STERILOID

AND

SUB-FATUOID OATS

CYTO-GENETIC STUDIES

OF

STERILOID AND SUB-FATUOID OATS

BY

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I. General Introduction.

False wild oats or fatuoids, so-called because of the resemblance they bear to the wild oat Avena fatua, have been found in most varieties of A. sativa and A. byzantina and are analogous to the speltoids occurring in wheats. The most remarkable feature of the various fatuoids is that regardless of the strain in which they originate the one complex of characters involved, namely, callus pubescence, articulation and awning, is common to all fatuoids. In all other morphological characteristics fatuoids resemble the strains in which they originate.

The problem of the origin of fatuoids has been a subject of much discussion during the last fifty years. Early investigators were concerned with the natural crossing hypothesis on the one hand and the mutation hypothesis on the other. Later, gene mutation and chromosome aberration hypotheses were mooted by differeht workers, the natural crossing hypothesis having been ruled out by most as invalid. Although a recent paper has revived the natural crossing hypothesis, evidence presented in this thesis strongly supports the chromosome aberration hypothesis.

Semi-fatuoid and sub-fatuoid types have also been reported but no combined cytological and genetical study has been made on them. The author presents, in this thewis, a unified chromosome aberration hypothesis as an explanation for the aberrant types studied. Semi-fatuoid (steriloid) types resemble the species A. sterilis. Sub-fatuoids do not, as far

as has been ascertained, resemble any naturally occurring oat species. The author studied these forms cytogenetically as it was expected that since the steriloid and sub-fatuoid types are allied to the fatuoids, an elucidation of the cytological rearrangements involved in strains of the former would yield data complementary to those obtained from strains of the latter.

True fatuoids do not display the "delayed germination" characteristic of A. fatua and some of the segregates of fatua-sativa crosses, so that the former are not a serious weed menace from that standpoint. Their appearance in strains of "Registered" seed oats is, however, very troublesome.

A solution of the problems involved in the origin of the various fatuoid and sub-fatuoid types has a direct bearing on the general question of variation in polyploids, to which so many of our economic plants belong.

II. Critical Review of the Literature.

One of the earliest papers on this much-discussed subject is that by Haussknecht in 1884 (quoted by Zade, 1918). According to Zade Haussknecht pointed out that intermediate forms appeared in a continuous chain between Avena sativa and A. fatua in fields of cultivated oats. He called these spontaneously developed

variations Avena fatua var. transiens. He argued in accordance with the general belief at that time that since there was no diminution of fertility in this form they could not be the result of a cross between two distinct species. He reported that in continuous cultures this form tends to lose the hairy base so that in later generations it cannot be distinguished from the cultivated oat.

Max Fischer in 1900 (also quoted by Zade, 1918) maintained that winter oats were more inclined to revert to wild oats and he therefore recommended caution in sowing winter oats. He said that the intermediate forms which he found might be the product of crossing but were more likely "throw-backs".

Howes in 1908 (quoted by Huskins and Fryer, 1925) collected oat grains showing "wild tendencies", i.e. prominent basal articulation, and strong twisted awns from four varieties derived from A. sativa, namely, Black Mesdag, Swedish Select, Early Victor and Early Ripe. The plants grown from these selected seeds were found to be similar to the parent varieties in every way except that the seeds they produced again showed the same "wild tendencies". During a period of three years in which the experiments were conducted there was no evidence of "reversion", which was at that time one of the most commonly accepted explanations of the origin of false wild oats.

Howes found that the false wild oats do not have the property of delayed germination which makes A. fatua a serious weed menace.

Thellung in 1911 (quoted by Zade, 1918) found intermediate forms of oats and supported Haussknecht's view that they were spontaneously developed variations.

Criddle (1910) used the English term "False Wild" for the first time, and reported (1912) the results of experiments in growing different types of false wild oats showing the outward characteristics of wild oats, i.e. having a long twisting awn and typical horseshoe base, but which in other respects could not be distinguished from the cultivated variety in which they originated. In addition he reported the results of experiments with Banner oat derivatives showing a tendency in the direction of the wild type, which Mr. A. Cooper, Treesbank, Manitoba had collected in a field free from wild oats. The first year about two per cent. developed into fully formed false wild oats, the majority remaining as before. A certain number developed stronger awns and these were again sown and produced about twenty per cent. false wild oats, about forty per cent. of the long awned kinds and the remainder normal oats. Some of the original seed was again sown and less than one per cent. produced false wild oats and about one per cent. dwarf sterile false wild oats about six inches in height.

Criddle pointed out that germination in false wild oats is not delayed and for this reason alone they need not be classified as noxious. He arrived at no conclusion concerning their origin, but remarked that since they seem to be no more numerous in oats grown in contact with wild oats than in varieties kept free from wild oats it is hardly likely that they are the result of a cross between the wild and cultivated species.

The editor of Criddle's bulletin, G.H. Clark, states that the "accidental or natural crossing of cultivated oats with Wild Oats may seem logical and might be accepted were it not known that in some countries, as in Sweden for instance, False Wild Oats also appear amongst cultivated varieties which, as far as human control can be relied upon, have never come in contact with Wild Oats. Dr. H. Nilsson-Ehle of the Swedish agricultural experiment station for plant breeding at Svalöf, one of the best authorities in that science, gives us a very logical scientific explanation of the occurrence of these False Wild Oats, as being apparent mutants occurring through disturbance in the arrangement of certain hereditary units of characters in the reproduction. According to Dr. Nilsson-Ehle, the idea that the False Wild Oat is the product of crossing is absolutely out of the question; he contends that <u>such hybrids would show different characters and that the similarity to both of the parents would extend itself over a larger number of characters, instead of being confined to anatomical changes in the hulls and rachilla only.</u>" (italics supplied).

Zade (1918) pointed out that in samples of threshed oats one often finds grains showing characters intermediate between wild and cultivated oats. He says that the principal points of difference between A. fatua and A. sativa are found in these intermediate forms, but in a lesser degree. He seeded a quantity of the intermediate forms and followed their progeny to the fourth generation. He found three types, Sativa, Fatua and Intermediate, segregating in a 1 : 2 : 1 ratio. The sativa and fatua types with a few exceptions remained constant.

Zade remarks that a variety of transition forms would result from a crossing of the probable parents, and the reason that only the three types were found is that the original cross took place many years previously and that the generations he studied were not the F_1, F_2, F_3, etc. but should be designated Fx_1, Fx_2, Fx_3, etc. He concluded that the intermediate forms are the result of a cross between A. sativa and A. fatua because:

1. Segregation is a simple mendelian one.
2. There is positive correlation between the number of intermediate forms and the number of wild oats found in the sample.
3. The intermediate forms are numerous, whereas mutations appear only rarely.

Zade questions the observations made by Nilsson-Ehle. In the first place he doubts the absence of wild oat forms in the latter's fields; he also doubts that the pure-lines, which

Nilsson-Ehle said he had obtained and which gave a monohybrid segregation in the F_1, were pure lines in Johannsen's sense of the term; and finally he says that some of the deviations of Nilsson-Ehle arise in varieties which have arisen through crossing.

Zade finally quotes Tschermak's (1914) opinion that the greater frequency of the appearance of intermediate forms which Fischer found in winter oats throws light on the hybridization of oats, viz. that such hybridization is more successful in cooler seasons.

Tschermak (1914) supported Zade's contention that the wild-type oats found in cultivated varieties are the products of natural crossing between A. fatua and A. sativa and opposed Nilsson-Ehle's mutation hypothesis. In later papers (1918, 1929a, 1929b) he discussed more fully the results of some hybridization experiments, trying to demonstrate that a 9 : 3 : 4 ratio was more tenable than a 2 : 1 : 1, the more complicated ratio proving that the origin of the so-called fatuoids was due to natural crossing.

In view of the fact that Crepin (1920) and others have accepted without question Tschermak's statements, it may be well to analyse his work and statements in detail.

Tschermak (1918) proposed a bifactorial arrangement to explain his data. In the F_2 of crosses between wild and cultivated types only three main types segregated out: intermediate, cultivated and wild in the ratio 9 : 3 : 4. The appearance of only three main types was due, he argues, to incompatibility of coexistence between the character "brown glumes" of the wild forms and "glabrous glumes" of the cultivated forms, and to incompatibility of coexistence between the character "hairiness of the glumes" peculiar to the wild forms and the "yellow glumes" character of the cultivated forms.

Later (1929a, 1929b) Tschermak reiterated his earlier hypothesis and attempted to demonstrate that the data of Surface (1916), Garber (1922), Marquand (1922), Goulden (1926), Huskins (1926) and Raum and Huber (1929) fit the 9 : 3 : 4 ratio better than they do the 2 : 1 : 1 (intermediate, cultivated and wild). The "Goodness of Fit" of these alternative hypotheses has been ascertained by the χ^2 test (table 1.).

How Marquand's (1922) results fit the natural crossing hypothesis better than the mutation hypothesis is difficult to say, for he reported finding a homozygous fatuoid which arose directly from a normal type. Huskins (unpublished data) sowed seeds from this homozygous fatuoid and there was no segregation. A careful scrutiny of the literature on this subject failed to reveal a similar report by any other investigator. Assuming

Table 1a.

X^2 Test for Goodness of Fit of Monofactorial Hypothesis.

Investigator		m+x	m	x	x^2	$\frac{x^2}{m}$
Surface	Sativa	117	116.25	.75	.56	.00
	Intermediate	236	232.5	3.5	12.25	.05
	Fatua	112	116.25	4.25	18.06	.16
		465				X^2= .21
						P = .9 to .8
Garber	Sativa	54	74.25	20.25	410.06	5.52
(Victory)	Intermediate	163	148.5	14.5	210.25	1.42
	Fatua	80	74.25	5.75	33.06	.45
		297				X^2= 7.39
						P = .05 to .02
Garber	Sativa	81	64.5	16.5	272.25	4.22
(Garton 784)	Intermediate	122	129	7	49	.38
	Fatua	55	64.5	9.5	90.25	1.39
		258				X^2= 5.99
						P = .1 to .05
Garber	Sativa	135	138.75	3.75	14.06	.10
(Combined)	Intermediate	285	277.5	7.5	56.25	.20
	Fatua	135	138.75	3.75	14.06	.10
		555				X^2= .40
						P = .9 to .8
Huskins	Sativa	76	73.25	2.75	7.56	.10
	Intermediate	153	146.5	6.5	42.25	.29
	Fatua	64	73.25	9.25	85.56	1.17
		293				X^2= 1.56
						P = .5 to .3
Raum and	Sativa	73	76.25	3.25	10.56	.14
Huber	Intermediate	150	152.5	2.5	6.25	.04
(Black Oats)	Fatua	82	76.25	5.75	33.06	.43
		305				X^2= .61
						P = .8 to .7
Raum and	Sativa	121	116	5	25	.22
Huber	Intermediate	226	232	6	36	.15
(White Oats)	Fatua	117	116	1	1	.01
		464				X^2= .38
						P = .9 to .8
Raum and	Sativa	194	192.25	1.75	3.06	.02
Huber	Intermediate	376	384.5	8.5	72.25	.19
(Combined)	Fatua	199	192.25	6.75	39.06	.20
		769				X^2= .41
						P = .9 to .8

Table 1b.

χ^2 Test for Goodness of Fit of Bifactorial Hypothesis.

Investigator		m+x	m	x	x^2	$\frac{x^2}{m}$
Surface	Sativa	117	87.19	29.81	888.79	10.19
	Intermediate	236	261.56	25.56	653.44	2.49
	Fatua	112	116.25	4.25	18.06	.16
		465			χ^2=	12.84
					P =	<.01
Garber (Victory)	Sativa	54	55.69	1.69	2.85	.04
	Intermediate	163	167.06	4.06	16.50	.11
	Fatua	80	74.25	5.75	33.06	.45
		297			χ^2=	.60
					P =	.8 to .7
Garber (Garton 784)	Sativa	81	48.37	32.63	1064.39	22.00
	Intermediate	122	145.12	23.12	534.77	3.68
	Fatua	55	64.5	9.5	90.25	1.39
		258			χ^2=	27.07
					P =	<.01
Garber (Combined)	Sativa	135	104.06	30.94	957.13	9.19
	Intermediate	285	312.19	27.19	739.16	2.37
	Fatua	135	138.75	3.75	14.06	.10
		555			χ^2=	11.66
					P =	<.01
Huskins	Sativa	76	54.94	21.06	443.63	8.07
	Intermediate	153	164.81	11.81	139.54	.85
	Fatua	64	73.25	9.25	85.56	1.17
		293			χ^2=	10.09
					P =	<.01
Raum and Huber (Black Oats)	Sativa	73	57.19	15.81	250.04	4.37
	Intermediate	150	171.56	21.56	464.94	2.71
	Fatua	82	76.25	5.75	33.06	.43
		305			χ^2=	7.51
					P =	.05 to .02
Raum and Huber (White Oats)	Sativa	121	87	34	1156	13.29
	Intermediate	226	261	35	1225	4.69
	Fatua	117	116	1	1	.01
		464			χ^2=	17.99
					P =	<.01
Raum and Huber (Combined)	Sativa	194	144.19	49.81	2481.29	17.21
	Intermediate	376	432.56	56.56	3199.32	7.39
	Fatua	199	192.25	6.75	39.06	.20
		769			χ^2=	24.80
					P =	<.01

that it is not an accidental mixture it is improbable that such would ever occur. It is impossible to explain its origin on the natural crossing hypothesis, but it might be explained on the mutation theory.

Tschermak cites Huskins (1926, p. 324) for Garber's (1922) ratio 163 intermediate : 54 sativa : 80 fatua (which is undoubtedly high in homozygous fatuoids) but fails to mention the ratio Garber obtained in Garton 784 (cited in the same sentence in Huskins' paper) 122 : 81 : 55 (low in homozygous fatuoids), which, as Garber pointed out, together give the ratio 285 : 135 : 135 for which $\chi^2(2 : 1 : 1) = 0.4053$, giving P = .90 to .80. On the basis of Tschermak's natural crossing hypothesis which he tries to substantiate by means of his 9 : 3 : 4 ratio, $\chi^2(9 : 3 : 4) = 11.641$, and P = less than .01 (a very poor fit).

In trying to make out a case for his 9 : 3 : 4 ratio (i.e. more fatua type progeny than sativa) Tschermak failed to notice that the ratios of Raum and Huber and of Garber (the Victory fatuoids) were the only ones cited by him which were higher in fatua than cultivated type progeny. A glance at table 1 is sufficient to show that the ratios of Surface, Huskins, Garber and Raum and Huber are actually higher in sativa-type progeny. He also fails to mention Goulden's remark that "a cytological study of the pollen mother cells of the oat dwarf showed that reduction division is extremely

irregular". Huskins, too, pointed out that cytological irregularities occurred in the reduction divisions of his fatuoids which deviated markedly from the 1 : 2 : 1 ratio.

Tschermak's statement (1929a): "Braune, wirklich glatte Intermediärformen (bei einzelnen Kombinationen, z. B. Dollar (Fahnenhafer) x A v e n a f a t u a, sind auch die braunen I-Formen wiederholt am Rücken nur äusserst spärlich behaart) können nach meinen Erfahrungen nur durch Bastardierungen von braunspelzigen S a t i v a -Formen entstehen oder durch Bastardierungen intermediärer Formen mit solchen oder gelb-, weiss- oder grauspelziger I-Formen untereinander" had already been answered by Nilsson-Ehle (1921) who, after stating that he had investigated carefully the segregation of panicle type, chaff colour, size of grain and shape of grain, etc., says: "Um Kreuzungen mit A. f a t u a als Ursache für das Entstehen der Fatuoidheterozygoten vorauszusetzen, würde man zu der nicht weniger als unsinnig zu bezeichnenden Annahme genötigt werden, das z. B. weisse Haferlinien immer mit weissem Flughafer gekreuzt wurden, gelbe Haferlinien immer mit gelbem Flughafer, Fahnenhafer immer mit Fahnenflughafer usw."

