

# Sketch for simulating chemodiversity

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2018-09-21

## (rough) sketch components responsible for chemodiversity

### Genes

- define which enzymes are produced in which quantities
  - list in fig. 1 in [1]
- can be scaled down/inactivated (i.e. when predators leave for generations)
  - easy to ramp up production as long as the genes are still there
- plants can survive without problems with inactive PSM-cycles when no adversaries are present.

### Inheritance & Mutation

- via whole-genome and local-genome duplication
- copies accumulate mutations that lead to neofunctionalization
- e.g. subtle differences in terpene synthases can yield vastly different products
  - i.e. these changes can appear easily
  - need to classify products by “chemical distance” for simulation
  - **TODO**: Map/Markov-Chain of mutations that may occur here?

### Evolutional strategies

- “Bet-hedging”: reduce variations of fitness over time
  - **TODO**: understand
  - different effects of intra-cohort-variation vs. inter-cohort-variation
- Plants with inactive PSM can survive if predators are deterred by other individuals due to automimicry-effect which *could* foster wider genetic variance

- the more of those individuals are present in a population, the less their overall fitness becomes.
- **TODO:** fitness must also be able to depend on relative appearance of adversarial traits in the population
  - \* Keyword: Frequency-dependent-selection (FDS)

## Pathways to produce chemical compounds

- 40k+ compounds just stem from compounds of the calvin-cycle taking the MEP-pathway or from the krebs-cycle taking the MVA-pathway
  - both yield the same intermediate product that forms the basis.
- 10k+ compounds are amino-acid-derivatives
- Chapter VI in [1] exemplary describes 4 complete different pathways that yield compounds.
  - similar compounds/pathways should be found in the simulation

## Consequences of producing compounds

- taking away parts of the calvin/krebs cycle puts pressure on those
  - **TODO:** find out what they do and on what they depend.
- **TODO:** where do amino-acids come from? How much impact has the diversion of these components?

## Maintaining chemical diversity

### + screening hypothesis

- many PSM found have no *known* biological activity
- plants “keep them around” in case another mutation needs them to produce something “useful”
- creating things without use increase the need for photosynthesis and/or nutrient uptake.

### - screening hypothesis

- it is suggested that local abiotic & biotic selection pressures are the main driver
- inactive molecules are not maintained long
- it was observed that some plants “rediscovered” some compounds in their evolution suggesting they got rid of them when no pressure to maintain them was applied

## questions resulting from this that should be answered in the simulation

- details in chapter VIII of [1]
- how quick can lost diversity be restored?
- how expensive is it to keep producing many inactive substances while also producing active deterrents? Does this lead to a single point-of-failure due to overspecialisation? What must be done to prevent this?
- strong selection pressure *should* decrease quantity of compounds due to costs, but plants do not seem to care.
  - is this diversity needed in presence of multiple different adversaries?
  - does the simulation specialize when only presented with one adversary? What about adaptive adversaries?
  - adaptation in the qualitative & quantitative evolution in response to changed pressure? (i.e. those who cannot adapt quick enough die?)

## Scenario

### Plants

```
data Foo = Bar
```

### Enzymes

### Herbivores

### Environment

### Fitness

### Mating & Creation of diversity