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Genetics and Plant Breeding, 1910-80

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Genetics and plant breeding, 1910–80

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The early successes of genetics and plant breeding and the still earlier successes of microscopy and chromosome study led to disputes, which were aggravated by lack of understanding between languages, professions and techniques. But their primary source lay in each pioneer's insistence on a uniformity of his own natural law. Bateson's exclusion of nucleus and cytoplasm was followed (in 1926) by Morgan's exclusion merely of the cytoplasm. An anti-genetic and anti-evolutionary revival was favoured by these disputes and has left its traces with us today.

The idea of a uniformity in heredity or the genetic system is once again an obstacle to understanding. For, in the practice of plant breeding, we are faced by a conflict between evidence on experimental and evolutionary time-scales. Louis de Vilmorin, Darwin and Mendel thought of this problem under the title of the 'causes of variability'. We can now recognize that the experimental or classical models of mutation and recombination of genes and chromosomes is no longer universally sufficient either for organisms or for their chromosomes. Variability in higher organisms seems to have a variety of pre-nuclear as well as nuclear foundations.

I

The great pioneers of our subject were tormented by crises of belief and uncertainty, which we need to understand in facing our own problems today. It is only today after 70 years that such understanding is coming within our reach – and may soon slip out of our reach.

Consider the crisis (on 18 July 1910) when Bateson, arriving in Merton to set up his new John Innes Horticultural Institution, received a letter from Morgan‡ in Woods Hole telling him about his 'extraordinary luck with a sex-limited case in *Drosophila*'. Morgan enclosed the corrected manuscript of an article for *Science* and offered to send Bateson stocks of his white-eyed fly. 'Drop me a line', he added, 'about what you think of the result.'

Morgan's 'result' was one that neither of the two men could at once understand. But it quickly divided them. On the one hand, Morgan learnt to see it as reconciling Mendelism with the chromosome theory. He did so with the help of Wilson, who told him of another piece of chromosome theory, known in German by the name of 'crossing over'. On the other hand, Bateson refused to learn this lesson, as he might have done from his mentors in Cambridge, Doncaster and Gregory. Instead he devoted the rest of his life to protecting the mystery of Mendelism from the rival mystery, which he privately called the 'chromosome cult'. So it was that when he returned from his last visit to Morgan in 1922 he exclaimed, in despair (again privately), that 'all my life's work has gone for nothing'.‡

It is important for us to understand the paradox of Bateson. The very fact that he had attached his idea of genetics to his own verbal framework of Mendelism (F_1, F_2, allelomorph, homozygote and heterozygote, etc.) prevented him admitting that the microscope might make this framework part of something bigger, indeed something deserving his name of genetics.

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† Died 26 March 1981. ‡ See notes.

It was tragic that this happened at the moment when he was engaged in setting up his own centre of genetic research. He began by appointing W. O. Backhouse. Within 2 years this young but already experienced plant breeder had discovered the capacities and limitations of inbreeding and outbreeding in a wide range of horticultural plants. This was not Mendelism, however, and Backhouse was quickly despatched to breed wheat for the Argentine government. But from seeing the results of this work, another young man, N. I. Vavilov, who came a year later, picked up and took away his own long-range ideas about species and their systems of variation. He could distinguish polyploid wheats, $4x$ and $6x$, by their diseases before their chromosome numbers were known.

One after another the plant species and genera that Bateson was breeding revealed polyploidy, a chromosome property, that he was determined to disregard as irrelevant to heredity. Chromosomes and genes, and above all the incompatibility gene, were set on one side.

A climax came in 1926, when two opposed and partly mistaken views of heredity were reached. On the one hand, Morgan in *The theory of the gene* gave the whole of heredity to the nucleus and had nothing left for the cytoplasm. On the other hand, Bateson, because he refused to give anything to the nucleus, also could allow nothing for the cytoplasm. He found himself caught in a trap by the cross between oil and fibre flaxes, with its conflict between nucleus and cytoplasm. He extricated himself with a verbal fantasy, a piece of abstract Mendelism which he called 'anisogeny'.