Although Tschermak questions the care with which other workers have classified their material and thus discounts their results and consequent hypotheses, he says of himself: "Ich selbst habe bei der Analyse meiner Bastarde zahlenmässig nur die Ährchenmerkmale analysiert, während ich über die Aufspaltung in Fahne und Rispe, Behaarungsweise der Spelzenbasis, Behaarung der ersten Blätter, Kornform, Höhe usw. nur kurze Notizen gemacht habe, die sich auf beobachtete Konstanz oder

Spaltung in allgemeinen beziehen."

Heribert-Nilsson in 1916 (quoted by Nilsson-Ehle, 1921) constructed an ingenious theory to explain the origin of fatuoids and other "recessive mutations" through segregation of concealed heterozygotes (apparently homozygotes) thus opposing the mutation theory. The strain in which the fatuoids arise cannot be a monomer (monohybrid with respect to the fatuoid complex) because the homozygous fatuoid type does not arise directly. The distinction between the normal and <u>fatua</u> types must therefore consist in two or more complex inheritance units which must be linked since segregation ratios 15 : 1, 63 : 1, etc. are never obtained. Although it does not explain the origin of fatuoids Nilsson-Ehle says it must be taken into account.

According to Heribert-Nilsson the changing heterozygote has arisen in the simplest cases from the union of two gametes Ab x aB. The factors A and B, each alone, produce the normal type, but if both are lacking the recessive "mutant" appears. In consequence of the very close linkage between A and b and between a and B the gamete combinations AB and ab would rarely occur.. The unions Ab x ab and aB x ab give rise to the rare heterozygotes. They would then segregate to give normal types AAbb (or aaBB), heterozygous fatuoids Aabb (or aaBb) and homozygous fatuoids aabb in the simple 1 : 2 : 1 ratio. The theory demands that the segre gating heterozygotes (apparently homozygotes) $\overline{Ab}\ \overline{aB}$ be phenotypically similar to the real homozygotes $\overline{Ab}\ \overline{Ab}$ and $\overline{aB}\ \overline{aB}$.

The difficulty lies, says Nilsson-Ehle, in the fact that the concealed heterozygotes $\overline{Ab}\ \overline{aB}$ must give rise regularly to real homozygotes $\overline{Ab}\ \overline{Ab}$ and $\overline{aB}\ \overline{aB}$ in half their progeny. Since these cannot change further according to the theory, in selecting plants from the progeny of a concealed heterozygote one has a 1 : 1 chance of obtaining a homozygote.

The oat lines at Svalöf were subjected to pedigree selection for from 2-6 years before the occurrence of heterozygous fatuoids was reported in them. It is therefore quite certain that they were pure lines, and Nilsson-Ehle claims that the theory of Heribert-Nilsson is not applicable to the fatuoid mutations reported by him. Nilsson-Ehle admits that fatuoid-like forms can arise through natural crossing with A. fatua. There are indeed, he says, many cases known whereby similar types arise by crossing and by mutation. He is certain, though, that the fatuoids segregating from heterozygotes which he found in Sweden are mutations, and by "mutation" he means nothing more than a spontaneous hereditary modification which is not due to crossing and segregation.

Crepin in 1920 (quoted by Huskins and Fryer, 1925) found an intermediate type plant in a field of cultivated oats. It segregated to give a varied progeny. Crepin concluded that it was a natural hybrid from the cross A. fatua x A. sativa and that natural crossing in oats is probably responsible for much of the degeneration commonly ascribed to reversion.

Åkerman (1921) reports the occurrence of "Fatuoiderna" in pure lines of oats and in mixed populations. Except for the strong, geniculate awn, the basal ring articulation and the tuft of stiff hairs at the base of the seed, the fatuoids were identical with the variety in which they were found. The heterozygous fatuoids segregated in the 1 : 2 : 1 ratio in respect of the fatuoid complex, and because of Nilsson-Ehle's conclusions, and because the hybridization experiments of Tschermak, and Surface (1916), showed a very complicated segregation in the progeny of true crosses between A. sativa and A. fatua, he excludes the possibility of hybrid origin for these fatuoids and accepts the mutation hypothesis.

Marquand (1922), in addition to the homozygous fatuoid, mentioned in the discussion of Tschermak's hypothesis, reported the occurrence of homozygous false wild oats in six different varieties of oats at the Plant Breeding Station, Aberystwyth, Wales. In all cases they were identical with the parent varieties except for the character of the awns, basal articulation and hairiness. The varieties differed widely in many characters including colour and panicle type; two of them belonged to the subspecies orientalis.

Marquand stated that in addition to this well-marked type, hybrids of A. fatua x A. sativa occur. In the F_1 of the cross the solidified base type (sativa) is dominant, but the plant has many of the vegetative characters of the wild parent, and in the F_2 wide segregation occurs. He says it is quite well established that fatuoids have no direct connection with A. fatua.

Garber (1922), whose data have already been mentioned in connection with the natural crossing hypothesis as developed by Tschermak, concludes that a single factor difference is involved in the progeny of heterozygous false wild oats, whereas several factor differences are undoubtedly involved in the fatua x sativa crosses. There is also the possibility, he states, of confusing segregates of a fatua-sativa natural cross with false wild oats. He points out that to accept the natural crossing hypothesis as an explanation for the origin of false wild oats one must account for the selective elimination of all phenotypes except the three: cultivated, intermediate and false wild oats, or one must assume that the progeny of a natural cross do not segregate as do those of an artificial cross.

Garber and Quisenberry (1923) found the character "delayed germination", characteristic of the wild oat forms, to behave as a recessive in the fatua x sativa crosses. Delayed germination was not found in seed from heterozygous false wild, homozygous false wild or sativa plants. This, they say, is direct evidence against the natural crossing hypothesis, and this refutation supports the mutation theory of the origin of fatuoids.

Newman (1923) stated that in Prince Edward Island fatuoids were found by a grower in one strain continuously for over twenty years and no A. fatua plants were present in that particular district. "Analyses of the samples taken annually from the crops in question, however, do not indicate that the

percentage of these forms has increased during all these years," he wrote.

Huskins and Fryer (1925) reviewed the literature on the subject of false wild oats and concluded that "the evidence is overwhelmingly against the theory that false wild oats owe their origin to natural crossing between either Avena sativa and A. fatua, or between cultivated varieties or individuals of A. sativa." They state that Haussknecht's terminology for fatuoids: "Avena fatua var. transiens" is no longer in general usage, that it is misleading and therefore they suggest the name "Avena sativa mut. fatuoida" for all homozygous forms of false wild oats. They used Winge's (1924) chromosome aberration hypothesis to explain the origin of speltoid forms of wheat as a working hypothesis.

Huskins (1926, 1927, 1928a, 1928b, 1932a, 1932b) has carried out a long series of breeding experiments with fatuoid oats and with speltoid wheats. Some of Huskins' 1926 data have been considered in the discussion of Tschermak's hypothesis. His homozygous fatuoids "were of the normal Banner type except for the presence on them of the typical fatuoid characteristics of the grain. They were a uniform group of plants and gave no indication that the strain is other than a pure line."

In his 1927 paper, Huskins groups heterozygous fatuoids into three or possibly four classes or types based on segregation ratios and distinctive cytological conditions. The commonest type

(1) segregates normals, heterozygous and homozygous fatuoids, all of approximately equal vigour, in a ratio of about 1 : 2 : 1 and all segregates have the normal chromosome number (2n = 42) although chromosome irregularities are found fairly frequently in the heterozygous and homozygous fatuoids. Type 2 is characterised by the segregation ratio of approximately 1 normal : 1 heterozygous fatuoid : a few dwarf, sterile homozygous fatuoids. The chromosome complement of this type of heterozygote is 2n = 41. A 41-chromosome heterozygote and a sister plant gave rise to type 3 in which heterozygous fatuoids and normals occur in a ratio which approximates 4 : 1 plus a few sterile dwarf homozygotes, the latter having 40 chromosomes which undergo a very irregular meiosis. Five heterozygous fatuoids of type 3 were found to have 2n = 41, forming $20_{II} + 1_{I}$. A possible type 4 appeared to approach the 1 : 2 : 1 ratio and the chromosome complements of the three segregates were 42, 43 and 44 for normals, heterozygous and homozygous fatuoids, respectively. In type 4, too, the homozygous fatuoids were dwarf and sterile.

Biometrical studies conducted by Huskins failed to show any significant differences in the vigour of the different segregates of type 1, but there were marked differences in vigour in the types which were characterized by chromosome excesses or deficiencies.

Huskins points out the close analogy between fatuoids and speltoids and adapts Winge's (1924) speltoid scheme to interpret

the fatuoid aberrancies.

In discussing the origin of fatuoids and speltoids, he emphasizes that chimaeras furnish direct proof of mutational origin. Speltoid chimaeras occurring in normal wheats have been reported by Åkerman (1927) and Nilsson (1933). Huskins (1928) describes a fatuoid chimaera which he obtained in the progeny of a normal segregate from heterozygous fatuoid Kanota strain 26-13, and mentions one found by Dr. H. Hunter of Cambridge. Not all chimaeras prove to be genetically speltoid or fatuoid, but this is hardly to be expected since sometimes only the superficial layer of cells mutates. (cf. Neilson Jones 1934).

In his earlier work (1927, 1928) Huskins considered whole chromosome changes involved in these abnormal forms although it was stated that the formulae presented were submitted only as a general scheme and working hypothesis. Later (1932, 1933) he pointed out that "the hypothesis that fatuoids arise through chromosome aberrations is, I consider, now so well established for most strains I have studied (including strains of Series A) that it appears to me unnecessary at present to resort to the alternative gene mutation [cf. Jones, Nishiyama - considered later in this review] for those cases in which the evidence of aberration is not yet so clear."

Stanton, Coffman and Wiebe (1926) studied fatuoids in a number of varieties of A. sativa L., A. byzantina C. KOCH and A. nuda L. Their data from Fulghum indicated that the inheritance of the fatuoid form in that variety depends upon several factors.

They found that fatuoids were nore numerous in Fulghum than in other varieties. This is interesting in view of the fact that Fulghum is generally considered to be a derivative of A. sterilis culta (A. byzantina C. KOCH) rather than of A. fatua.

Jones (1927) conducted crossing experiments with white Fatuoid (ex. Golden Rain) x Golden Rain, (a pure line selection of Probsteier oats obtained from Svalöf, see Newman 1912), and the reciprocal cross and found negative correlation between yellow grain colour and the fatuoid characters. The immature fatuoid panicles were characterised by a greenish yellow colour which subsequently disappeared. Jones concluded "the absence of yellow fatuoid types here is not due to inhibitory effects of yellow colour or any factor associated with it, but rather due to the disappearance of yellow in these particular genotypes ."

Jones stated that the absence of yellow colour in this case cannot be considered analogous with the absence of yellow, strongly-awned types as found by Nilsson-Ehle (1914) and by Love and Fraser (1917), because although the results are similar in that they exhibit negative correlation between the development of yellow colour and strong awns, they differ considerably in that in the fatuoid the strong awn is closely linked with the other characters which form the fatuoid complex. Nilsson-Ehle found that the yellow colour persisted whereas the awn development was suppressed.

A modified mutation hypothesis of general application to fatuoids is submitted by Jones (1930) in place of the chromosome aberration hypothesis of Huskins.

Nine varietal strains of fatuoids appeared in established varieties of oats, five in the varieties Fulghum, Orion, Ceirch-du-bach and Royne, two in the varieties Golden Giant and Record, one in the species A. nuda and the variety Cornellian respectively. All fatuoids were of the common, or A series, type. The fatuoid of A.nuda occurred in the line progeny of one of a few plants of this species which had been grown in pots the previous season for hybridization purposes. "The offspring of this particular individual segregated for the characters of hulled and hull-less, fatuoid and non-fatuoid, and for black, grey and white colour of grain. Such complex segregation at once distinguished this type from all the others and pointed to its possible occurrence through natural crossing, probably by stray pollen from some black-grained fatuoid plant."

"The fatuoid of Golden Giant (A. sativa orientalis) ..... unlike the type varieties of other strains, possesses a panicle of unilateral character and leaves which are all eligulate, characters which contrast strongly with the spreading panicle and ligulate leaves, characteristic of the wild oat, A. fatua. The Golden Giant fatuoid nevertheless agreed with its parent stock in being unilateral and eligulate. The grain of this form is fully yellow in colour as in the type variety."

Jones crossed Fulghum fatuoids with Golden Rain fatuoids and from the resulting data concluded that there was a similarity of genotype in respect of the fatuoid characters in these two specific strains. In the F3 of the cross Red Algerian fatuoid x Golden Rain "one of the plant rows was found to consist of 14 panicles bearing grain of a peculiar 'sub-fatuoid' or 'semi-steriloid' type, and 36 panicles bearing grain of either normal or intermediate type".[1] "The occurrence of partial articulation surfaces or planes of cleavage at the apices of the rachillae, and of the strong, twisted and geniculate awn on all grains of the spikelet, indicates a closer affinity to a fatuoid than to a 'steriloid' type of grain; for these reasons this mutant form has been designated 'sub-fatuoid' rather than 'sub-steriloid', or 'steriloid'".

Jones concluded from his studies that a definite association exists between awn, articulation and pubescence in A. fatua, in fatuoids, and in the several aberrant forms occurring in the species A. sativa and A. sterilis culta and their hybrids.

Jones discusses the three hypotheses regarding the origin of fatuoids: natural crossing, mutation and chromosome aberration, and puts forward a modified mutation hypothesis.

1 Except for the „presence of„ an awn on the fourth spikelet, it resembles the "sub-fatuoid" described in this paper.

The differences in breeding behaviour of the fatuoids which occurred in hybrid material and those which arose in pure lines is sufficient to invalidate the natural crossing hypothesis, he argues. In addition, though, "as a possible interpretation of the sub-fatuoid and various 'awned' types, the natural crossing hypothesis is still less satisfactory, for here the aberrant forms are completely different in grain characters from either normal A. sativa or typical A. fatua plants."

The mutation hypothesis, as stated by Nilsson-Ehle on analogy with speltoid wheats, is not applicable to Huskins' B series fatuoids, in the opinion of Jones.

Huskins' (earlier whole) chromosome aberration hypothesis is not applicable to the A series in which all types of segregates possess 42 chromosomes, although it does adequately explain fatuoids of the B and C series. When considered in relation to the sub-fatuoid and strongly awned types the chromosome aberration theory is inadequate, he says. His data indicate that "the factors which determine the different aberrant forms, both fatuoid, sub-fatuoid and awned types, are located in one and the same chromosome, and that these several types have a similar and related mode of origin. There appears, therefore, to be no justification for explaining the origin of fatuoids by chromosome aberration, and of strongly awned types by crossing-over and/or factor mutation."

Accepting the theory of the polyploid origin and di-triploid chromosome constitution of the 42-chromosome species as formulated by Huskins in relation to his chromosome aberration hypothesis, he agrees that the homozygous fatuoid may arise through loss of the "normal" chromosome. "The 'fatuoid' and 'normal' chromosome pairs, however, probably carry factors other than, and additional to, those affecting awn, articulation and pubescence, and therefore, in the absence of the 'normal' chromosome pair, associated differences between the fatuoid and normal segregates in characters other than those mentioned would be expected to appear. Actually there occurs in the B series group (and in the C series also) a reduction in height, tillering capacity and spikelet numbers, and a general lack of vigour and fertility, associated with the fatuoid genotype; but to what extent these associated differences are wholly, or even partly due to the absence of the 'normal' chromosomes, or to the unbalanced condition of the cell arising through chromosome disarrangement and deficiency, it is difficult to say. It is apparent that the 'fatuoid' and 'normal' chromosomes carry factors other than those which determine the fatuoid and normal types of grain, and that they are in consequence not interchangeable in the sense demanded by the chromosome aberration hypothesis."

