ABSTRACTS

Evolutionary and Molecular Genes: The Case of Cystic Fibrosis
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I will discuss the issue of the identification of the gene from a viewpoint of the relationship between evolutionary and molecular genes. Moss makes noteworthy remarks on cystic fibrosis (CF) in the context where he introduces the well-known distinction between Gene-P and Gene-D (Moss 2003), which invited me to conduct some historical as well as contemporary research on the pathophysiological mechanism which makes this lethal genetic disorder happen—especially, the relationship between genotype (mutations) and phenotype (symptoms) governing its onset, by examining several medical articles with the purpose in mind of bringing out how the two types of gene locutions are intertwined in the medical discourse about CF and, eventually, of elucidating what it really means when we speak of “the gene for CF.” Followings are the primary points reached:

Collins, Riordan, Tsui, and colleagues’ historical feat of identifying the cystic fibrosis transmembrane conductance regulator (CFTR) gene in 1989 employed the approach of “reverse genetics,” for they had to start to discover it while knowing little about the protein synthesized from it. On the other hand, discovering the mutations that cause pathological symptoms involved the approach of “forward genetics,” because it started with the CF phenotypes of patients and then went downward to track down the underlying genotypes (mutations).

As for the relationship between evolutionary and molecular genes, first we can point out a kind of conceptual isomorphism between “the gene for X” talk emblematic of evolutionary biology and “the gene for CF” one which is one of the exemplars of molecular biology. Second, and more important, the evolutionary gene concept is viable today even in the modern molecular biology. For instance, in some context, it is a prerequisite for an arbitrary DNA sequence to be identified as a gene (coding region) that it is an evolutionarily conserved (homologous) sequence.

On the Concept of Genetic Distance: the perils of misinterpretation
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The notion of ‘genetic distance’ or ‘genetic difference’ between individuals and between populations has been a focal point of much interest in the scientific literature in the recent decade. But also of much controversy as both empirical and theoretical studies have often arrived at somewhat contradicting claims. For instance, in a widely-cited paper, Rosenberg et al. (2002) conclude from analysis of molecular variance that “The average proportion of genetic differences between individuals from different human populations only slightly exceeds that between unrelated individuals from a single population”. On the other hand, Bamshad et al. (2004) have showed that pairs of individuals from distinct populations are often more similar than pairs from the same population, but at the same time stress that for any level of population differentiation “individuals from different populations are, on average, slightly more different from one another than are individuals from the same population”. More recently, elaborate empirical and theoretical analysis has highlighted how the relation between such differences crucially depends on the number of polymorphisms sequenced and the some measure of closeness of source populations. Expanding the scope to include the phenotypic aspect, Witherspoon et al. have recently speculated that a hypothetical trait, primarily determined by some identified set of additive loci with known worldwide distributions, could be analyzed using simple measures of genetic distances, since the allele sharing “genetic distance… is equivalent to a phenotypic distance”. In my talk, I will trace the source of some of the confusion and suggest new perspectives for conceptualizing genetic distances. I will also show how an inferential leap from genetic to phenotypic distances is hardly straightforward, and that claims of equivalence between such distances should be handled with much care.

Fitness and Variance
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In this paper I argue that a consequence of natural selection in populations with variance in reproductive success is that the fitness of a type is not grounded in the fitnesses of individuals of that type. I then argue that this entails that some fitness-involving evolutionary explanations are neither wholly causal nor wholly noncausal. I begin by introducing the propensity interpretation of fitness, here defined as the conjunction of four distinct theses:

PROPENSITY: Fitness is a propensity.
UNIFORMITY: Fitness has a single measure.
OFFSPRING: Fitness is measured by expected offspring number.
PRIORITY: The fitness of a type is grounded in the fitnesses of individuals of that type.
Previous discussions of variance in reproductive success have not adequately distinguished these theses, with
the result that the implications for PRIORITY have not generally been appreciated. A notable exception is
Sober (2000), whose argument against the propensity interpretation I turn to next. I describe and respond to
two objections that have been made to Sober's argument, and then generalise the argument in two ways. First,
I argue that it is PRIORITY, and not the propensity interpretation as a whole, that is the target of the argument.
Second, I argue that the fundamental source of the problem for PRIORITY is not population size, but rather
correlation in reproductive success. Here I rely on a model described by Frank and Slatkin (1990). By
presenting the Frank and Slatkin model, I explain both why correlation is fundamental and why PRIORITY is
false.
I conclude by arguing that the failure of PRIORITY entails that some fitness-involving evolutionary explanations
are neither wholly causal nor wholly non-causal. This in turn suggests the possibility of a middle way between
causal and statistical interpretations of natural selection.

On the relation between biological information and biological inheritance
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The relation between biological inheritance and biological information is close but tricky and unclear, and has
not been directly explored. Since the rise of molecular biology, genes have been thought of not only as
heritable units, but also as informational units. Moreover, they were considered the only heritable and
informational units. The traditional focus put almost exclusively on genetic inheritance, and the early
introduction of the term 'information' to refer to genes, may have given the impression that both properties
come in a package, as if every heritable unit were an informational one, and vice versa. This, however, may
constitute a confusion about these two concepts.
At present, we recognize other, non-genetical, inheritance systems, but this only redoubles the confusion.
Usually, biologists do not offer a clear definition of either of the concepts. Additionally, they sometimes refer to
non-genetical heritable units as informational units as well. Among philosophers, there is no consensus on
whether non-genetical structures can be said to carry information. While some authors presuppose the
property of heritability in their accounts of biological information without explicitly specifying what the relation
between the two concepts is, others seek to extend the concept of information so as to cover a larger set of
heritable units. The heterogeneity of the positions on the matter raises some puzzling questions.
My aim is to analyze the conceptual relation between inheritance and information, and to suggest that the
confusion can be addressed in two complementary directions. A first issue takes form when analyzing the
notion of information: is the notion of inheritance constitutive of the notion of information in biology? A second
issue arises when looking at the notion of inheritance, in connection with the multiplicity of inheritance systems:
are there any reasons to treat every heritable unit as an informational one?