Thus the role of the cytoplasm, which was evident to the German botanists, Erwin Baur, Fritz von Wettstein and Otto Renner, was hidden from our zoologists, Bateson and Morgan, and hidden also from a generation of plant breeders.

At this point, the John Innes work had comfortably split itself as though in two adjoining university departments. On the one side there was breeding of plants without looking at their chromosomes; on the other there was looking at chromosomes without breeding the plants. This could not go on for long and in 1924, innocently unaware of the rules, I began to look at the chromosomes of *Prunus*, *Pyrus* and *Rubus*, the original subjects of Backhouse's experiments. At a meeting of the Genetical Society in December 1925, with Bateson in the chair for the last time, I explained my results. Polyploidy occurred and was to be connected with the fertility and sterility found in fruit-tree breeding. I had no idea that in reaching this obvious conclusion I was exposing to the public view what had been Bateson's private nightmare.

With the death of Bateson in February 1926 the taboo in his own institution against the chromosome theory quietly crumbled. *Primula*, *Pisum* and *Campanula* as well as the fruit species soon yielded to the attack. Plant breeding could be put on diverse, verifiable and particulate foundations. Natural and artificial selection also fell into place. But, owing to Bateson's prestige, the damage that he had done to his own subject, to genetics, remained. His prejudice reinforced just those ignorant beliefs in the world outside that he himself had most strenuously condemned. Anti-evolutionism, behaviourism, Lamarckism, and later Lysenkoism and mere obscurantism, were all sustained and the ground for future conflict was prepared.

One of these conflicts concerned the time-scale or, if you like, the evolutionary horizon, proper to our kind of enquiry. In plant breeding, I would say, there is a scientific horizon some 10000 years back and far, far, longer forward. But there is a practical horizon no longer than a man's lifetime. The difference between the two horizons was already apparent in 1923 when Bateson was invited to be Chairman of a new joint committee of the Ministry of Agriculture and the Royal Horticultural Society (a kind of hybrid or quango). Its scientific object was to 'compare' all fruits, including new products of plant breeding. Its practical object was to 'standardize' and maintain old commercial varieties of fruit.

On account of the double basis of fruit growing in this country, these two objects were inevitably, as they still are, in conflict. On one side were a few hundred commercial growers with a united economic interest in their capital investment. They sell fruit; they resist the introduction of better new varieties. On the other side were a thousand times as many private gardeners who grow fruit. They eat it themselves and they want the best. These latter have relied for 200 years on the success, a world-wide success, of enterprising nurserymen such as Veitch, Rivers and Laxton. It was they who had collaborated with Bateson and with Backhouse. They were the people who would be needed in the future as in the past for conserving the diversity of perennial fruit crops on the principles of Vavilov. Since the death of Bateson, these principles have been disregarded in favour of short-term aims. Together with the name of the Institution, the new John Innes fruit varieties have accordingly been suppressed, with economic consequences that are now painfully recognized.

II

Fifty years after Bateson and Morgan, microscopic, molecular and experimental techniques have revealed to us connections and continuities they did not and could not know. The genetic materials of plants and animals in Nature, we can see, are programmed in genetic systems that have evolved and, what is more, are evolving on an evolutionary time-scale. We are compelled to work mainly on an experimental time-scale. But the continuity of the chromosomes allows us to compare the two scales. We can therefore now ask ourselves how the chromosomes transfer from one scale to the other.

This question may be put in several ways. The information in the chromosomes represents their experience accumulated under natural selection and sometimes promoted by feedback trends over immense periods of time. Why, then, can the plant breeder, attempting to draw on this credit account, sometimes draw only a blank? Or, to put it in the forgotten terms of Louis de Vilmorin, Darwin and Mendel: what are the natural causes of differences in variability between species?