To bring the above facts into line he suggests a modified mutation hypothesis.

The C or "normal" chromosome, as a result of mutations of different degrees of complexity, C_1, C_2, C_3, etc., would give rise to the several types of heterozygous mutants; for example $\frac{ABC}{ABC_1}$, $\frac{ABC}{ABC_2}$, $\frac{ABC}{ABC_3}$, etc. might represent mutant heterozygotes of different types, "all of which would show simple segregation in relation to the normal type of grain, behave as simple allelomorphs in relation to one another and give simple segregation on intercrossing."

Oehler (1930) reviews the literature on the subject of speltoids and fatuoids but reaches no conclusion concerning their possible origin. In view of the fact that such a variety of "mutant" types have arisen he thinks that some types of commercial value may be found in the progeny of speltoids and fatuoids.

Nishiyama (1931) groups the fatuoids as follows:

Series	Segregation ratio Norm.	Het.	Hom.	Chromosome number Norm.	Het.	Hom.	Chromosome combination of heterozygous fatuoid	Examples
I	1	2	1	42	42	42	$\frac{abc}{abc_1}$	A series
II Type a	1	5-10	few	42	41	40	$\frac{abc}{abo}$	Huskins' B series
b	few	1+	1	42	41	40	$\frac{abc}{abo}$	Type III fatuoids Goulden and Nishiyama
c	1	1+	few					Type II Goulden C series Huskins

Types a and b homozygous fatuoids exhibit very irregular meiotic features, the majority are asynaptic and therefore the plants are highly sterile. "Owing to the deficiency of the c-chromosome pair, morphological differences were observed between the homozygous fatuoids and normals in characters other than the grain type; viz. the diminution in the number of spikelets and culms, in the plant height, etc. From the meiotic behaviour of chromosomes in the three phenotypic segregates, it may be suggested that furthermore the c-chromosome bears a significant factor which controls the normal conjugation of homologous chromosomes." Further investigation is required, however, in order to determine whether or not the factor has a direct effect on chromosome pairing.

Later, Nishiyama (1933a, 1933b) considers it "clearly proved that no synapsis takes place between homologous chromosomes when the c-chromosome or precisely s1 [fragment constituting one end of the c-chromosome], which bears the synaptic gene or genes, is absent." He does not comment on Huskins' (1932) modified chromosome aberration hypothesis because no details have yet been published, but he says "The mutation hypothesis does always hold its validity, until the more substantial evidence is given for the aberration hypothesis" and concludes that A series fatuoids arose through complex gene mutation, and the other fatuoids through chromosome aberration.

Coffman and Taylor (1932) state that Fulghum commonly produces more fatuoids than any other important American oat; results of experiments conducted with Fulghum at 28 agricultural stations in 17 states, showed an average percentage of fatuoids less than 1/4 of 1 per cent. "After four years of continuous selfing of Fulghum oat strains the fatuoid type suddenly appeared in about 0.2 per cent. of the plants, suggesting mutation as the probable cause of its appearance. In these studies the mutation produced the heterozygous fatuoid type and not the fatuoid directly. The Fulghum fatuoid was observed to be a comparatively sterile type highly susceptible to natural cross fertilization with the cultivated form. Nearly 7 per cent. of crossing occurred in one year in open pollinated fatuoid lines. When the fatuoid lines were self-pollinated no aberrant types were observed. In closely allied studies the occurrence of apparent pollination of cultivated oats by A. fatua was observed."

This is direct evidence against the natural crossing hypothesis.

Ru (unpublished thesis, 1933) studied the inheritance of the fatuoid complex in the following crosses: White fatuoid x Swedish select, Swedish Select x Yellow Fatuoid and Yellow Fatuoid x Avena sterilis Ludoviciana No. 4. The fatuoid complex was found to be inherited as a unit. (In the last-mentioned cross the fatuoid characters were found to be completely

recessive.) Ru states that his genetical observations tend to favour the hypothesis of chromosomal aberration to account for the occurrence and segregation of fatuoids. He did not study the inheritance of yellow glume colour.

Philp (1933) studied the genetics and cytology of hybrids between Avena fatua and two varieties of A. sativa, Banner and gigantica. The reciprocal F_1's of fatua x Banner were identical. Apparently no geniculate awns were found on any of the F_1 plants (Philp makes no definite statement concerning geniculate awns in the F_1) but a number of lower grains had weak straight awns. No awns were produced on the upper grains. The cultivated base was partly dominant to the wild. The rachilla on both grains was mostly hairless. The back of the lower grain was almost as hairy as that of the wild parent, but the back of the upper grain was hairless. In the F_1 of fatua x gigantica "one or two hairs were found on the back of a small proportion of the upper grains and also more often on the rachilla on the lower grain. Fewer awns were produced and the earlier panicles were practically awnless." In the F_1 of gigantica x fatua "the hairs on the base and back of the lower grain were longer and slightly more numerous than usual." In other respects "the fatua-gigantica and reciprocal F_1's were identical with the fatua-Banner F_1."

In view of the recent paper by Aamodt *et al.* (discussed later) it is interesting to note that in classifying awn types Philp found it necessary to group the plants into four classes: fully awned, almost fully awned, partly awned and awnless. "The wild type was classed as fully awned. Plants with an awn on practically every lower grain were classed as almost fully awned, the others as partly awned and finally the awnless class." The ratio of the fully awned plants to the others was a good 1 : 3, and when the "almost fully awned" were grouped under the "partly awned" plants a complete correlation was shown between base type and awns. In order to explain the occurrence of almost fully awned cultivated F_2 plants "which again gave a high proportion of awned plants in the F_3" Philp postulated "that awning is affected by one or more modifying factors."

Philp does not mention the occurrence of fatuoids in the progeny of any of his crosses. Although fatuoids occur fairly frequently in Banner oats he does not report finding any among more than 1000 plants in the F_2 and F_3 of the cross *fatua* x Banner and reciprocal. It would seem to the writer that this fact is another in the long chain of evidence against the origin of fatuoids through crossing of *fatua* x *sativa*. It will be seen later that Aamodt *et al.* report finding three bI (white intermediate) and one bW (white wild) plants, which they called heterozygous and homozygous

fatuoids respectively in an F_2 (A. sativa Selection 76 x A. fatua) population of only 69 plants.

That Philp was looking for off-types is indicated in his paper where he reports the occurrence of "an exceptional intermediate plant with pubescence on the rachilla.....in one of the present F_2's. Associated with this unusual condition was a character peculiar to another species - A. sterilis (Pl. XIV, figs. 6-8)." Concerning the breeding behaviour of this exceptional plant he says, "As expected, segregation for base type occurred in the progeny of this plant, but it was found that all intermediates had the pronounced tuft of long hairs on the rachilla, and the sterilis fracture of the upper grain. Plants with cultivated base either had none or a few short hairs, or occasionally a few long hairs on the rachilla, but never the tuft as found in the intermediates. The sterilis type of break was common to all the cultivated plants but was much less pronounced than in the intermediates, and often a number of upper grains fractured in the normal manner. Wild type segregates had the usual characteristics." These observations were verified in the F_4.

Aamodt, Johnson and Manson (1934) investigated natural and artificial hybridization of A. sativa with A. fatua and its relation to the origin of fatuoids. The variety of A. sativa used in the artificial cross was Selection 76 which has "never produced awned grains nor given rise to fatuoids." They consider that their studies "indicate the probability that the common

fatuoid is a normal Mendelian segregate from the crosses in question." They believe "that more or less complete selective elimination of non-fatuoid segregates can be explained" and "that natural hybridization is the usual means by which fatuoids originate" but they "nevertheless entertain the possibilities of origin by chromosome aberration or by gene mutation."

To explain the results of the cross A. sativa Selection 76 x A. fatua the following "hypothetical inheritance scheme" was proposed:

P_1	A. sativa bbggww (bw)	x	A. fatua BBGGWW (BW)
F_1	Black intermediate BbGgWw (BI)		

F_2 12BW : 24 BI : 12Bw : 3GW: 6GI : 3Gw : 1bW : 2bI : 1bw

The symbols represent the following phenotypes:

BW - black fatua BI - black intermediate Bw - black sativa

GW - gray fatua GI - gray intermediate Gw - gray sativa

bW - white fatua bI - white intermediate bw - white sativa

By "intermediate" they mean the form heterozygous for basal articulation and awning.

In the F_2 69 plants were grown and classified as follows:

Phenotype	BW	BI	Bw	GW	GI	Gw	bW	bI	bw	Total
Observed	14	21	19	1	4	6	1	3	0	69
Calculated	12.94	25.87	12.94	3.23	6.47	3.23	1.08	2.16	1.08	69

$\chi^2 = 10.163$ and $P = 0.2547$

The authors then proceed to call the bI and bW type plants heterozygous and homozygous fatuoids respectively ! "The appearance of three heterozygous fatuoid types (bI) and one homozygous fatuoid type (bW) as Mendelian segregates from this artificial cross is especially interesting since it furnishes definite proof that these types can originate as a result of hybridization between A. sativa and A. fatua. Their further appearance in the F_3 as Mendelian segregates from such F_2 plants as BbWw and BbWW makes the proof rather conclusive. In morphological characteristics and in breeding behaviour these 'synthetic' fatuoid types correspond exactly to the common fatuoid described in the introduction to this paper and illustrated in Plate II, C." But, the grains illustrated in Plate II, C are themselves F_2 segregates, not common fatuoids, and further evidence than that presented is necessary to prove that their white fatua segregates "correspond exactly to the common fatuoid" even in grain characteristics alone. It should be emphasized further that no account is presented of the vegetative characteristics of the "fatuoids", despite the fact that identity in all characteristics excepting those comprising (or, one should now add, linked with) the "fatuoid complex" of grain characters has been emphasized by Nilsson-Ehle and others as an essential in the differentiation of sativa-fatua segregates from "true fatuoids".

The validity of conclusions on the hybrid origin of fatuoids might also be questioned when based on a cross involving A. sativa Selection 76 which has "never produced awned grains nor given rise to fatuoids." In this connection it may be noted that the sativa-type progeny from 30 seeds of bI "heterozygous fatuoids" which were kindly supplied by Mr. Johnson all had weak twisted awns on the primary grains when grown at Macdonald College, P.Q. in 1934. The "heterozygous fatuoid" therefore differs from "common fatuoids" in that it does not produce sativa segregates which are identical with the original variety.

The authors mention that "the point which really needs to be explained is not the appearance of the fatuoid, but the more or less complete selective elimination of segregates other than fatuoids." They claim that "the non-fatuoid type segregates are eliminated more or less as a result of selection", but no very conclusive evidence is presented in support of this claim.

In districts where unselected seed is used, they say, field inspectors state that fatuoids have been found with considerable regularity in association with aberrant black and gray off-types. In well-selected seed stocks, however, "fatuoids are usually found unassociated with other off-types." "When selection is practised in a field or plot, the black and gray segregates from a natural cross would be readily observed and removed, the homozygous fatuoid types would also be removed, but the heterozygous

fatuoid type, similar in color to the cultivated parent, would, in many cases, escape notice and be propagated with the selected seed stock."

Colour types are very difficult to distinguish in the field, especially if the plants are still as immature as they usually are at the time when field rogueing is carried out and it is doubtful whether the gray types would be "readily observed and removed."

III. Material and Methods.

Two fatuoid strains have been studied in detail. The Banner strain originated from a single heterozygous fatuoid panicle found in a head-selection plot of Elite Banner Oats in the Department of Field Husbandry Investigation Field, University of Alberta, Edmonton. This is the 24-20 strain which Huskins (1927) investigated. The aberrant "steriloid" form arose in 1928 among the fourth generation (D_4) progeny of the original plant 24-20. Two steriloids were found that year among the progeny of a heterozygous fatuoid (27-438) which segregated 2 normals, 17 heterozygous fatuoids, 1 fatuoid ("homozygous fatuoid" of earlier papers) and 2 steriloids. The four types have been investigated.

The Kanota strain originated from a single heterozygous fatuoid plant obtained in 1926 from Professor John H. Parker, Kansas Agricultural Station, Manhattan, Kansas, U.S.A. Kanota, a selection from the variety Fulghum, is generally considered to belong to the species Avena byzantina C. KOCH (A. sterilis culta MARQ.). This is the 26-13 strain which Huskins (1927, 1928) investigated. In the progeny of 26-316, a heterozygous fatuoid, there appeared a chimaera which segregated 10 fatuoids, 7 sub-fatuoids and 20 steriloids. The fatuoids typically breed true, the sub-fatuoids either breed true or segregate sub-fatuoids and fatuoids and the steriloids either breed true or segregate steriloids and fatuoids. The genetics and cytology of heterozygous fatuoids, sub-fatuoids, steriloids and fatuoids have been investigated and compared with the behaviour of normal Kanota oats.

The plants included in this study were grown by Dr. C.L. Huskins or under his supervision, prior to 1930 at the John Innes Horticultural Institution, Merton, England and since then at Macdonald College, P.Q. where facilities for field work and laboratory studies during the summer months have been kindly provided by the Departments of Agronomy and Plant Pathology respectively.

McClintock's (1929) permanent aceto-carmine method which was used for fixing and staining the pollen mother cells

proved to be very satisfactory. All drawings were made with Abbe's camera lucida, using a 2 mm., 1.3 N.A. Zeiss apochromatic objective and a 10 and 15 x compensating ocular giving magnifications at bench level of ca. 1400 and 2100 diameters. For the photomicrographs a Reichert Photomicrographic Camera, model S (designed by Romeis), was used.

IV. Description of Types.

1. Types in the Banner strain. (Arranged in order of deviation from the Normal type).

Normal Banner (*Avena sativa* var. Banner). (See Plate I, fig. 1 and Plate III, fig. 15)

- awnless, excepting on secondary tillers or on plants grown under unusual environmental conditions, particularly greenhouse plants, which may have strong awns.
- all grains have solid *sativa*-type articulation.
- rachilla hairless.
- no hairs surrounding articulation surfaces.

Heterozygous fatuoid. (See Plate I, fig. 2 and Plate III, fig. 16)

- twisted, geniculate awn on the primary grain only.
- small ring-shaped basal articulation (hereafter called "sucker mouth") on the primary grain only; other grains have solid articulation.

- rachilla hairless.
- short tuft of fine white hairs surrounding the sucker mouth of the first grain.

Steriloid. (See Plate I, fig. 3 and Plate III, fig. 17)

- twisted, geniculate awn on the first two grains, but not on the third.
- sucker mouth (about as large as, but slightly narrower than, that of the "homozygous" fatuoid) on the first grain only; the other grains have solid articulation.
- fine white hairs on both sides of the rachilla culminating in a long tuft at the base of the second and third grains.
- hairs surrounding the sucker mouth (longer than those of the heterozygous fatuoid).

Fatuoid ("Homozygous fatuoid" of earlier papers). (See Plate I, figs. 4 and 5 and Plate IV, figs. 18 and 19)

- twisted, geniculate awn on all grains.
- large sucker mouth articulation on all grains.
- tuft of long hairs on the rachilla immediately below the sucker mouth.
- tuft of short hairs surrounding the sucker mouth of all grains.

2. Types in the Kanota strain. (Arranged in order of deviation from the Normal type).

Normal Kanota. (Avena byzantina var. Fulghum Selection Kanota) (See Plate II, fig. 7 and Plate V, fig. 21).

