Some thoughts about things I learned at Dan Dennett's excellent party

I have been thinking about the relationship between the kind of models that Pete, Joe and I have made and the ECM framework sketched by Dan and Nicolas in their paper. In particular, I have trying to understand how the mechanistic processes represented in our approach relate to homo-hetero distinction central to the ECM approach. In my remarks on Sue Blackmore's papers I sketched a simple model in which there were two variants with a selection stage and a transformation stage. So, first I'd like to convert this model to the ECM framework developed by Sperber and Cladiere. Label the variants 1 and 2. Let W_i be the fitness of variant i in the selective stage. Individuals who acquire variant 2 remain variant 2, but individuals who acquire variant 1 transform to variant 2 with probability m. Like Nicolas and Dan, I will assume that populations are large enough that drift-like sampling processes can be ignored. These assumptions lead to the following recursions for the frequencies of the two variants, f_1 and f_2:

$$f_1' = \frac{(1-m)f_1W_1}{f_1W_1 + f_2W_2}$$

and

$$f_2' = \frac{mf_1W_1 + f_2W_2}{f_1W_1 + f_2W_2}$$

Since $(1-m)f_1W_1 + mf_1W_1 + f_2W_2 = f_1W_1 + f_2W_2$ these recursions are equivalent to the following Evolutionary Causal Matrix

$$\begin{pmatrix} (1-m)W_1 & 0 \\ mW_1 & W_2 \end{pmatrix}$$

This exercise illustrates that the "population genetics/epidemiology" formalism and the ECM formalism can be equivalent ways of representing exactly the same underlying processes, and that the transformation of variant 1 into variant 2 appears as a hetero effect of variant 1 on the frequency of variant 2. Notice that variant 2 has no hetero effect on variant 1. This suggests, to me at least, that the magnitude of the coefficients in the ECM may not represent the causal processes involved in cultural evolution. For example, it could be that the variants are two versions of a story, and learners have existing theories that transform a fraction m of all stories into variant 2. A fraction of the learners who hear variant 1, think they have heard variant 2. This seems to me best thought of as variant 2 having a hetero effect on 1, not the reverse as the coefficients suggest. On the other hand, it might be that variant 1 is more complicated and harder to remember, so that learners learn 1 but sometime later remember 2. This seems to me to be best thought of causally as an effect of 1 on 2. I'd be interested to know what Nicolas and Dan think about this.

I also think it's important to see that even very simple models will require ECM's in which matrix elements to depend on variant frequencies. Consider

a second, even simpler model which only contains transmission and transformation (a guided variation model in the RHB jargon and analyzed in Boyd and Richerson 1988)—no selection at all. Suppose individuals have an individual learning mechanism that responds to environmental cues. There are two variants again labeled 1 and 2. With probability p_i they receive a cue that tells them that variant i is best and they adopt that variant. With probability $L = 1 - p_1 - p_2$ the cue does not clearly tell them which variant is best and they imitate a randomly chosen individual from the previous generation. This leads to the following recursions for the frequency of the two variants:

$$\begin{aligned} f_1' &= Lf_1 + p_1 \\ f_2' &= Lf_2 + p_2 \end{aligned}$$

First notice that the population evolves to an equilibrium at which the variant that is more likely to be learned is at higher frequency $\hat{f}_1 = \frac{p_1}{p_1+p_2}$ and thus if $p_1 \gg p_2$ transformation alone can lead to cumulative adaptation. Also, notice that since there is no selection in this model, there is no need to normalize frequencies by dividing by an "average fitness" like term. Thus the ECM is

$$\begin{pmatrix} L + \frac{p_1}{f_1} & 0 \\ 0 & L + \frac{p_2}{f_2} \end{pmatrix}$$

Since the off-diagonal elements are both zero, there are no hetero effects in the model at all. The L components of the homo terms are fine; they just represent incomplete transmission. But the second part of these terms are peculiar in that they are inversely proportional to the frequency of the relevant variant. This is formally necessary in order to represent frequency independent learning, but it doesn't seem to reflect the causal structure of the processes modeled. What is really going on is that there is a frequency independent learning process that creates variants one and two with probabilities $p_1$and p_2. This suggests that the ECM frame work should be extended by adding a frequency independent term that represents the effect of the non-cultural environment on the frequencies of the cultural variants.

Finally, I think that it will be important to think carefully how to incorporate multidimensional cultural variants into the ECM framework. Consider a trait that has two dimensions. Each dimension can have two states which I will imaginatively label 1 and 2. For example, dimension 1 could the the length of the bow (long or short) and dimension 2 could be ether it is sinew backed (yes or no). So there are four variants 11, 12, 21, and 22. Individuals squire both dimensions form one of their parents. This cultural trait has no effect on the probability of becoming a parent and there is no error, so that transmission leaves the frequency of the four variants unchanged, and thus the ECM matrix is

$$\begin{pmatrix} 1 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}$$

The influences are purely homo–there is no interaction between traits and no hetero influences.

