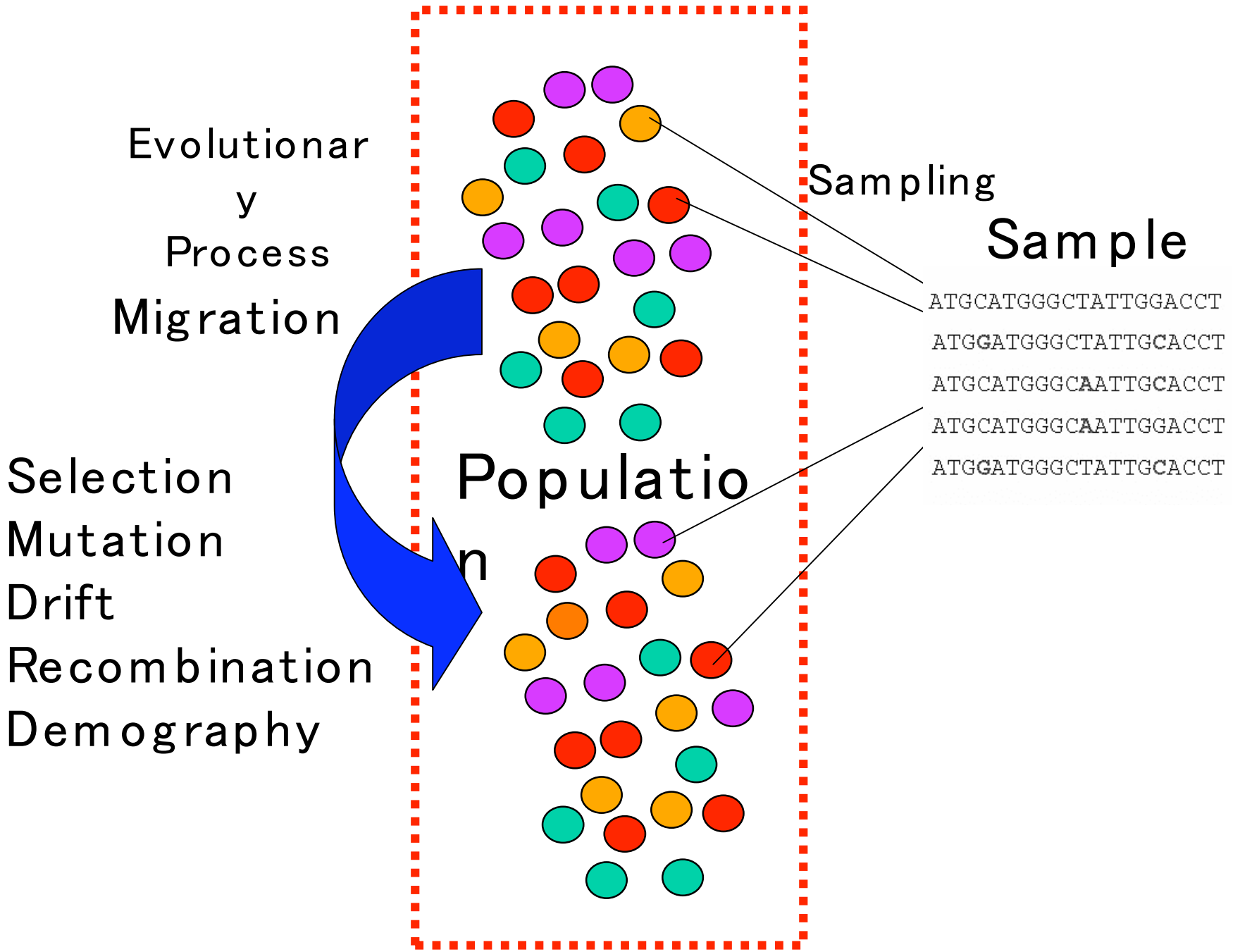
Evolutionar
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Process
Migration
Selection
Mutation
Drift
Recombination
Demography
Populatio
n