- weak, straight awn often present on the primary grain.
- all grains have solid sativa-type articulation.
- a few long white hairs often present on either side of the articulation surface of the primary grain.
- rachilla hairless.

Heterozygous steriloid.
Heterozygous sub-fatuoid.
Heterozygous fatuoid. (See Plate II, figs. 8, 9 and 10 and Plate V, figs. 22, 23 and 24).

- weak to medium-strong to twisted, geniculate awn on the primary grain only.
- small sucker mouth articulation on the primary grain only.
- a few long hairs on either side of, to a tuft of medium-length hairs surrounding, the sucker mouth of the first grain.
- few, if any, hairs on the rachilla.

It is not always possible to distinguish the three phenotypes, but typically their degree of deviation from the normal is in the order given above. Their karyotypes and breeding behaviour mark them as distinct types.

Steriloid. (See Plate II, fig. 11 and Plate VI, fig. 25).

- twisted, geniculate awn on the first two grains.
- sucker mouth (nearly as large as that of the fatuoid) on the primary grain only.
- thick tuft of hairs surrounding the sucker mouth.
- hairs on either side of the rachilla culminating in a long tuft at the base of the second grain; base of the third grain (when present) hairless.

The hybrid fatuoid x steriloid is steriloid in phenotype.

Sub-fatuoid. (See Plate II, fig. 12 and Plate VI, fig. 26).

- twisted, geniculate awn on the first three grains but not on the fourth.
- sucker mouth on the first grain; reduced sucker mouth on the second and much reduced on the third grain; almost solid articulation on the fourth grain.
- tuft of long hairs surrounding the sucker mouths of the first two grains.
- hairs on both sides of the rachilla culminating in a long tuft at the bases of the second and third grains.

Fatuoid ("Homozygous fatuoid" of earlier papers). (See Plate II, figs. 13 and 14 and Plate VI, figs. 27 and 28).

- twisted, geniculate awn on all grains.
- sucker mouth articulation on all grains.
- tuft of hairs surrounding the sucker mouth of all grains.
- hairs on the rachilla culminating in a tuft at the base of the second and third grains.

Photographs of Avena fatua spikelet (Plate I, fig. 6) and grains (Plate IV, fig. 20) have been included for comparison with the other types.

V. Observations.

1. The Banner Strain.

The pedigree of the heterozygous fatuoid (27-438), which numbered two steriloids among its progeny of 22 plants, is given in diagram 1; the breeding behaviour of the steriloids (ST) and their progeny through five generations is given in diagram 3 and more complete data from the strain are included in table 2.

The steriloids segregated steriloids and dwarf, sterile fatuoids (F). The steriloids which have been examined cytologically, except two which will be discussed later, were 41-chromosome plants, 20 bivalents and 1 univalent being present at first metaphase. (Text fig. 2 and Plate VII, Photomicrographs 1 and 2). Rather a high degree of non-viability is exhibited in the steriloids, but a comparison with the heterozygous fatuoids and even normals in this strain indicates that it is not an attribute associated particularly with the steriloids. Of the 450 seeds from nine closely related 41-chromosome steriloid plants 158 failed to mature, most of these failing to germinate; 117 of the 292 progeny proved to be steriloids and 175 fatuoids. Neither normals nor heterozygous

fatuoids appeared in the progeny of Banner steriloids.

Of the two exceptional steriloids mentioned above, one (30-114) had 40 chromosomes which regularly formed 19 bivalents and 2 unlike univalents at metaphase I. The univalents did not synapse and their behaviour was not uniform. Text fig. 1 is a camera lucida drawing of an anaphase I configuration showing the two univalents, one of which had split at first metaphase. The 40-chromosome steriloid segregated 28 steriloids and 16 fatuoids.

The other exceptional steriloid ($30\text{-}114_{1/4}$) examined cytologically was the offspring of plant 30-114 and had 42 chromosomes which regularly formed 21 bivalents at first metaphase. Seeds from this plant have been sown this year in order to determine its breeding behaviour.

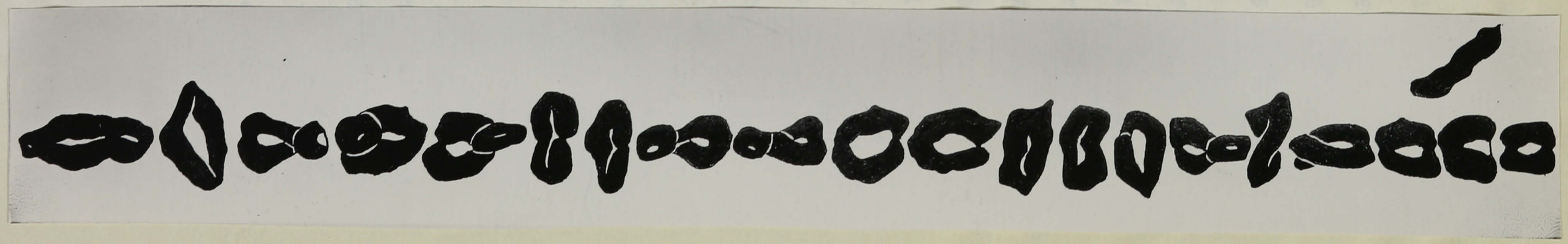
The fatuoid segregates from steriloids were 39- or 40-chromosome plants and were asynaptic and sterile.

In two fatuoids (30-117 and $30\text{-}117_{1/12}$) the meiotic chromosomes were synaptic. On the other hand, two "normals" ($30\text{-}118_{3/6}$ and $30\text{-}118_{4/11}$), closely related to these fatuoids (see diagram 2), were asynaptic and sterile.

The fatuoid 30-117 was 55 inches tall and its 22 fatuoid sibs varied from 12 to 27 and averaged 17.77 ± 4.8 inches

Text fig. 1. First anaphase in a pollen mother cell of the 40-chromosome Banner steriloid. The C_{st} chromosome failed to split.

Text fig. 2. Diakinesis chromosomes in a 41-chromosome Banner steriloid. (cf. Plate VII, Photomicrograph 1). Description in the text, p. 40.



in height. Its chromosome complement was 41 plus a fragment, 20 bivalents, one univalent and a univalent fragment being regularly present at first metaphase. The 12 heterozygous fatuoid sibs varied from 35 to 58 and averaged 51 ± 7.96 inches in height, and three normals in the family averaged 45.6 inches.

Of 48 seeds from 30-117, which were sown the following year, 34 developed into mature fatuoid plants, 22 of which were sterile and 12 fertile. The average height of the sterile plants was 24.73 inches, and of the fertile plants 39.17 inches. Plant $30\text{-}117_{1/12}$ of this family was fertile, 33 inches in height, and its chromosome complement was 20 bivalents and a univalent fragment at first metaphase. Plate VII, Photomicrographs 3 and 4 illustrate typical metaphase I and anaphase I configurations respectively. A sib, $30\text{-}117_{4/2}$, was 18 inches tall, sterile and had 40 chromosomes which failed to undergo synapsis.

It is very difficult to be certain of the chromosome number of asynaptic plants, but the asynaptic normal $30\text{-}118_{3/6}$ appeared to have either 41 or 42 chromosomes. Plate VII, Photomicrograph 5 is an illustration of a typical anaphase configuration. Occasionally chains of chromosomes are formed and fragmentation is also common (cf. Huskins and Hearne, 1933). This plant was 36 inches tall; its normal sib ($30\text{-}118_{4/11}$) was 33 inches tall; the heterozygous fatuoids of the family averaged 35 and the fatuoids / 16.92 inches in height.

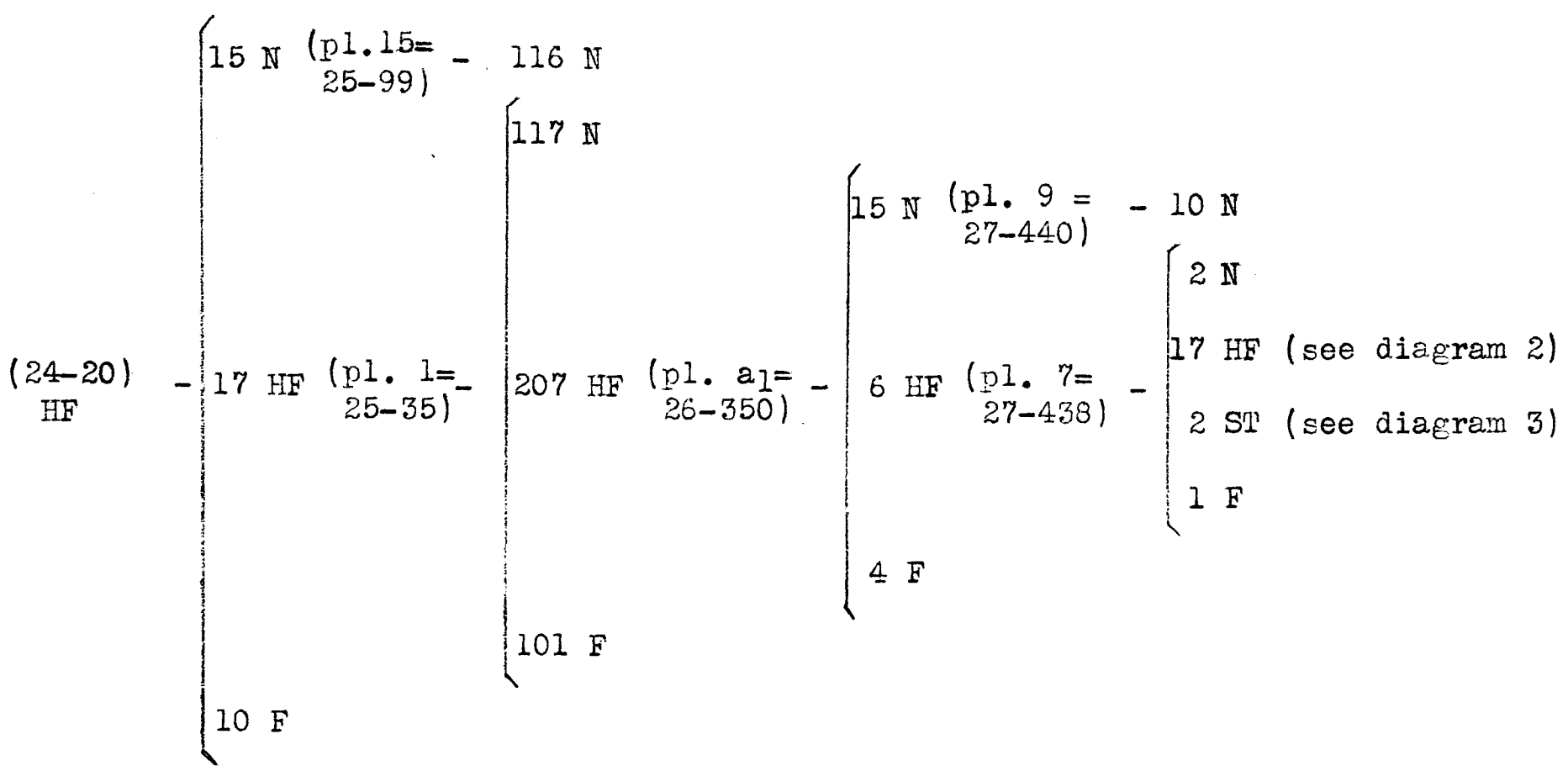


Diagram 1. Pedigree of the two original Banner steriloids.

Symbols: N = normal
HF = heterozygous fatuoid
F = fatuoid
ST = steriloid

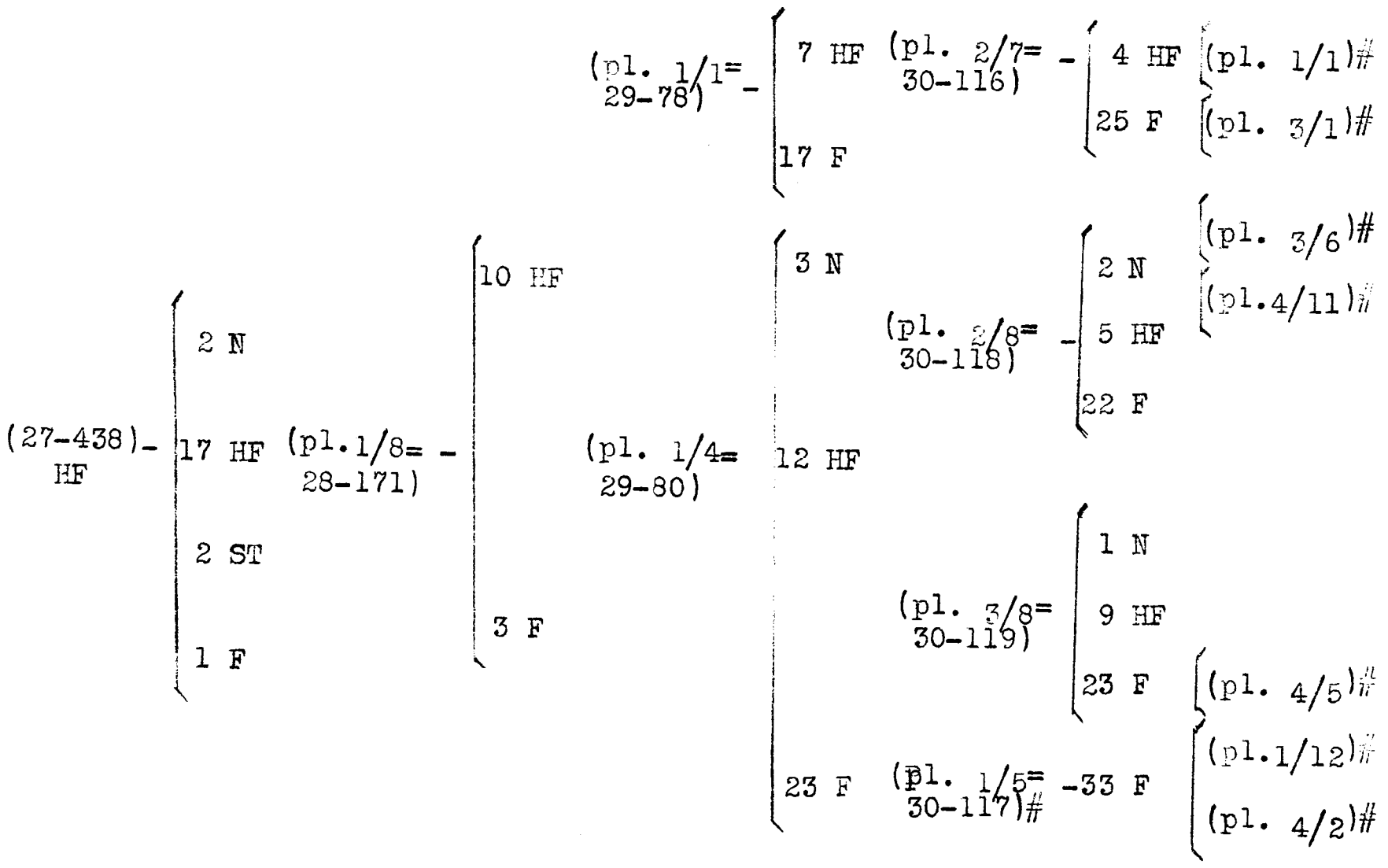


Diagram 2. Pedigree of asynaptic normals and synaptic fatuoids (Banner).

Symbols: N = normal
HF = heterozygous fatuoid
F = fatuoid
ST = steriloid
# indicates plants examined cytologically.

