

## Re: Ernst Mayr: Where Are We (1976)

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- *From:* "John Edser" <edser@xxxxxxxxxx>
  - *Date:* Fri, 3 Jun 2005 13:39:02 -0400 (EDT)
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"Perplexed in Peoria" jimmenegay@xxxxxxxxxxxxxxxx

>> JE:-

>> Given the empirically true fact that all genomic gene fitness in nature remain epistatic and therefore 100% dependently selectable whereas the opposite is assumed within population genetics oversimplified models that have ended up providing evolutionary theory with everything from Haldane's (false) Dilemma to Hamilton's heuristic rule that was misused to determine when organism fitness altruism could evolve in nature, where are we now in 2005? Why hasn't a theory of heritable epistatic gene fitness been developed allowing a minimally VALID simplified model of TWO loci with two alleles that are dependently and not independently selected at the empirically true, single, fertile organism level of selection?

> JM:-

> Because it is an "empirically true fact" that epistatic gene fitnesses (as you seem to use this term) are not heritable.

JE:-

Mayr is discussing the same point that I have been making for more than 5 years. Polygenetic traits empirically exist (traits whereby the phenotypic is simply the addition of each gene's effect + or - e.g. human height) allowing each gene to have an independent effect. However the FITNESS of the trait is NEVER the simple addition of the fitness of each polygene disallowing any assumption that the FITNESS of each gene is independent. It remains incorrect to infer that because polygenes exist polygenetic fitnesses also exist. Not one single additive in gene fitness trait has ever been documented in nature yet population genetics models assume that the fitness of one gene can simply be added to another.

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> JM:–

> There do exist models of two epistatic loci dependently selected at the organism level.

JE:–

The "organism level" is not empirically sufficient but the fertile organism level is where these levels are not at all the same.

Epistemologically: all models remain simplifications/over simplifications of an existing valid theory. Gene centric Neo Darwinists always refuse to just name the theory these models were simplified/over simplified from i.e. their models are attempting to replace valid theory. Model results have to be corrected by the theory they were simplified/over simplified from in order just to make any sense. So far they remain uncorrected because a multilevel theory has never been proposed. More than just the mere suggestion that multilevels of selection exist is required to produce a valid theory of same. To my knowledge nobody has even attempted to provide a minimal dualistic theory, i.e. how two independent levels of selection could possibly operate in nature. All sorts of problems have been ignored. Are these two model independent levels selected simultaneously or serially? If simultaneously how is such an event even possible? If serially what happens when they compete? Does the first level to be selected dominate the other? What is the missing multilevel theory Neo Darwinistic models were simplified/over simplified from and where are the required missing theory corrections for all these model multilevel results?

> JM:–

> The key issue in such models is the degree of linkage (segregation independence) between the two loci. It turns out that if the loci are unlinked (on different chromosomes, say), then \*\*under the assumption of random mating\*\* the process of recombination undoes any linkage disequilibrium created by selection.

JE:–

Linkage and fecundity rates (the rate of reproduction of sterile immatures) determine the probability that two alleles at two different loci may be inherited together within just a sterile immature body. When a female tick, which can only reproduce itself once, mass produces about 10,000 sterile immatures and then dies, many recombination events are represented in these immatures. What matters is which of these 10,000 are raised to fertile adulthood and exactly how is each gene is actually selected in nature. In this species only about 2.something sterile immatures are mostly raised to fertile adulthood, i.e. just a tiny fraction of the fecundity rate. The mass of infertiles that die along with their gene combinations are only subject to a process of sub

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selection and not selection because they are only dependently and not independently selectable. This means that high fecundity rates can be advantageous because they allow the less costly act of sub selection of the better epistatic gene combinations. Sub selection is more efficient than selection because a selection of them would require immatures to be raised to fertile adulthood so they acquire an independent fitness.

> JM:–

> Hence, the standard "independent selection of loci" model  
> are close to correct, as long as you allow the fitness of alleles at one  
> locus to depend upon the frequency of alleles at another locus.

JE:–

These models do not distinguish between fertile and infertile forms so they have no way of discriminating between the much more costly act of independent selection from the much less costly act of the dependent sub selection of expendable sterile immatures.

> JM:–

> On the other hand, if the loci are tightly linked (close together on the  
> same chromosome, say), then recombination will disrupt the linkage  
> disequilibrium only rarely. Under these circumstances, you can think of  
> the two loci (each with two alleles) as a single locus (with four alleles)  
> and the situation is again well handled by the standard models.

JE:–

Since the overwhelming majority of genomic loci are empirically homozygous 2 epistatic genes within most two locus two allele dependent fitness models can be inherited together. However, only the heterozygous exception is discussed. Apparently the much more common homozygous event is regarded as less interesting yet it is more compelling for the heritability of epistatic gene fitnesses.

