Cumulative Natural Selection

**Discussion Responses:** Here are some examples of the sorts of responses you might expect. There will certainly be some other responses, some appropriate and reasonable, others possibly not so. The following comments could be used to offer clues and views to either get discussion going, or to channel it along productive lines, perhaps by giving some hints for responses like these, if necessary. Be sure to note the comments at the end regarding the point of this lesson.

1. In what ways is shuffling the equivalent of genetic mutations?
   Shuffling is just a simplified model of all the random events involved in sexual reproduction (e.g., mutations, meiosis, mating and fertilization).

   In what ways is it not?
   As in any model or analogy, it’s never perfect. For example, for point mutations, nucleotide sequences are not really shuffled; some individual bases may be changed or removed or added. During crossing over, entire chromosome segments are exchanged - it’s really not a shuffling process, as we do with cards.

   Does the model (card "game") distinguish between phenotype and genotype?
   The model does not distinguish between phenotype and genotype; it could reflect either level.

2. What is the one, critical respect in which the actions of the odd- and even-numbered teams differed?
   The way in which the “functional” specified sequence was obtained. Team A could only finish if all the cards were in the proper sequence. Team B was able to build the "functional" specified sequence one card at a time, retaining the next needed card when it was played, and simply adding to the played stack already in sequence.

   What is the biological equivalent of this difference?
   In real organisms, mutations (or genes) are simply added to the existing genome as they arise. The entire gene sequence or collection of functional genes does not have to be produced before it is incorporated.

3. What, in the game, represented selection?
   The action of allowing one or more cards to be retained. In Team A, all the cards had to be in order to be retained. In Team B, each card could be retained if it fit into the desired (“functional”) sequence.

4. Why, in the game, was selection cumulative?
   In Team B, selection was cumulative to illustrate how mutations, genes and traits are accumulated, added to existing mutations, genes and traits if they didn’t ruin the “functional” sequence. Selection in Team A was not cumulative - it was an “all or none” process.

5. What was the average number of observed generations needed to evolve the organism by the even-numbered teams?
   This will vary, but will virtually always be much fewer than for the odd (A) teams.

   How does this figure compare to the calculated average number of generations? (Hint: On the average, in each round, the ace has a 1:13 chance of coming up, the "2" has a 1:12 chance, etc. The sum of the numbers from 1 to 13 is 91) This will vary
6. What was the average number of observed generations needed to evolve the organism by the odd-numbered teams? 
This will always be a much higher number than for the even (B) teams. Most (all?) A teams will never get the complete sequence.

Do we have the data to answer this? Probably not; few if any of the A teams will get the whole sequence.

What would be the calculated number of generations? (Hint: We need to have the ace show up first, with a probability of 1/13, then the "2." with a probability of 1/12 ... to the king with a probability of 1/1. 1/13 X 1/12 X 1/11 ... 1/1 is approximately 1.6X10^-10. 1/1.6X10^-10 is about 6.2X10^9. Shortcut: 13! = 6,227,020,800.)

7. How many times faster is the evolution of our model organism with cumulative selection versus without cumulative selection among the mutations? 
Probably not possible to calculate, because (usually) none of the A teams get the sequence, so there is nothing with which to compare.

8. What new understanding has this lesson taught you? 
When selections are allowed to accumulate (as they do in real organisms), virtually any new combination that could contribute to survival (or not impede it) has a very good chance of arising and being selected (or retained).

The point of the lesson is that functional genes (and their respective expressions) are formed by the accumulation of changes over time, each change being incorporated and retained in the population as long as it does not reduce the survivability of the organism. Also, this kind of accumulative change can happen much more quickly than any kind of “perfect storm” - where all the right ducks need to be in a row before any part of the gene (or its product) can influence survival. All of the “mathematical disproofs” of evolution are based on the (wrong) assumption that all the components (parts, genes, or nucleotides) need to be “right” and properly functional before it would work, so anti-evolutionists have claimed that such complex systems as living organisms would be so improbable as to be virtually impossible - and students should be made aware of this fallacy, because it is based on a false premise. In reality, genes and traits do not need to arise de novo (from scratch), but rather are modified from, and added to, pre-existing genes and traits. They are retained (or not) in the population depending on whether or not they contribute to survival in whatever environment exists at the time, which is constantly changing. Therefore, genomes, genes and traits are constantly changing.

A major misconception: Many people assume that, logically, any mutation will most certainly gum up the works and be fatal - like adding a grain of sand to the works of a fine watch. The only problem is that the biological “machinery” is actually very flexible and accommodating to many disruptions. For one thing, students probably realize that there can be 3-4 different codons coding for the same amino acid, and they may also realize that many of the amino acids in a protein are simply place-holders, not critical to the functional shape of the protein, so alternative amino acids can make no functional difference. In addition, entire segments of DNA can replicate and be retained, often in tandem with their original copy, sometimes many times. While one segment (gene) continues to function normally, the other(s) can mutate and, from time to time, produce better (more effective) genes, or genes with different functions. There are many clusters of such genes that are clearly related, with very similar sequences that had to have been derived from one original version. The hemoglobins and gamma globulins are two examples of such “gene families.” In addition, there are many non-functional “pseudogenes” scattered throughout genomes - each DNA segment with nearly the same sequence as a functional gene, but which does not code for any function.

Furthermore, many (most?) biochemical processes can be accomplished by way of alternative pathways (redundancy), so if one pathway is blocked due to mutation, another pathway can suffice.