Sampling
Sample
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ATGGATGGGCTATTGCACCT
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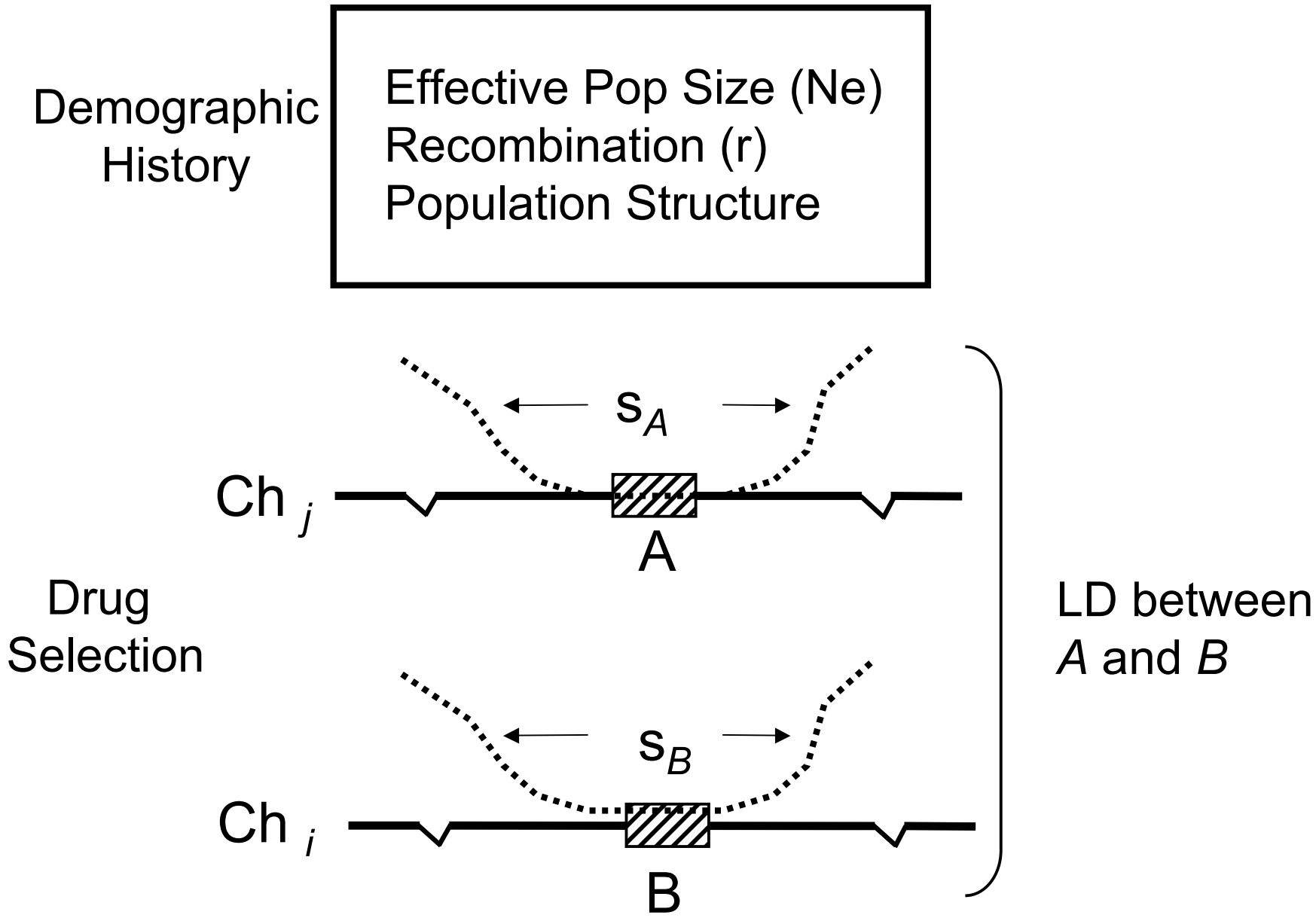

