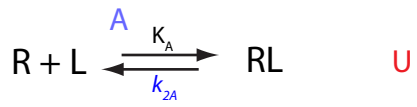


# 1:1 binding mechanisms

Generic 1:1 binding



General convention:

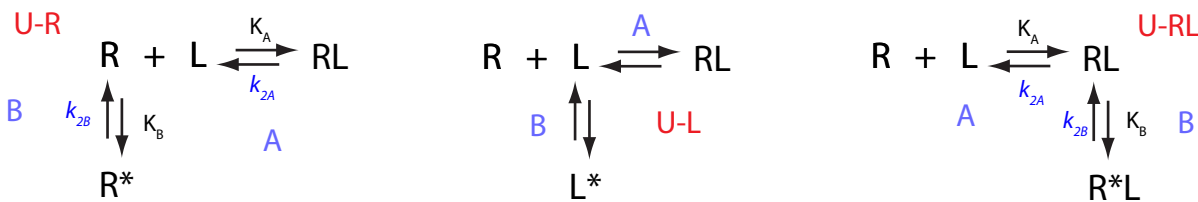
R - receptor, NMR observable species;

L - ligand, NMR invisible

My code (molecularity-highest order species)

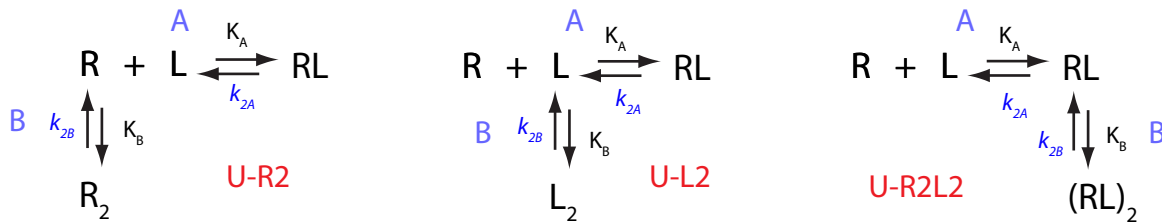
Transition name: A, B, C...

1:1 binding coupled with a unimolecular dead-end interconversion



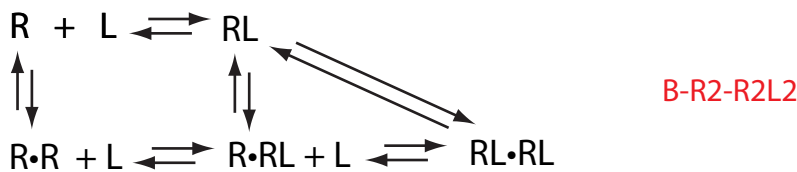
In these reactions we have concentration-independent isomerization that simply equilibrium concentration may only manifest itself through rate-limitation phenomena producing 'linear' population changes on titration.

1:1 binding coupled with a dead-end dimerization reaction



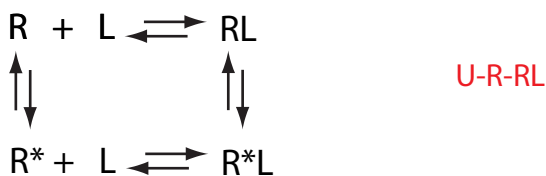
In these reactions the dead-end complex formation depends on concentration so will both influence population profiles (ITC) as well as kinetic behavior through rate-limitation.

1:1 binding coupled with a binding-competent dimer formation



U-RL-RL reaction scheme includes dimerization steps described above and adds a balance between the dimeric species through parallel ligand binding equilibria. We should see both non-linear population changes in the course of titration as well as rate-limitation phenomena.

Parallel 1:1 binding reactions coupled via unimolecular receptor isomerization



U-RL-RL reaction scheme includes concentration-independent isomerization steps and adds a balance between the isomers through parallel ligand binding equilibrium. Equilibrium concentration of species will be scaled by isomerization reactions, which will only manifest themselves in rate-limitation phenomena.

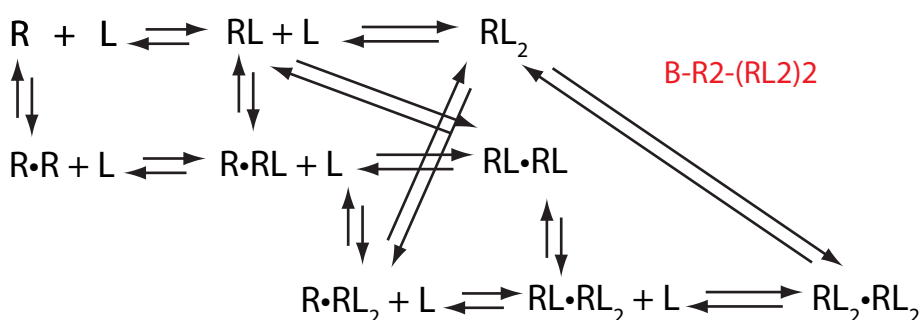
# 1:2 binding mechanisms

Generic 1:2 binding



B mechanism will produce both non-linear population changes and rate-limitation steps.

1:2 binding coupled with a dimerization reaction



B-R2-(RL2)2 reaction scheme includes dimerization steps as well as second ligand binding steps. The scheme is overly complicated and only useful in some of its limiting cases. Which cases are related to real world?