Heredity and Statistics Before Fisher

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1 Fisher's 1918 paper responds to a long debate about continuous and discontinuous heredity. Blending. Can evolution be explained as an accumulation of small variations? For Darwin this was fundamental.

A critique well-known by Fleeming Jenkin challenged the idea that "sports" or monstrosities could ever find mates to pass on a new trait. This paper was often read backwards, that the results of continuous selection run up again limits.

Although Darwin's theory had no mathematics, it was in a way utterly statistical.

Contrast with Galton who (like Fisher) was thinking in eugenic terms (avant la lettre) from 1860s. After *Hereditary Genius* (1869) he turned briefly to experiments on peas. He found what he called **reversion** toward the (racial) mean. But the distributions did not narrow.

In his publication he described the problem as a combination or summing of error curves, much like what Fisher framed in terms of "variance" (nicely modeled by the quincunx).

Its stability depended on the reappearance of variation even when peas at at the mean were bred together. Also humans.



Reversion 1875 was about a return to an ancestral form. He later switched to a different word, regression. This tended toward an ancestral **type**, a point of stability. This could pose a problem for Darwin's evolution.

Stability of type was structural not simply ancestral. Real evolution required getting over the hump to a new point of stability. He used a metaphor of types of towns for this: some can blend, some not. Continuous variation might still lead to new types if the force of selection was strong.

He never spoke of blending inheritance. Mendelism as break from blending models is a myth. Some traits blended, he said; some not. Race was exemplary of blending, like the mixing of a liquid, of **blood**. His peas, unlike Mende's, blended, because Galton was not studying hybridity.



Reversion to past forms was supported by regression to a point of stability. The accumulation of ancestral "gemmules"--Galton used Darwin's word--was, for Galton, an obstacle to species change.

The law of ancestral inheritance supposed that half the gemmules were expressed in each offspring, and half remained latent.

Galton was more worried than Pearson about the dead weight of the past. Pearson supposed that correlations could work in favor of hereditary (or eugenic) change. (and he did not support the stability argument.

Fisher I think anticipate random or undirected transmission of genetic units



Fisher's paper is partly an effort to identify ancestral sources that will allow him to identify unexpressed as well as the expressed ancestral genetic units. Looking to ancestral traits to get at latent (or dormant) ones.

This is like but not the same as the Law of Ancestral Inheritance. It is in a way an old eugenic story (older than the word). Compare madhouse tradition: genetics: F. W. Hagen and Wilhelm Tigges's worked in 1860s and 1870s to measure the effect of insane vs. healthy on offspring by recovering as much as they can of the extended family history (of these traits).



There was a widely-shared sentiment in early 20th century that small or continuous variations were not inherited. De Vries argued this way; Bateson and Johannsen still more emphatically. Pure lines. Mendelian genetics, too, was set against evolution by natural selection.

Fisher, like Pearson, backed Darwin. But he too began later to insist on discontinuity (in a way the 1918 paper does not).

Here (1918) Fisher already takes as given that continuous variation can arise from multiple discrete factors (such as genes). *The Genetical Theory of Natural Selection* (1930) makes Mendelian discontinuity fundamental.

THE GENETICAL THEORY OF NATURAL SELECTION

BY R. A. FISHER, Sc.D., F.R.S.

The 1918 paper sets out from a (by then) widely shared idea that Mendelian discontinuity can turn to continuous variation by combining multiple genetic factors.

But he allows weak or absent dominance and other effects that seem to weaken genetic discontinuity even when a single gene is involved specifically in a trait.

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