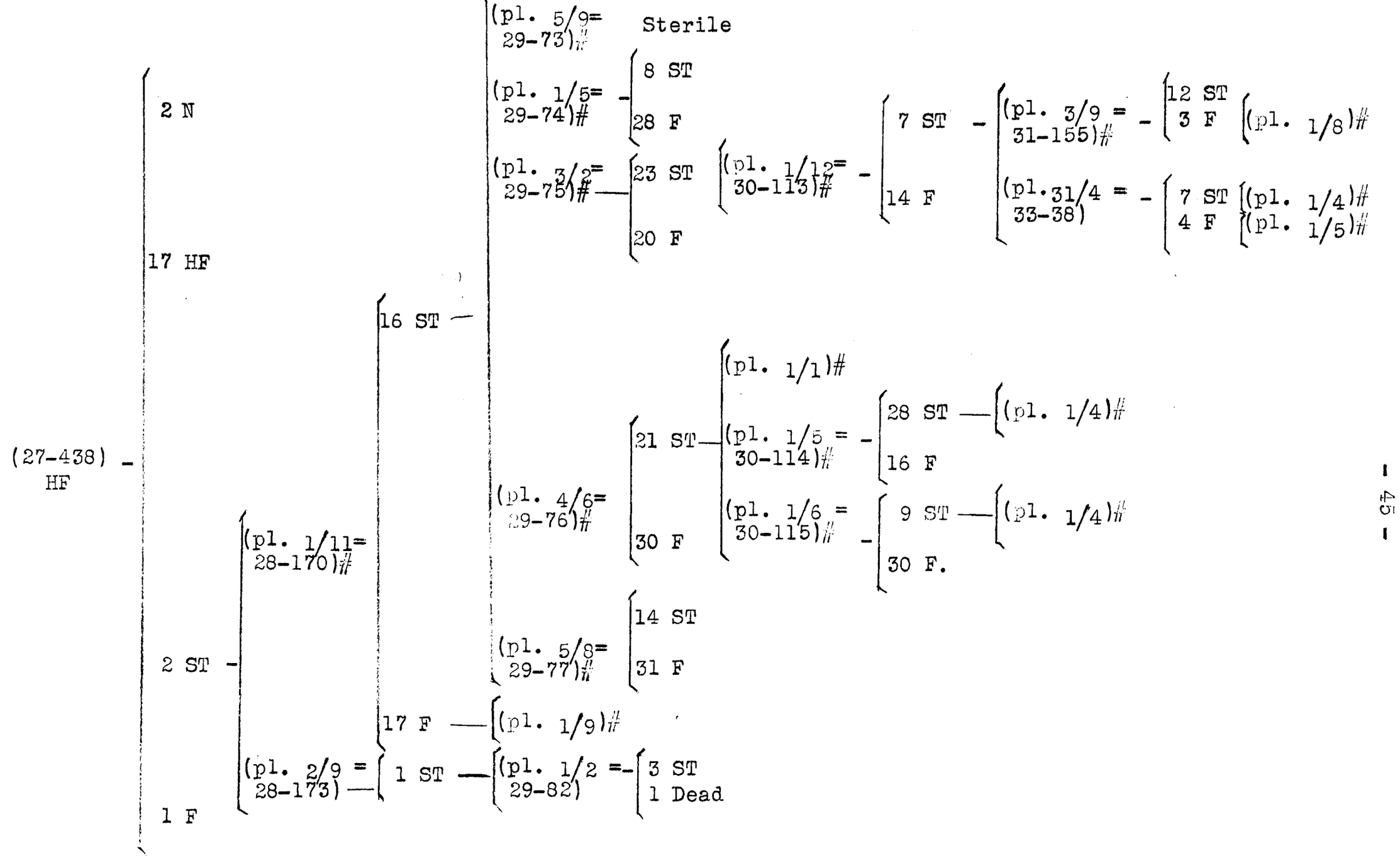


Diagram 3. Breeding Behaviour of Banner Steriloid (ST).

Symbols: N = normal
HF = heterozygous fatuoid
F = fatuoid
ST = steriloid
# indicates plants examined cytologically.

Table 2.

Summarized Record of the Banner Strain.

Accession Number	Parent Plant No.	Chr.		Phenotype	Remarks	Seeds Sown	Progeny N	Progeny H	Progeny ST	Progeny F	Total Matured
25-134	25-35a16			HF	"A" series	48	4	12	-	13	29
26-350	25-35a1			HF	"A" series ex 24-20	48	15	6	-	4	25
27-438	26-3507			HF	18 died early	48	2	17	2	1	22
28-161	26-350-8a3/2			HF		31	11	12	-	4	27
-162	-8b1/9			HF		24	5	11	-	5	21
-163	1/10			N		24	23	-	-	-	23
-164	-8d1/4			HF		24	9	6	-	3	18
-165	2/4			HF		24	9	5	-	3	17
-170	27-4381/11	41?		ST		72	-	-	16	17	33
-171	1/8			HF		24	0	10	-	3	13
-172	2/7			HF	12 died early	24	0	6	-	5	11
-173	2/9			ST		3	-	-	1	0	1
-174	2/11			HF	11 died early	33	0	8	-	2	10
-175	3/2			HF	6 died early	12	0	5	-	1	6
		"	"		" "						
29-60	28-1641/3			HF	C series from A	48	14	18	-	3	35
-61	1/6			N		42	33	-	-	-	33
-62	2/8			HF		116	36	33	-	18	87
-63	2/11			HF		96	17	30	-	8	55
-64	28-1651/3			HF		96	31	39	-	10	80
-65	1/4			F		24	-	-	-	24	24
	1/11	21	II	N							
	4/10	21	II	N							
-73	28-1705/9	41		ST	sterile						
-74	1/5	41		ST		60	-	-	8	28	36
-75	3/2	41		ST		48	-	-	23	20	43
-76	4/6	41		ST		60	-	-	21	30	51
-77	5/8	41		ST		60	-	-	14	31	45
	1/9	40		F	asynaptic						
-78	28-1711/1			HF	10 died early	48	0	7	-	17	24
-80	1/4			HF		48	3	12	-	23	38
-81	28-1721/8			HF		48	0	10	-	13	23
-82	-1731/2			ST	immature	24	-	-	3	0	3
-83	-1741/12			HF		48	0	10	-	18	28
-84	-1751/6			HF		48	1	12	-	12	25
-88	-1831/9			HF		48	8	19	-	13	40

Table 2 (continued).

Accession Number	Parent Plant	Chr. No.	Phenotype	Remarks	Seeds Sown	Progeny N	Progeny H	Progeny ST	Progeny F	Total Mature
30-112	29-603/1		HF		48	0	8	-	22	30
	-683/1	42	HF							
-113	-751/12	41	ST		48	-	-	7	14	21
	-761/2	41	ST							
-114	1/5	40	ST	19 II + 2 I	60	-	-	28	16	44
-115	1/6	41	ST		60	-	-	9	30	39
-116	29-782/7		HF		48	0	4	-	26	30
-117	-801/5	41+f.	F	synaptic	48	-	-	-	34	34
-118	2/8		HF	gave asynaptic Normals	48	2	5	-	26	33
-119	3/8		HF		48	1	9	-	22	32
-120	29-821/11		ST		72	-	-	25	22	47
-121	-831/12		HF		48	0	5	-	27	32
-122	4/8	41	HF		48	0	19	-	12	31
-123	3/3		HF		48	0	13	-	27	40
-124	4/4	41	HF		48	0	6	-	25	31
31-147	25-134-11/4	42	HF		42	15	14	-	2	31
-162	1/5	42	F		24	#1	-	-	10	11
-148	29-882/1		HF		48	12	14	-	16	42
-149	-892/2		HF		48	14	15	-	11	40
-155	30-1133/9	41	ST	3 died early	22	-	-	12	3	15
-169	from 24-20 strain		HF	cf. Nishiyama, 1931	48	0	11	-	21	32
	30-1222/11	42	HF	multivalents						
	2/6	41	HF							
	-1244/9	42+f.	F							
32-22	31-1471/3	42	F		22	-	#1	-	18	19
-23	3/2	42	HF		48	16	19	-	5	40
-24	4/2	42	N		24	11	-	-	-	11
-25	31-1481/1	42	F	multivalents	24	-	-	-	17	17
-26	1/2	42	HF		48	6	23	-	6	35
-27	4/6	42	N		24	19	-	-	-	19
	31-1551/8	40	F	asynaptic						
-39	29-63-22/3		HF		48	4	7	-	5	16
-40	-64-22/11	42	HF	2 univalents	48	0	8	-	18	26
	1/11	42	N							
-41	3/11		F		24	-	-	-	21	21
-48	31-1692/2	41	HF		48	0	9	-	21	30
	32-482/4	41	HF							

Table 2 (concluded).

Accession Number	Parent Plant	Chr. No.	Phenotype	Remarks	Seeds Sown	Progeny N	H	ST	F	Total Matured
33-36	30-112-3 3/1	41	HF		24	1	14	-	7	22
	4/9	42+f	F							
-38	-113-3 1/4		ST		12	-	-	7	4	11
	-114-4 1/4	42	ST	from 40-chromosome steriloid						
	-117 1/12	40+f	F	synaptic semi-tall						
	4/2	40	F	asynaptic, dwarf						
	-118 3/6	42	N	asynaptic						
	-120-4 1/4	40	F	asynaptic, dwarf						
	33-36 2/11	40	F	asynaptic, dwarf						
	-38 1/4	41	ST							
	1/5	40	F	asynaptic, dwarf						
	1/6	41	ST							

Total seeds sown = 2699
Total matured = 1808
Per cent. non-viable = 33

Could not these observations on Bananas be
presented at this stage in terms of the general
? The reader cannot keep mass of observations in
mind and distinguish from those on Kano's which
at least a summary of outstanding points of
[illegible]
[illegible] for the [illegible]

2. The Kanota Strain.

The pedigree illustrated in diagram 4 is that of the chimaera (27-475), which gave rise to three types of offspring: homozygous fatuoid (F), sub-fatuoid (SF) and steriloid (ST).

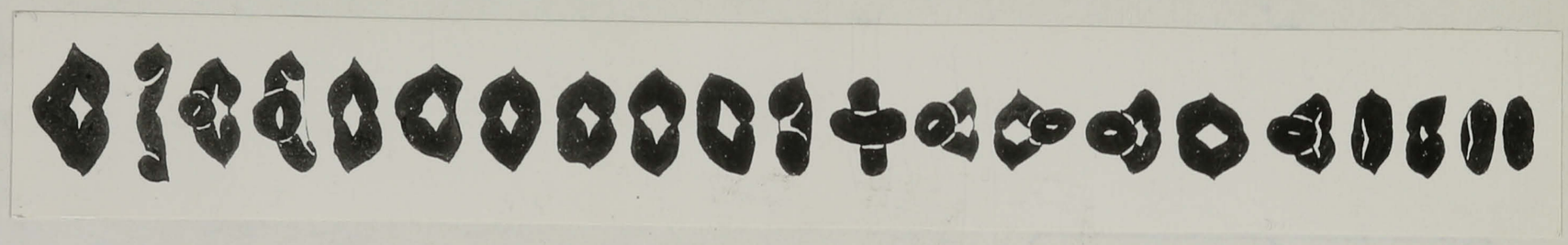
Although 27-475 was a chimaera this was not discovered until after the first progeny were obtained from it. From 24 seeds sown in 1928, 7 homozygous fatuoid, 11 steriloid and 2 plants with spikelets of a new (sub-fatuoid) type were obtained. On examination of the remaining seeds of the original plant both homozygous fatuoid and steriloid type grains were found. Sub-fatuoid plants can be distinguished with certainty from homozygous fatuoids when some spikelets having three or four grains are present but isolated grains of the two are indistinguishable and secondary grains, or spikelets bearing only two grains, cannot always be distinguished. It was therefore impossible to determine whether the homozygous fatuoid type grains were really homozygous fatuoid or sub-fatuoid. Since sub-fatuoids may give some homozygous fatuoid progeny, it is possible that these grains were really all sub-fatuoid, and if this assumption is made the coincident origin of the two types (steriloid and sub-fatuoid) rather than three can, of course, be explained more simply.

The breeding behaviour of the sub-fatuoid plant, 28-232 (= $27\text{-}475_1/_1$) and additional tests of sub-fatuoid grains are included in diagram 5. Similarly, diagram 6 illustrates the breeding behaviour of the steriloids from the chimaera. More complete data from the strain are given in table 3.

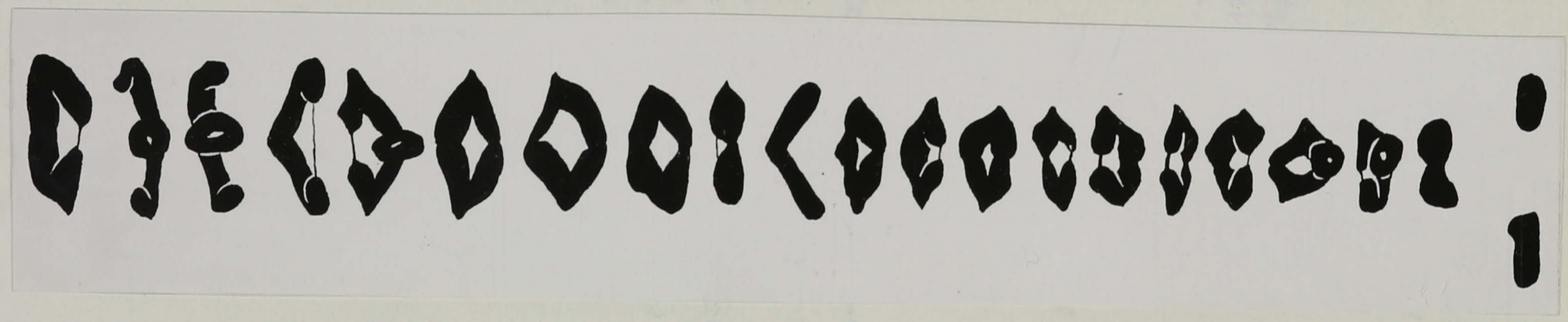
As mentioned above, sub-fatuoids either breed true or segregate sub-fatuoids and fatuoids. Steriloids either breed true or segregate steriloids and fatuoids. The fatuoid segregates from both types are almost sterile but synaptic with 21 pairs of chromosomes. Although not obvious in every pollen mother cell, it appears that a small terminally attached pair of chromosomes is present in these fatuoids (Plate VIII, Photomicrograph 6).

All sub-fatuoids are similar in external phenotype and those examined cytologically have 21 pairs of chromosomes at first metaphase. No heteromorphism has been observed in the chromosomes of true-breeding sub-fatuoids (cf. text fig. 3), whereas those which segregate sub-fatuoids and fatuoids are characterised by the presence of a heteromorphic pair of chromosomes (cf. Plate VIII, Photomicrograph 7, and text fig. 4).

There are also two types of Kanota steriloids, similar in external phenotype but distinguishable both by breeding behaviour and cytology. One breeds true; the other segregates



Text fig. 3. First metaphase chromosomes (21 II) in a pollen mother cell of a true-breeding Kanota sub-fatuoid.



Text fig. 4. First metaphase chromosomes (21 II, including a heteromorphic II) in a pollen mother cell of a segregating Kanota sub-fatuoid. The members of the heteromorphic pair failed to synapse in this cell.