In broaching these questions, we are better equipped than our predecessors. We have our models. We have, first, a heredity, determined by the linear organization of DNA in the chromosomes of the nucleus and of the pre-nuclear plastids and mitochondria. And secondly, we have evolution, proceeding, we presume, on classical experimental assumptions, by breakage, reunion and recombination in these chromosomes, limited or promoted by natural selection.

At once, equally in the vertebrate animals and in the angiosperm plants that we know best, these models face us with four apparently non-classical and non-Darwinian questions:

(i) Why are there great evolutionary jumps in chromosome size unrelated to any apparent change in the character of the organism?

(ii) Why are there evolutionary changes in parts of chromosomes that we know to be excluded from crossing over?

(iii) Why are there sharp differences in the variability of species?

(iv) Why do such differences occur in respect of both the structure of organisms and the structure of their chromosomes, the two sometimes apparently unrelated to one another?

The last two questions are the ones of practical interest to us here. Can we now distinguish species of flowering plants of a known and comparable history that differ decisively in their variability? For comparison, they must be diploids, free from hybridization, with a common uniform origin, and with still largely uniform and interfertile chromosome complements. Among such plants we may take four examples of extremely high variability revealed and preserved by selection (table 1).

Table 1. Four highly variable cultivated plant species compared in history, character and chromosomes (Darlington & Wylie 1965)

1. *Zea mays* from *Euchlaena mexicana*	$2n = 20$	America (Galinat 1971; Beadle 1975; Darlington 1973)	agricultural annuals, disruptively selected for some 5000 years: innumerable varieties
2. *Brassica oleracea*	$2n = 18$	Europe (Pease 1926; Darlington 1973)	
3. *Hyacinthus orientalis* 5000 named varieties since 1640	$2n = 16$	Syria to Europe (Darlington *et al.* 1951)	horticultural perennials consciously selected over 100–300 years
4. *Chamaecyparis lawsoniana* 200 named varieties since 1876	$2n = 22$	USA to Europe 1854 (Den Ouden *et al.* 1965)	

In all these cases there seems to have been a general preservation of interfertility among a wide range of diverse products. This is true even in *Hyacinthus*, where polyploidy has been selected but has significantly failed to affect the range of variation. Analogies in animals would be with man and perhaps his domestic animals selected for that reason.

The converse situation is that where violent chromosome changes occur without a serious alteration in the phenotype of the organisms or even their interfertility. The most obvious example is in an animal, the muntjac deer, where two species exist. They exist as closely similar kinds of organisms and in breeding they are still infertile, but one has three pairs of chromosomes (in the female) and the other has twenty-three pairs, the two having a similar total quantity of DNA (Chiarelli & Capanna 1973).

There are several directions in which we may look for explanation of these breakdowns of our classical model. One is general: perhaps the nuclear revolution failed to abolish the pre-nuclear plasmid devices of recombination whose relics therefore sporadically reappear to destabilize species. A second more specific possibility is that these relics are seen as very diverse forms in the intra-nuclear infections or transposable elements of McClintock, the genotrophic changes of Durrant, and the transformations of Pandey. Yet a third lies in the undoubted instability of heterochromatin due to the potentialities for illegitimate crossing over of highly repetitive segments (Darlington & Shaw 1959).

I suggest, however, that there are many more separate or sequential possibilities. Heredity in the higher organisms is not, as Bateson or classical genetics supposed, the expression of natural laws on inorganic principles; rather it is itself the result of evolution and is undergoing evolution. The transition from pre-nuclear to nuclear heredity is not necessarily complete or perfect or universal; genetic systems are therefore subject to many kinds of breakdown. These deserve our examination.

Notes

(i) Morgan's letter I have placed in the John Innes Archives.

(ii) Bateson's cry of despair was uttered in the Athenaeum club to Leonard Darwin and reported at once to E. B. Ford.

(iii) The first John Innes Report seems to have been published by Bateson without permission of the John Innes Trustees. I persuaded them to reverse this rule in 1937.

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