Demographic History
Effective Pop Size (Ne)
Recombination (r)
Population Structure
s_A
Ch $_j$
A
Drug Selection
LD between *A* and *B*
s_B
Ch $_i$
B

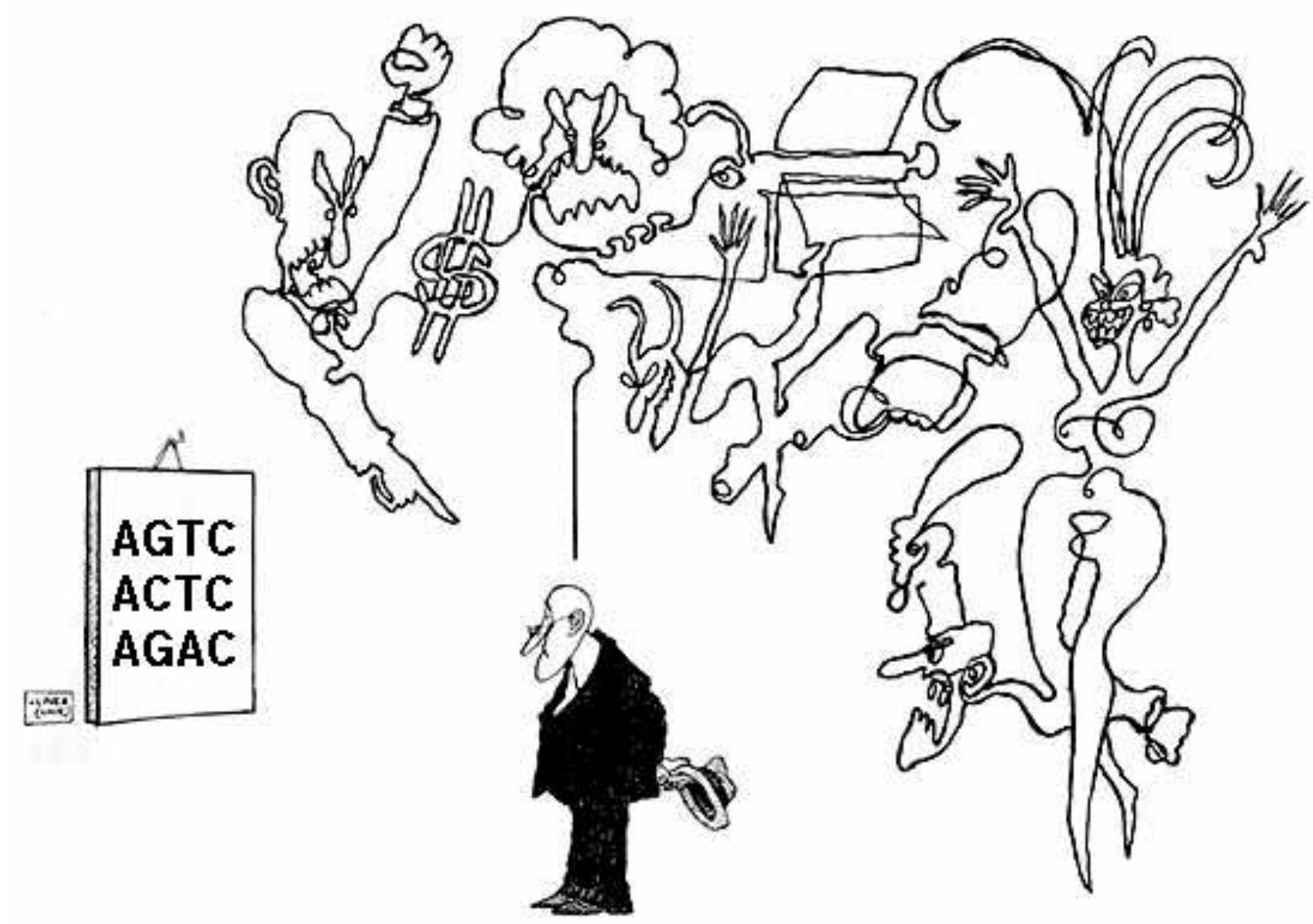
- *Population ecology models:* The frequency of the resistant mutant is modeled from generation to generation without regard to stochastic fluctuations due to changes in the *effective population size*.
- Epidemiological models where the proportion of hosts infected (*Census population size*) is modeled alongside the resistant allele's relative frequency.
- These approaches provide an overall theoretical framework, however, the models were not meant to be real experimental designs with parameters that actually could be defined in the field and estimated.
- However, we need to incorporate the evolutionary dynamic of mutations in the context of the populations demographic history

Evolutionary Biology

- Theory that allow analyzing data of several alleles sweeping simultaneously in populations with complex demographic histories
- The STRs high density in the *P. falciparum* genome, together with their high mutation rate makes them a good choice for characterizing the dynamics of sensitive versus resistant alleles
- There are robust attempts for developing experimental and theoretical methods for SNPs (is a good proxy for human genetic diseases).
- We need to understand the dynamic of STRs (mutation models) and how they relate with SNPs under complex demographic models

Disease Ecology

- Complexity of infection as measured by a set of genetic markers instead of intra-host dynamics
- Immunity defined in terms of specific antigens
- Contribution of the clinical and sub clinical infections into malaria transmission
- Gene flow among populations (parasites /mosquitoes/ humans)
- Transient linkage (inbreeding) as it effects the risk of multi-drug resistant lineages


AGTC
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