				1st sowing	2nd sowing	Total from chimaera	
26-13 (HF Kanota from J.H. Parker)	8 N		26 N	2 SF	5 SF	7 SF	(see diagram 5)
	19 HF..	(pl.1 = 26-316)	42 HF.. (pl. 6/7= 27-475) chimaera	7 F	3 F	10 F	
	6 F		4 F	11 ST	9 ST	20 ST	(see diagram 6)

Diagram 4. Pedigree of Kanota chimaera 27-475

Symbols: N = Normal Kanota
HF = heterozygous fatuoid
F = fatuoid
ST = steriloid
SF = sub-fatuoid

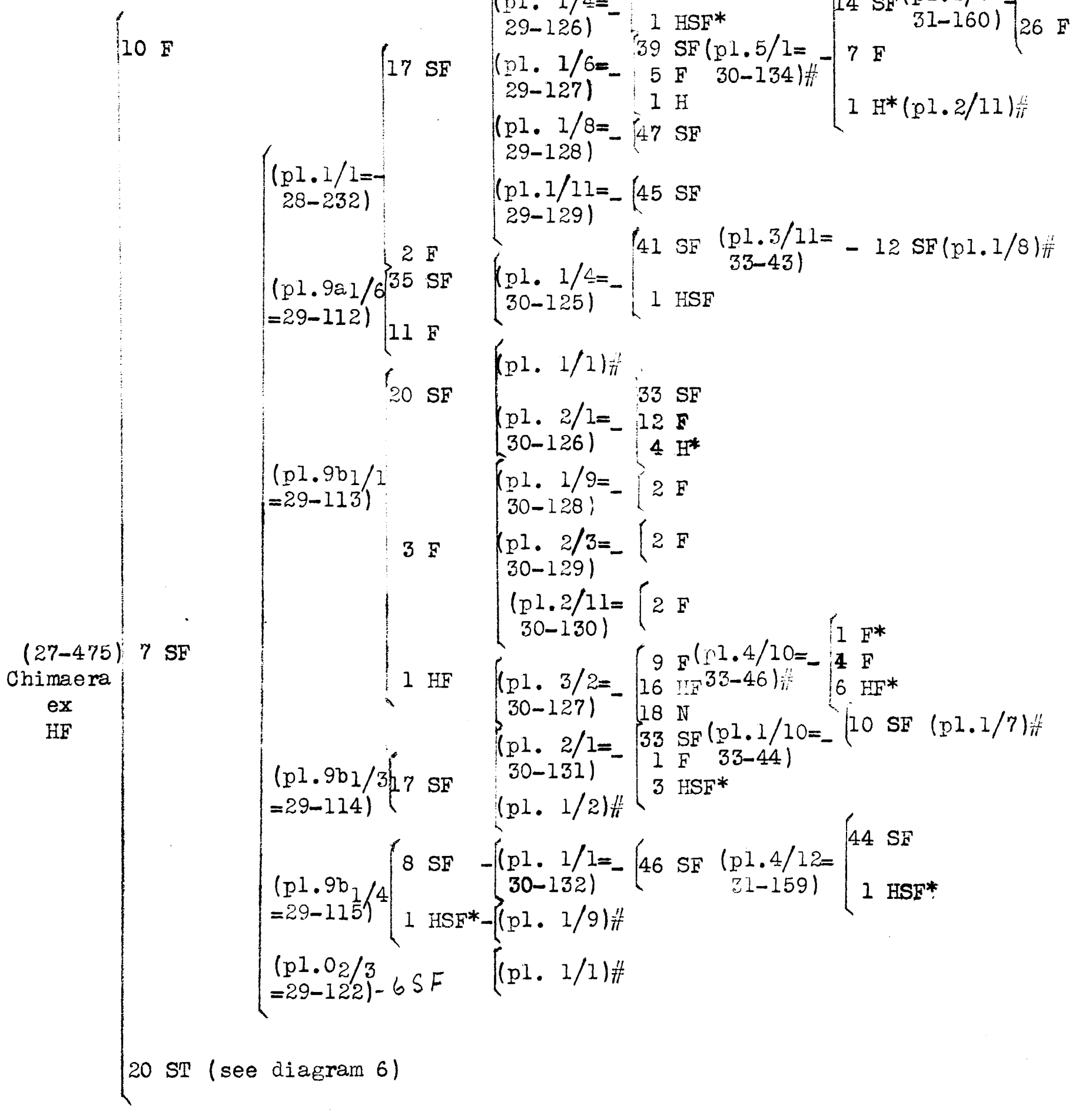


Diagram 5. Breeding Behaviour of Kanota sub-fatuoids (SF)

Symbols: H = heterozygous sub-fatuoid (or fatuoid)
HF = heterozygous fatuoid
F = fatuoid
HSF = heterozygous sub-fatuoid
SF = sub-fatuoid
ST = steriloid
* indicates natural hybrids
\# indicates plants examined cytologically

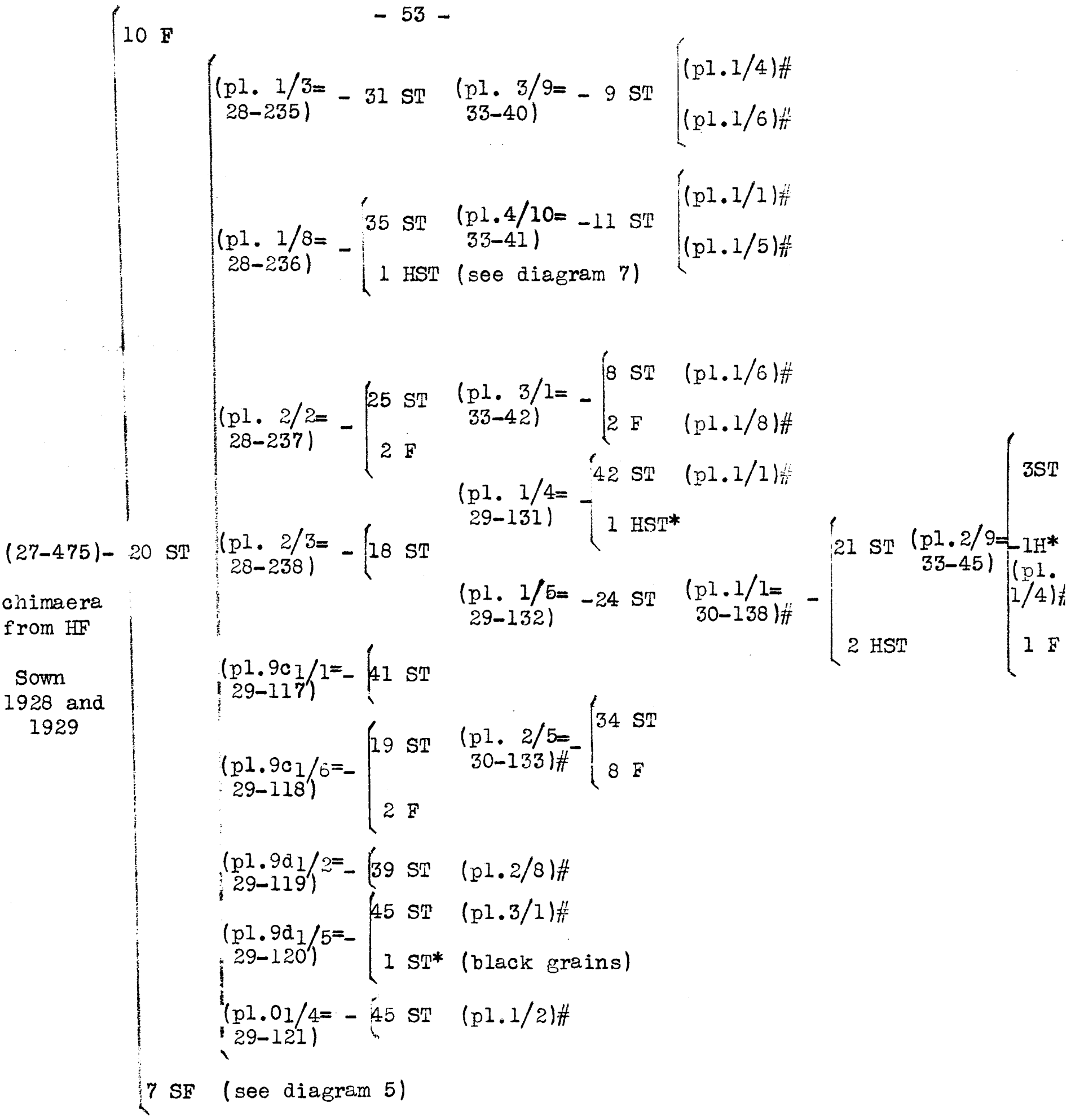


Diagram 6. Breeding behaviour of Kanota steriloids.

Symbols : H = heterozygous (fatuoid or steriloid)
HF = heterozygous fatuoid
F = fatuoid
HST = heterozygous steriloid
ST = steriloid
SF = sub-fatuoid
* indicates natural hybrids
# indicates plants examined cytologically

Table 3.

Summarized Record of the Kanota Strain.

Accession Number	Parent Plant	Chr. No.	Phenotype	Remarks	Seeds Sown	Progeny N	H	ST	SF	F	Total Matured
26-13			H	from J.H.Parker	44	8	19	-	-	6	33
26-316	ex. $26\text{-}13_1$		H		80	24	42	-	-	4	70
27-475	$26\text{-}316_{6}/7$		Chimaera		48	0	0	20	7	10	37
28-232	$27\text{-}475_{1}/1$		SF	F progeny nearly sterile	24	-	-	-	17	2	19
-233	1/2		F		24	-	-	-	-	9	9
-234	2/6		F		24	-	-	-	-	23	23
-235	1/3		ST		37	-	-	31	-	0	31
-236	1/8		ST		36	-	1	35	-	0	36
-237	2/2		ST	F progeny sterile	36	-	-	25	-	2	27
-238	2/3		ST	only 18 seeds	18	-	-	18	-	0	18
29-111	$27\text{-}475\text{-}9a_{1}/2$		F		48	-	-	-	1	42	43
-112	1/6		SF		48	-	-	-	35	11	46
-113	$-9b_{1}/1$		SF	F progeny nearly sterile	30	-	*1	-	20	3	24
-114	1/3		SF		18	-	-	-	17	0	17
-115	1/4		SF	F progeny sterile	12	-	*1	-	8	1	10
-117	$-9c_{1}/1$		ST		48	-	-	41	-	0	41
-118	1/6		ST	F progeny sterile	30	-	-	19	-	3	22
-119	$-9d_{1}/2$		ST		42	-	-	39	-	0	39
-120	1/5		ST		48	-	-	*46	-	0	46
-121	$27\text{-}475\text{-}0_{1}/4$		ST		48	-	-	45	-	0	45
-122	2/3		SF	only 6 seeds	6	-	-	-	6	0	6
-123	2/4		F		42	-	*1	-	-	35	36
-124	$28\text{-}232_{1}/2$		SF		24	-	-	-	21	0	21
-125	1/3		SF	F progeny nearly sterile	36	-	-	-	28	5	33
-126	1/4		SF		22	-	*1	-	16	0	17
-127	1/6		SF	F progeny sterile	50	-	*1	-	39	5	45
-128	1/8		SF		48	-	-	-	47	0	47
-129	1/11		SF		48	-	-	-	45	0	45
-130	$28\text{-}236_{3}/1$		H	from ST	48	11	20	10	-	0	41
-131	$-238_{1}/4$		ST		47	-	*1	42	-	0	43
-132	1/5		ST		24	-	-	24	-	0	24
-136	$28\text{-}240_{2}/3$		H		48	9	22	-	-	17	48

Table 3 (continued).

Accession Number	Parent Plant	Chr. No.	Phenotype	Remarks	Seeds Sown	N	Progeny H	Progeny ST	SF	F	Total Matured.
	29-111$_1$/1	42	F	terminally attached II							
30-125	29-112$_1$/4	42	SF	21 II	48	-	1	-	41	0	42
	-113$_1$/1	42	SF	including heteromorphic bivalent							
30-126	29-113$_2$/1	42	SF	including heteromorphic bivalent F progeny sterile	48	-	**4	-	33	2	39
-127	3/2	42	H	from SF	48	18	16	-	0	9	43
-128	1/9	42	F	dwarf nearly sterile	2	-	-	-	-	2	2
-129	2/3	42	F	dwarf 21 II	2	-	-	-	-	2	2
-130	2/11	42	F	dwarf 21 II	2	-	-	-	-	2	2
	29-114$_1$/2	42	SF	21 II							
-131	2/1	42	SF	21 II	48	-	*3	-	33	1	37
-132	-115$_1$/1	42	SF	21 II	48	-	-	-	46	0	46
	1/9	42	H*	ex SF multivalents							
30-133	29-118$_2$/5	42	ST	heteromorphic II F progeny sterile	48	-	-	34	-	8	42
	-119$_2$/8	42	ST	21 II							
	-120$_3$/1	42	ST	21 II							
	-121$_1$/2	42	ST	21 II							
	-122$_1$/1	42	F	21 II, including terminally attached pair							
	-123$_3$/1	42	F	21 II, including terminally attached pair							
	-125$_3$/1	42	SF	21 II							
30-134	29-127$_5$/1	42	SF	heteromorphic II F progeny sterile	24	-	*1	-	14	7	22
	-128$_2$/1	42	SF								
	- 129$_1$/1	42	SF								
30-135	29-130$_1$/1	42	H	from H heteromorphic II	48	4	33	11	-	0	48
- 136	2/4	42	H	from H heteromorphic II	96	16	46	27	-	0	89
-137	3/8	42	H	from H heteromorphic II	48	9	24	13	-	0	46
	29-131$_4$/2	42	ST	21 II							
30-138	29-132$_1$/1	42	ST	21 II	24	-	2?	21	-	0	23
	-136$_2$/2	42	N	from H 21 II							

Table 3 (continued).

Accession Number	Parent Plant	Chr. No.	Phenotype	Remarks	Seeds Sown	Progeny N	H	ST	SF	F	Total Matured
31-156	30-136 $1/1$		H		24	7	7	4	-	0	18
	-136- $2_{2/4}$	42	H								
	$4/12$	42	H								
31-157	30-137 $3/2$	42	H	including heteromorphic II	24	8	10	5	-	0	23
-158	$4/2$		H		24	6	7	9	-	0	22
	$4/12$	42	ST								
31-159	30-132 $4/12$	42	SF		48	-	*1	-	44	0	45
-160	-134 $1/7$		SF		48	-	-	-	20	26	46
	$2/11$	42	*H	heteromorphic II ex SF							
33-39	28-228 $1/7$		N		12	11	-	-	-	-	11
33-40	28-235 $3/9$		ST		12	-	-	9	-	0	9
-41	-236 $4/10$		ST		12	-	-	11	-	0	11
-42	-237 $3/1$		ST	F progeny sterile	12	-	-	8	-	2	10
-43	30-125 $3/11$		SF		12	-	-	-	12	0	12
-44	-131 $1/10$		SF		12	-	-	-	10	0	10
-45	-138 $2/9$		ST		12	-	*1	3	-	1	5
-46	-127 $4/10$	42	F	ex *H	12	-	*6	-	-	5	11
	33-39 $1/2$	42	N								
	33-40 $1/4$	42	ST	21 II							
	$1/6$	42	ST	21 II							
	-41 $1/1$	42	ST	21 II							
	$1/5$	42	ST	21 II							
	-42 $1/6$	42	ST	including heteromorphic II							
	$1/8$	42	F	sterile							
	$1/9$	42	ST								
	-43 $1/8$	42	SF								
	-44 $1/7$	42	SF								
	-45 $1/4$	42	*H	heteromorphic II							

Total seeds sown = 1984
Total plants matured = 1758
Per cent. non-viable = 10
* indicates natural hybrids

steriloids and fatuoids. Both types have 42 chromosomes which form 21 bivalents at first metaphase. No heteromorphism has been observed in the chromosomes of true-breeding steriloids (cf. text fig. 5), but the second type has a heteromorphic pair (cf. Plate VIII, Photomicrograph 8, and text fig. 6).

For comparison with the aberrant types a pollen mother cell of a normal Kanota oat is illustrated (Plate IX, Photomicrograph 9, and text fig. 7).