Now lets modify the model a bit. Suppose that with probability r individuals individuals acquire the two dimensions of their behavior from two different randomly chosen individuals, and with probability $1-r$ they acquire both dimensions from the same individual. Thus, for example, the frequency of variant 11 in the next time period is

$$\begin{aligned} f_{11}^{'} &= r\left(f_{11}+f_{12}\right)\left(f_{11}+f_{21}\right)+\left(1-r\right)f_{11} \\ &= r\left(f_{11}^{2}+f_{11}f_{21}+f_{12}f_{11}+f_{12}f_{21}\right)+\left(1-r\right)f_{11} \\ &= r\left(f_{11}\left(1-f_{22}\right)+f_{12}f_{21}\right)+\left(1-r\right)f_{11} \\ &= f_{11}-r\left(f_{11}f_{22}-f_{12}f_{21}\right) \\ &= f_{11}-rD \end{aligned}$$

The population genetics student will recognize this as a recursion giving the effect of recombination on gamete frequencies. The recursions for the other three cultural variants are

$$\begin{aligned} f_{12}^{'} &= f_{12}+r\left(f_{11}f_{22}-f_{12}f_{21}\right)=f_{12}+rD \\ f_{21}^{'} &= f_{21}+r\left(f_{11}f_{22}-f_{12}f_{21}\right)=f_{21}+rD \\ f_{22}^{'} &= f_{22}-r\left(f_{11}f_{22}-f_{12}f_{21}\right)=f_{22}-rD \end{aligned}$$

This set of recursions can be represented by many distinctive ECM's. To see this, let's concentrate on the first row of the ECM which is

$$1+rf_{22} \quad -rf_{21} \quad 0 \quad 0$$

or

$$1+rf_{22} \quad 0 \quad -rf_{12} \quad 0$$

or

$$1 \quad -rf_{21} \quad 0 \quad rf_{11}$$

or

$$1+rf_{22} \quad -\tfrac{1}{2}rf_{21} \quad -\tfrac{1}{2} \quad f_{12}0$$

And there an infinite number of possibilities. The problem is that the terms proportional to products of frequencies like $f_{12}f_{21}$ can be represented in two different columns of the matrix, or as any convex combination of the two terms (i.e. the weights have to sum to one). I think this means that it is not possible to read the hetero/homo effects directly from the matrix, but I am not sure. Perhaps some convention could be established that would allow the matrix to accurately reflect the causal process of partial recombination.

I am also puzzled by another aspect of this model. Let $D=f_{11}f_{22}-f_{12}f_{21}$. This is the covariance between the trait value along dimension 1 and dimension 1. For example if short bows are more likely to be sinew backed $D>0$. Let p_i be the frequency of variant i (long or short) for dimension 1 and q_j be the

frequency of variant j (backed or not) for dimension 2. We can write down recursions for the frequencies of each dimension, and for D.

$$\begin{aligned} p_1^{'} &= p_1 \\ p_2^{'} &= p_2 \end{aligned}$$

which is equivalent to the ECM

$$\begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}$$

which represents pure homo effects. The ECM for the q_i's is the same and there is a recursion for D, $D' = (1 - r)D$. It is not easy for me to see how to incorporate the later recursion into the ECM framework. If we added selection-like processes to the model, I believe that they would end up as homo processes in the first representation (one trait, four variants) and as hetero processes in the second representation (two traits with two variants and a covariance) in which the degree of hetero influence would be proportional to the covariance divided by a variance (i.e. how much a variant on of one trait predicted the variant of the other trait).

At the end of the meeting I commented to Dan that Pete, Joe, and I tended to look at these processes at coarser scales than he, Nicolas, and Olivier. And, I still think this is true in some ways. The latter three are more interested in the micro causal details that give rise to persistence. For example, Pete, Joe and I have thought of there being alternating cycles of internal and external representations. However, we preferred to zoom out and try to write models that black boxed the details of this processes while Dan, Nicolas and Olivier want to think about the causal processes in detail. However, the current exercise makes me think that there are also ways in which the kinds of models that Pete, Joe, and I have made are less coarse grained than the ECM approach because they build in more meso scale detail about the dynamic processes.

Reference

R. Boyd and P.J. Richerson, An Evolutionary Model of Social Learning: The Effects of Spatial and Temporal Variation. In: *Social Learning: A Psychological and Biological Approaches*, T. Zentall and B. G. Galef, eds., Lawrence Erlbaum Assoc., Hillsdale, NJ. pp 29–48, 1988.