Although the phylogeny of extant salamanders may be inferred using molecular data, the inclusion of fossils is more difficult. Fossil salamanders were thus placed on the extant phylogeny using relationships proposed by the most recent phylogenetic assessment of those fossil species (see SOM). However, two major clades of fossil salamander, the scapherpetonids and batrachosauroidids, are problematic, and the placement of these clades remains uncertain. To handle this, we ran analyses over seven different topologies, each with a different placement for those two clades. One topology, where scapherpetonids and batrachosauroidids are sister to each other and outside of Urodela, produced notably different estimates for rates through time (a greatly enhanced spike in rates at the KPg boundary).

1:10 – Scap sister to all, Batrach sister to Crypts (S,((B,Crypt),Others)))

11:20- Scap sister to Crypts, Batrach sister to all (B,((S,Crypt),Others)))

21:30—Scap outgroup to crown, Batrach outgroup to Scap+Crown (B,(S,Crown))

31:40—Scap and Batrach sister to each other, outgroup to crown ((B,S),(Crown))

41:50—Scap sister to Batrach + Crown (S,(B,Crown))

51-60—Scap sister to crown, Batrach sister to Hyno+Crypt (S,((B,(Hyn,Crypt)),Others)

61-70—Batrach sister to crown, Scap sister to Hyno+Crypt (B,((S,(Hyn,Crypt)),Others)

We modelled neoteny using the liability model proposed by Felsenstein (#CITE) and implemented in a Bayesian framework by Revell (#CITE). This model treats the multiple observable discrete states as manifestations of a continuously-varying “hidden” or latent trait that evolves by Brownian Motion. This model has a useful biological property, in that metamorphosis mode in salamanders is thought to be controlled by variation in the level and reception of thyroid hormones (#Rose 1996, #CITE). Essentially, a continuous variable (thyroid hormone) controls the expression of a discrete state (life history) in salamanders, providing a match between model and data. The model also has several desirable statistical qualities. First, although the rate of change in the continuous character is constant throughout the tree, the *transition probability* between states may vary between clades. As an example, assume to lineages (a and b) alive at time t both have the same rate of change (sigma = 1), and there is a threshold at 0 (negative values = state 0, positive values = state 1). The liability of lineage a is 0.5, and the liability of lineage b is 100. At time t + delta\_t, then the probability of descendants in both state 0 and 1 is substantially higher for lineage a than it is for b. Thus, the model allows for clades to have different levels of discrete state heterogeneity without assuming a more complex model. Another useful property of the liability model (shared with several rate-matrix approaches) is that transitions between states may be uneven. That is, in a three-state model, it may be substantially “easier” to move from state 0 to state 1, than it is to move from state 1 to state 2 (represented by thresholds that are unevenly spaced relative to the rate of continuous character change; e.g., thresholds at 0, 10 with sigma = 1).

This approach has two useful statistical properties. First, although movement of the continuous trait is constant and symmetrical, transition rates between character states can be asymmetrical without having to optimize a large rate matrix. Second, although the rate of change for the continuous trait is constant across the tree, and the thresholds for discrete states are fixed, the actual frequency of transition between states among subclades can vary. This means that some clades can frequently shift between states while other clades may be “locked” in a single state, again without having to estimate transition points along the phylogeny. Biologically, this model is also very appropriate for neoteny in salamanders, as the discrete states described above are known to be influenced by continuously-varying levels of thyroid hormones (#Rose 1996; #CITE). Paedomorphosis in salamanders may occur in anything from the failure of skull bones like the pterygoid to be resorbed to the retention of gills and juvenile coloration at sexual maturity. For the purposes of this study, we discretized this array of forms with four states: direct development (no larval stage), biphasic/metamorphosing, facultative neoteny (some populations metamorphose in certain conditions), and obligate neoteny (no populations metamorphose).

Use of the Price equation necessitates a word of caution: it is a decomposition of pattern, not a causal model of process (#Frank 2012). That is to say, a trait found to be favored by species selection, it is not necessarily the case that the trait that *causes*increased net diversification, but simply the trait *has increased in frequency due to increased net diversification*. This is made clear by an analogy to microevolution: if two alleles (a and b) are closely linked on the genome, and allele a is strongly selected for, while allele b is neutral, both will increase in frequency. The increase in frequency of allele b is still due to selection, but not selection *for*b. In the case of species selection, the Price equation will identify traits that have increased in frequency due to selection, but cannot determine whether the traits observed were the cause of increased net diversification. If a single clade in the phylogeny undergoes a large increase in net diversification, then all synapomorphies of that clade will increase in frequency (from 0 to N\_clade / N\_total). In that sense, the Price equation should be interpreted as informing us as the cause of *heterogeneity in trait distributions* rather than the causeof *heterogeneity in diversification rates*.