> JM:–

> Models exist for the cases of close (but not too close) linkage, but they  
> are not exactly \*simple\* models. Models also exist which relax the  
> assumption  
> of random mating, and which thereby make the epistasis somewhat  
> "heritable"  
> in that way. These models are definitely NOT simple.

JE:–

All empirical gene fitnesses remain epistatic no matter what model attempts to dictate that they "cannot be". Any model that deletes such a basic empirical fact has to be defined as "oversimplified"\_. The non correction of oversimplified model results via the theory it was

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oversimplified from constitutes a gross misuse of that model. My example remains Hamilton's Rule. Here relatedness  $r^e$  has to set  $e$  to be minimally 2 and not just 1 because all empirical gene fitnesses are epistatic requiring a minimum of two dependent loci. The variable  $e$  must be minimally set to 2 if the rule is not to be just a non representative oversimplification. Unfortunately, in this case the rule fails (even as just a 100% relative proposition).

My epistemological objection has always been the same. I am happy to accept simplified/over simplified models as extremely useful tools to help understand/test the scientifically valid theory they were simplified/over simplified from, but only after any model result has been corrected by the parent theory. Quite obviously, models cannot contest or replace their parent theories. Yet, this is exactly what has happened over the last 50 years as mathematicians replace biologists within evolutionary theory so that Popper becomes discarded as irrelevant to just the mathematics.

> JM:–

> As I understand it,

> this is the kind of issue which distinguishes Wright's view of selection

> in structured populations from Fisher's view of selection in well-mixed

> populations. One of these days, I am going to have to dig up a good

> tutorial on Wright's F-statistics and see how they work.

JE:–

I think the main difference was that Wright allowed drift to produce many isolated sub populations that allowed the testing of drifted epistatic gene combinations within each sub population before they remixed with the main population.

> JM:–

> You also demand that the models work at the "fertile organism level of selection" rather than just at the "organism level". I understand

this

> to mean that the selective fate of an immature organism is taken to be

> a function of its parents' genomes, rather than its own.

JE:–

I do not "demand it", empirical science does. Fitness remains the only common point of test of any evolutionary theory so far proposed that can be tested. The focus of gene centric Neo Darwinism is entirely individual gene fitnesses. Genomic genes are not selectable within immature sterile forms for the obvious reason that these forms have no heritable fitness until they become fertile. However the same genes remain selectable from whence they came: within the fertile bodies of the parents that provided them. It remains biologically incorrect to regard sterile immatures as equals to fertile forms simply because gene

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centric models require them to be so. Immatures are entirely at the disposal of their parents because the genes in their bodies can only be dependently selected via their parents Total Darwinian Fitness (TDF). Eusocials are not unique. They prove that selection on just fertile forms can enslave immatures for the TDF benefit of their parents (and the sterile forms because they remain TDF dependent) but only under enclosed nest conditions where a controlling pheromone can circulate.

> JM:–

> But this is also

> empirically untrue, at least in part. While the parents' genomes are

> important (in plants and in animals with parental care, at least), the

> genome of the immature organism is different from that of its parents

> and it is very important in determining the fate of the immature organism.

JE:–

Yes, but their fitness fate remains entirely with their parents TDF until they become fertile themselves. The genes within infertile forms remain entirely non heritable until that form actually becomes fertile.

Only at this one, exact point can the epistatic fitness of their gene combination even possibly become subject to natural selection. This basic remains deleted within most models that simply assume independent gene fitnesses where the results of such a model oversimplification remains uncorrected, e.g. Hamilton's rule where  $e$  remains fixed to 1 and not minimally 2 within relatedness  $r^e$ .

> JM:–

> Models which make selection dependent upon two or three genomes are

> definitely NOT simple. They are also unnecessary, since Hamilton has

> shown us how to work with a simple model in which the *\*inclusive\** fitness

> of an organism is taken to be dependent upon only its own genome.

> I invite people who understand pop gen better than I do to comment upon

> my summary.

JE:–

Understanding how gene fitness epistasis can be coded and inherited remains one of the biggest challenges of biology. It does not help to just delete this problem within uncorrected oversimplified models and then declare the problem solved. Hamilton's model deleted all epistasis yet empirical gene fitnesses remain epistatic no matter if Fisher dictated that they can't be. Any valid theory must fit the facts and not the reverse. In reality each competing allele within Hamilton's model actually represents one entire fertile form. For his model to even approach biological reality  $e$  within  $r^e$  must be minimally set to 2 and not just 1. Assuming  $e=1$  allowed  $e$  to be invalidly deleted and then forgotten.

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Haldane's Dilemma was proven false. No dilemma empirically existed. Yet, nobody even thinks of correcting the oversimplified modelling assumptions that provided Haldane's false dilemma. Why?

Regards,

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• *Follow-Ups:*

◆ *Epistasis and Linkage – was: Ernst Mayr: Where Are We (1976)*

◇ *From: Perplexed in Peoria*

• Prev by Date: *Re: Yes Yes Yes*

• Next by Date: *Re: Why did eukaryotes evolve?*

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