Although no natural crossing was noticed in the Banner strain, 22 (1.7 per cent.) of the 1224 plants in the Kanota strain were classified as natural hybrids, their distribution being as follows:

2 among the 308 progeny of 11 true-breeding sub-fatuoids.

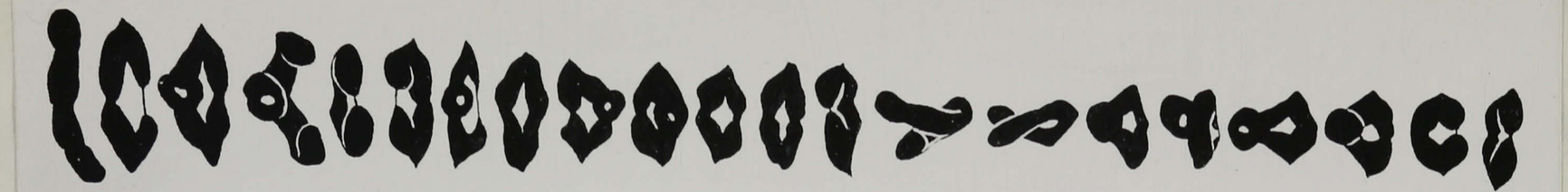
9 among the 321 progeny of 10 segregating sub-fatuoids.

2 among the 366 progeny of 12 true-breeding steriloids.

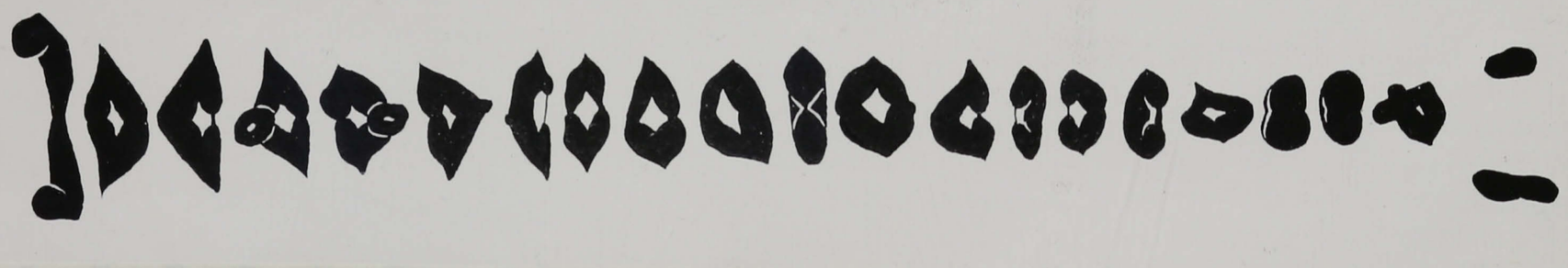
1 among the 106 progeny of 5 segregating steriloids.

1 among the 112 progeny of 6 fatuoids, and 7 among the 11 of one.

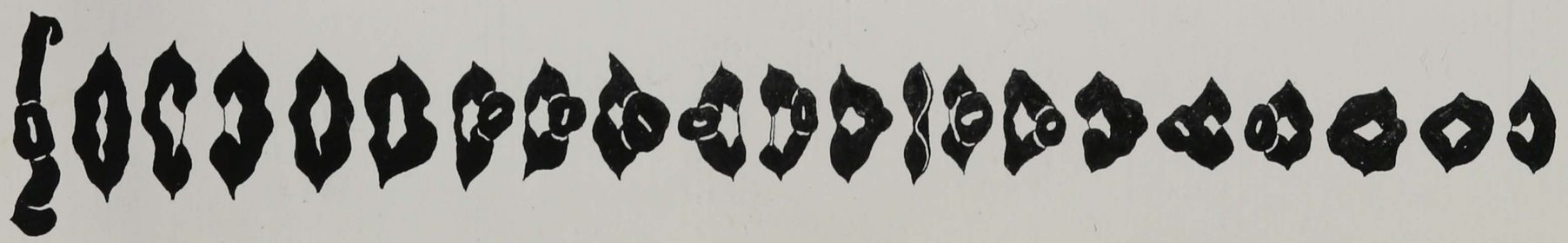
All the natural hybrids from sub-fatuoids were either heterozygous fatuoid or heterozygous sub-fatuoid plants, and a description of each was entered in the Field Book. They were usually distinguishable by a number of external characteristics,



Text fig. 5. First metaphase chromosomes in a true-breeding Kanota steriloid.



Text fig. 6. First metaphase in a segregating Kanota steriloid. The C_{st} and C_f univalent chromosomes failed to synapse in this cell.



Text fig. 7. Normal Kanota.

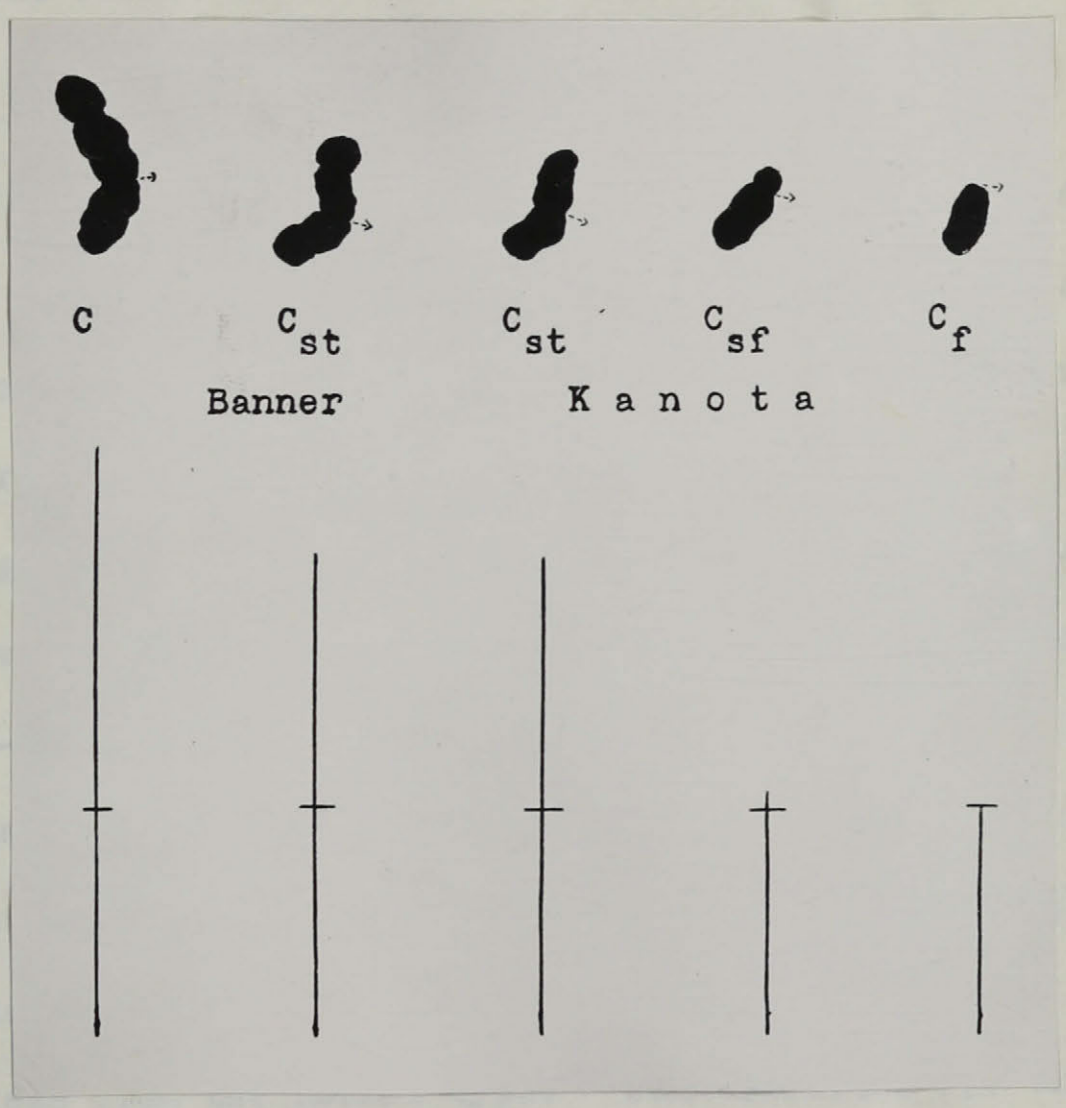
all but one obviously being the result of crossing with Banner or Victory oats (see Plate II, figs. 13 and 14, and Plate VI, figs. 27 and 28). Univalent and multivalent configurations at diakinesis and first metaphase are characteristic of these natural hybrids (cf. Plate IX, Photomicrograph 10, and text fig. 8). The remaining natural hybrid (30-126$_{3/2}$) from a sub-fatuoid bore black, hairy grains typical of a sativa-fatua hybrid.

Two of the three natural hybrids from steriloids were either heterozygous fatuoid or heterozygous steriloid plants. One (29-131$_{4/3}$) had black, hairy grains; the other (33-45$_{1/4}$) bore white grains (Kanota grains are, of course, reddish-yellow) and weak awns. The third, a natural hybrid from a true-breeding steriloid (29-120), was recorded in the Field Book as "steriloid - but black, glabrous backs with long hairs at the base of the spikelet; only two grains per spikelet." This latter was apparently the result of a cross with Black Winter fatuoid, since it was similar to the F_1 of such a cross made artificially.

One natural hybrid from a fatuoid (29-123) was noted in the Field Book as "heterozygous fatuoid type with black, hairy primary grains; black, nearly glabrous secondary grains; hairy bases." Many sativa-fatua segregates were grown in the plot the preceding year providing ample opportunity for a pollen grain carrying "black, sativa-base type" genes to fertilize a fatuoid egg cell.

a b c

Text fig. 8. Multivalent configurations in three cells of a Kanota natural hybrid.



Text fig. 9. The "C" chromosome, normal and aberrant, in Banner and Kanota oats.

Above - metaphase I chromosomes.

Below - proportionate length of the two arms.

See pp. 76 - 79 in the text.

Seven natural hybrids occurred in the progeny of a white fatuoid (33-46), itself the offspring of a heterozygous fatuoid natural hybrid (30-127) from a segregating sub-fatuoid x Banner or Victory cross. Six of these were white heterozygous fatuoid type plants with very weak awns and one was a black fatuoid.

In addition to the obviously hybrid heterozygous types just discussed there were six other heterozygous segregates from sub-fatuoids and steriloids. It is probable that they are the result of fertilization by "normal" pollen grains of normal or heterozygous Kanota plants - see Discussion.

Seeds of one of these (29-130) from a true-breeding steriloid (28-236) have been sown, and also seeds from its heterozygous progeny. The breeding behaviour of the group is illustrated in diagram 7. The vegetative characteristics of all segregates were as uniform as those of other Kanota types grown in those years. Since it appeared in the progeny of a true-breeding steriloid the original heterozygote was called "heterozygous steriloid". Although it is difficult, and sometimes impossible, to distinguish the heterozygous steriloids from heterozygous fatuoids, the former typically deviate less from the normal type. Justification for the term heterozygous steriloid comes from the segregates: normal, heterozygous steriloid and steriloid (not fatuoid, as would be expected from a heterozygous fatuoid).

Four of the heterozygous steriloid descendants of plant 29-130 were examined cytologically. All were 42-chromosome plants with a heteromorphic pair of chromosomes present in the pollen mother cells. Steriloid segregates from heterozygous steriloids were also 42-chromosome plants, but no irregularities in chromosome behaviour or morphology were noticed. Presumably they will breed true; they are being tested this summer.

The breeding behaviour of one of the heterozygous forms from a segregating sub-fatuoid (29-113) is available. It was classified as a natural hybrid, its progeny numbered 18 normals, 16 heterozygous fatuoids and 9 fatuoids.

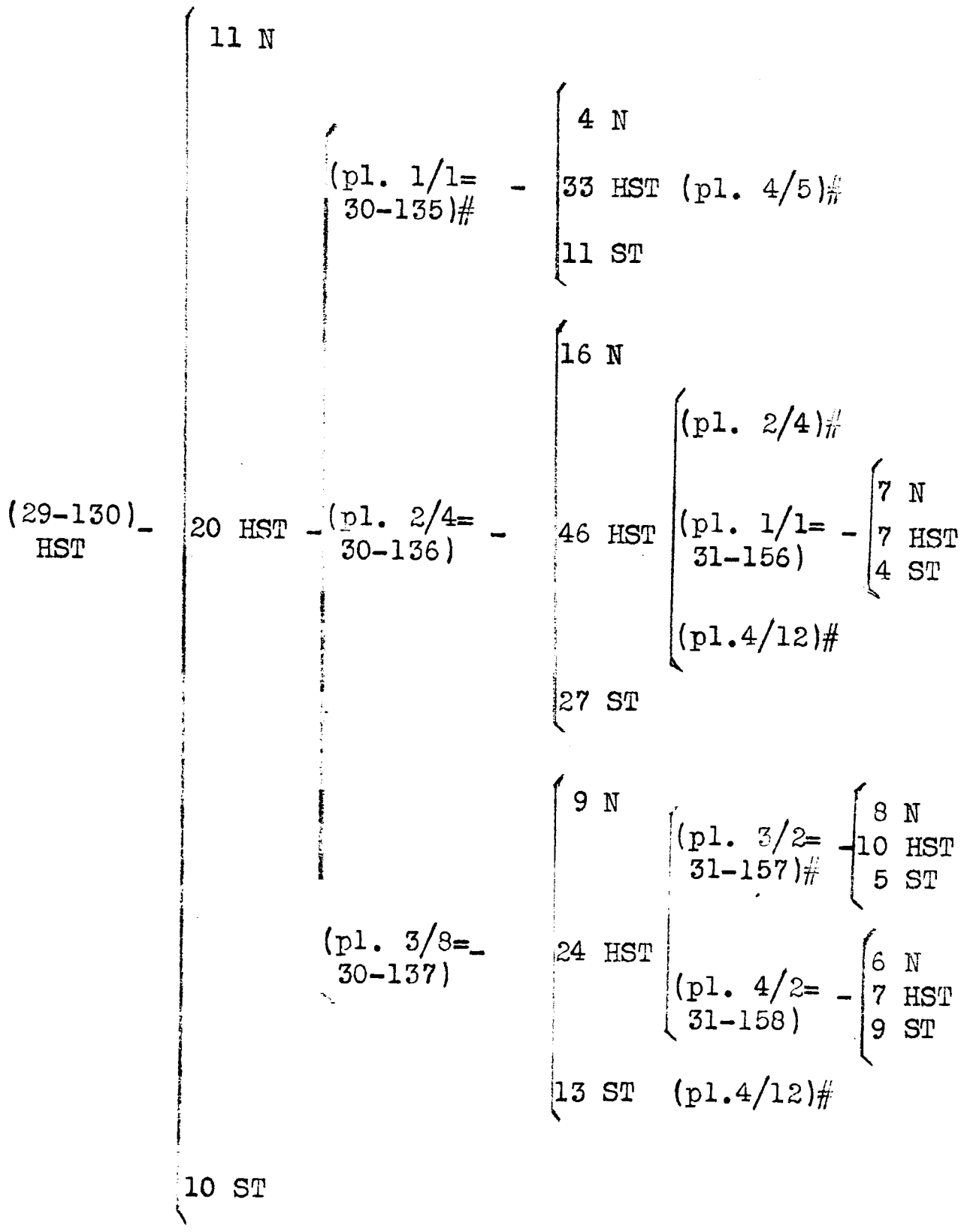


Diagram 7. Breeding behaviour of a "heterozygous steriloid" (29-130) from Kanota steriloid (28-236).

Symbols: N = normal
HST = heterozygous steriloid
ST = steriloid
# indicates plants examined cytologically.

VI. Discussion.

Fatuoids have been found in practically all varieties of A. sativa and A. byzantina. Strangely enough, Fulghum, a variety of the latter species has a higher rate of fatuoid production than A. sativa although A. byzantina is thought to be a derivative of the wild oat A. sterilis. On the other hand, A. sativa may give rise to steriloids, although not as frequently as A. byzantina. In the Banner strain reported herein, the steriloids arose not directly from the normal type but from the heterozygous fatuoid type. In the Kanota strain, the steriloids and sub-fatuoids arose in a chimaera, a segregate from a heterozygous fatuoid.

Speltoid and compactum aberrancies in Triticum vulgare are analogous to the fatuoid and related aberrancies in oats and a theory formulated to explain the origin of one type may be expected to be valid for all. Evidence from all the aberrant types should be used in the formulation of a unified theory.

The cultivated species of oats and wheat mentioned have 42 chromosomes. The aberrant forms may have either the same or different numbers of chromosomes. Most of the 42-chromosome types studied cyto-genetically have a pair of unequal chromosomes as seen by the heteromorphism during meiotic divisions.

The primitive species of oats and wheat have only seven pairs of chromosomes. Recent evidence from several sources on the origin of polyploid forms under observation indicates that the cultivated species of oats and wheat owe their origin to amphidiploidy.

The synthetic formation of naturally occurring specific types as a result of hybridization lends support to the theory of the polyploid nature of oats and wheat. There are many examples but a few will suffice. Florell (1929) reported the occurrence of the wild form A. sterilis from the crosses Coastblack and Fulghum (selections of A. byzantina) x A. fatua "typical fatuna". True-breeding families of A. sterilis were produced in the F_3 generation. Raum (1934) obtained segregating and homozygous speltoids (28 chromosomes) from the cross T. vulgare (42 chromosomes) x T. turgidum (28 chromosomes). Raum considers that the factors which differentiate the wild from the cultivated wheats are not absent in the latter but only suppressed by inhibiting factors which have arisen later in the evolution of the species. Jones (1930) found a "peculiar sub-fatuoid mutant from an F_4 family ex Red Algerian x Golden Rain." Nishiyama (1933c) obtained an F_3 diploid plant (2n=14) with the strigosa genom constitution from a cross A. fatua (2n=42) x A. barbata (2n=28).

Aamodt *et al.* (1934) reported fatuoid types occurring among the F_2 and F_3 segregates of the cross Selection 76 (*A. sativa*) x *A. fatua* although fatuoids have never been known to occur in the Selection used in this cross.

The direct origin of fatuoid and speltoid forms in normal oats and wheat (Nilsson-Ehle, Huskins, Stanton, Coffman and Wiebe, Jones, Coffman and Taylor) indicates that the *sativa* inhibitors have been changed or lost - thus allowing the expression of the various aberrant phenotypes. Chimaeras give definite proof of the origin of the aberrant types by mutation (in the widest sense), (cf. Åkerman, 1927, Huskins, 1928, Nilsson, 1933, and the present paper).

Sapehin (1934), conducting X-ray experiments on wheats, found that the winter varieties of soft wheat give a large proportion of heterozygous speltoids, the proportion and types of speltoid varying according to the variety. From 12 to 55 per cent. appeared in the progeny of different varieties. This is in direct contrast to the mutation rate in polyploids as reported by Stadler (1931). The difference, of course, is that the speltoid mutants of Sapehin were the result of chromosome aberration while those of Stadler were the result of point mutations.

It is evident that hybridization (accompanied by doubling of the chromosomes) has played an important part in the evolution of the economically valuable oats and wheats. In the hexaploids bivalents are formed at meiosis and the genetic behaviour is usually diploid. The occurrence of duplicate factors is common, however, and presumably these have been brought into the polyploids by the different parental species.

Huskins following Winge's (1924) scheme for wheat has put forward a theory on the origin of the hexaploid forms of oats. If the seven chromosomes constituting the haploid sets of the primitive species are designated 1, 2, 3, 4, 5, 6 and 7, then in normal A. sativa the hexaploid constitution is

$$\frac{1_A\ 1_B\ 1_C}{1_A\ 1_B\ 1_C};\ \frac{2_A\ 2_B\ 2_C}{2_A\ 2_B\ 2_C};\ \frac{3_A\ 3_B\ 3_C}{3_A\ 3_B\ 3_C}$$ etc. in which A, B and C

are similar, but not identical. There is both genetic and cytological evidence that only one set of six chromosomes is involved in the differentiation of A. sativa from A. fatua (with respect to the fatuoid complex) and thus the set $\frac{ABC}{ABC}$ only need be considered. Although bivalent formation is the rule in A. sativa, occasionally configurations of three and four chromosomes are seen: thus although A usually pairs with A, B with B, and C with C, B may occasionally pair along part of its length with C. Such pairing results in exchange

of segments or non-homologous crossing-over; it may also result in the loss of one of the chromosomes which has remained unpaired, or in the duplication of chromosomes. In the first case, if the paired segment includes the factors responsible for the fatuoid vs. normal characteristics, a 42-chromosome heterozygous fatuoid plant will appear in the next generation; in the second case, if it is a C chromosome which is lacking, a 41-chromosome heterozygous fatuoid may appear.

In the "B" series fatuoids, as Huskins (1927 and later) and Nishiyama (1931 and later) have observed, 41-chromosome heterozygous fatuoids segregate 42-chromosome normals, 41-chromosome heterozygous fatuoids and 40-chromosome fatuoids. Huskins suggested that the differences in respect of the fatuoid complex be referred to the C chromosome which carries sativa suppressors of fatuoid genes. One set of the sativa suppressors of fatuoid genes (as in the 41-chromosome heterozygous fatuoid) is insufficient to effect the sativa phenotype, but since the heterozygous fatuoid numbers normals among its progeny it must carry a whole C chromosome. The absence of both C chromosomes results in the fatuoid phenotype, also causing asynapsis and diminution of vigour and fertility.

Since the fatuoid progeny of 41-chromosome Banner steriloids have 40 chromosomes and are indistinguishable from the 40-chromosome fatuoid progeny of 41-chromosome heterozygous fatuoids, it is the same or part of the same chromosome (C) which

is unpaired in the 41-chromosome steriloids. The 41-chromosome steriloids have no normal progeny which shows that they do not carry a complete normal C chromosome. If the sativa and steriloid "C" chromosomes are designated C and Cst respectively, segregation in the 41-chromosome heterozygous fatuoids and steriloids would be as follows:

heterozygous fatuoid

♂ / ♀	C	-
C	C C	C -
-	C -	- -

steriloid

♂ / ♀	Cst	-
Cst	Cst Cst	Cst -
-	Cst -	- -

why can't plant be heterozygous

If Mendelian segregation occurred the progeny ratios would be 1 normal (CC) : 2 heterozygous fatuoids (C-) : 1 fatuoid (--) and 1 steriloid (CstCst) : 2 steriloids (Cst-) : 1 fatuoid (--) from 41-chromosome heterozygous fatuoids and steriloids respectively. The segregation is not as simple as this, however, as many investigators have found. Perhaps the primary factor in the modification of the ratios is the low frequency with which the univalent chromosome is included in the pollen grains. Other factors are gametic sterility in the 41-chromosome plants and the low viability of the 40-chromosome fatuoid zygotes either in the seed stage or later.

If univalents divided only once during meiosis and were never lost, segregation of the univalent in a pollen tetrad of a 41-chromosome heterozygous fatuoid or steriloid would result in the formation of gametes with 20 and 21 chromosomes in equal numbers. Frequently, however, the univalent fails to be included in the daughter nuclei and there is a greatly increased proportion of 20-chromosome gametes. Nishiyama (1931) reported the ratio of 20- to 21-chromosome male gametes to be ca. 6 : 1. Assuming the ratio of the female gametes to be the same, and taking into account the percentage sterility of the 41-chromosome plants (ca. 57) - assuming sterility to be due entirely to the death of 40-chromosome zygotes - he obtained a fair agreement between the observed and expected progeny ratios, viz. 1 normal : 12 heterozygous fatuoids : 8 fatuoids.

Huskins and Smith (unpublished data from pollen tetrad observations) found the ratio of 20- to 21-chromosome male gametes to be ca. 30 : 1 in one modified "B" series strain of heterozygous fatuoids; the zygotic ratio was 0 normals : 45 heterozygous fatuoids : 1 fatuoid. On the basis of gametic ratios alone, the expected zygotic ratio would be 1 normal : 60 heterozygous fatuoids : 900 fatuoids. It is patent that gametic ratios alone are insufficient to account for zygotic ratios where irregularities of chromosome behaviour are involved. There is evidently certation and

zygotic sterility also.

It will be recalled that of 450 seeds from nine closely related 41-chromosome steriloid plants 158 failed to mature, most of these having failed to germinate; 117 of the 292 progeny were steriloids and 175 fatuoids. Twelve of these steriloids were examined cytologically and all were 41-chromosome plants. One steriloid, the offspring of a 41-chromosome steriloid, had 40 chromosomes (19 bivalents and 2 unlike univalents) and one of its steriloid progeny had 42 chromosomes (21 bivalents). Phenotypically the three types of steriloids were similar. The steriloid : fatuoid progeny ratios (including some very small families) varied from 1 : 3.5 to 3 : 1 in the 41-chromosome group. Only two of the 41-chromosome steriloids, however, had more steriloid than fatuoid progeny. The 40-chromosome steriloid gave 28 steriloid and 16 fatuoid progeny.

Both in the Banner and the Kanota strain segregating and true-breeding steriloids are indistinguishable phenotypically. The 12 steriloid offspring of 41-chromosome Banner steriloids examined cytologically are a random group, and since the 12 were 41-chromosome plants the 42-chromosome segregates must be rare. The frequent failure of the Cst chromosome to be included in the male gametes would at least partially account for this. The investigation of the behaviour of the univalent and the amount of sterility of the 41-chromosome steriloids is being conducted, but so far, there are not enough data to warrant extensive

conclusions.

In the Kanota strain all the steriloids have 42 chromosomes. Steriloids which segregate steriloids and fatuoids have a heteromorphic bivalent; those which breed true have not. The fatuoid progeny are typically characterised by a short pair of chromosomes with terminal or sub-terminal attachments (cf. Nishiyama, 1933 on the occurrence of a few fatuoids with $20_{II} + 1_{ff}$ in a B series strain). Since this is the only chromosome pair which can be distinguished from any present in normal Kanota oats it appears that it is a part of the C chromosome lacking the sativa suppressors of fatuoid characters. It will be called the Cf chromosome. Exact measurements of the meiotic chromosomes in polyploids are difficult to make, but the Cf chromosome in the fatuoid segregates appears to be the same as the smaller member of the heteromorphic bivalent in the segregating steriloids.

No normals appear in the progeny of Kanota steriloids. The larger member of the heteromorphic bivalent is, on analogy with the univalent in Banner steriloids, the Cst chromosome. The constitution of true-breeding Kanota steriloids is, then, CstCst; that of segregating ones CstCf:

♀ \ ♂	Cst	Cf
Cst	CstCst	CstCf
Cf	CstCf	CfCf

The progeny ratio of the five segregating steriloids in the Kanota strain is 5.2 steriloids : 1 fatuoid (89 : 17 from 138 seeds). The steriloid progeny are either true-breeding or segregating. The former have no heteromorphic bivalent; the latter have one. The Mendelian ratio would be 3 steriloids : 1 fatuoid. The percentage non-viability is almost sufficient to account for the modified ratio. Amongst the progeny of true-breeding steriloids the percentage non-viability is 7.57. Amongst the progeny of segregating steriloids the percentage non-viability is 23.19. If the percentage non-viability due to the death of steriloids is 7.57 that due to fatuoids is then 15.62. Of the 32 non-viable zygotes, then, 10.5 would have been steriloids and 21.5 fatuoids. Thus, instead of the proportion

89 steriloids to 17 fatuoids

we add 10.5 " and 21.5 "

to get 99.5 " to 38,5 "

or the ratio 2.7 steriloids : 1 fatuoid.

All the sub-fatuoids examined cytologically have 42 chromosomes. Those which segregate sub-fatuoids and fatuoids have a heteromorphic bivalent; the true-breeding sub-fatuoids have none. Like the fatuoid progeny of Kanota steriloids, the fatuoid progeny of segregating sub-fatuoids have a short terminally attached bivalent. This is, presumably, the same

chromosome (Cf) present in the Kanota fatuoids from steriloids. No normals appear in the progeny of sub-fatuoids. The larger member of the heteromorphic chromosome is slightly shorter than that in the segregating Kanota steriloids (text fig. 9), which is, in turn, slightly shorter than that in the heterozygous steriloid which has one normal C and one Cst chromosome. The univalent (the C chromosome) present in 41-chromosome (B series) Kanota heterozygous fatuoids has two unequal arms, the proportion of the arms being ca. 1 : 1.6. The larger member of the heteromorphic bivalent in heterozygous steriloids is obviously the C chromosome; the smaller member is almost isobrachial, the ratio of the arms being ca. 1 : 1.1.

Apparently the constitution of the true-breeding sub-fatuoids is CsfCsf; that of the segregating sub-fatuoids CsfCf, segregation in the latter being as follows:

♂ / ♀	Csf	Cf
Csf	CsfCsf	CsfCf
Cf	CsfCf	CfCf

The 10 segregating sub-fatuoids gave 247 sub-fatuoids, 63 fatuoids and 11 heterozygous fatuoids (the latter will be discussed later). Leaving the 11 out of the question for the time being, the ratio is 3.9 sub-fatuoids : 1 fatuoid. The sub-fatuoid progeny are either segregating or true-breeding.

The former have a heteromorphic bivalent, the latter have none. Again the Mendelian ratio would be 3 sub-fatuoids : 1 fatuoid. Treating this ratio as we have done that of the segregating steriloids, we have:

11 true-breeding sub-fatuoids had 308 progeny, 7.78 per cent. of the 334 seeds sown being non-viable.

Of the 368 seeds from 10 segregating sub-fatuoids, 12.77 per cent. were non-viable, only 321 plants maturing.

Per cent. non-viable = 12.77 = 47 zygotes
Per cent. SFs " = 7.78 = 28 zygotes
So per cent. Fs " = 5 = 19 zygotes

Thus, instead of the proportion 247 sub-fatuoids to 63 fatuoids

we add	28 "	and 19 "
to get	275 sub-fatuoids to 82 fatuoids	
or the ratio	3.3 sub-fatuoids : 1 fatuoid.	

Heterozygous steriloids have been mentioned in the Observations. They are phenotypically almost indistinguishable from heterozygous fatuoids, but since they come from steriloids and give normal, heterozygous steriloid and steriloid progeny, (not fatuoids - as heterozygous fatuoids would) they have been called heterozygous steriloids. Typically they deviate very slightly less from the normal phenotype, with respect to the fatuoid complex, than do the heterozygous fatuoids. They are characterised cytologically by a heteromorphic bivalent (text

fig. 9). The distribution of 287 offspring of 7 closely related heterozygous steriloids (see diagram 7) was: 61 normals, 147 heterozygous steriloids and 79 steriloids. On the basis of an expected 1 : 2 : 1 ratio the χ^2 value is 2.28 for which the value of P is between .50 and .30 (Fisher's χ^2 table, 1930). This is good agreement.

Since the heterozygous steriloids have normal progeny they must have a normal C chromosome (as in the heterozygous fatuoids). Since steriloids also appear in the progeny the other chromosome must be Cst (cf. the Cst chromosome in Banner steriloids, and Kanota segregating and true-breeding steriloids). Segregation is, then, as follows:

♀ \ ♂	C	Cst
C	C C	C Cst
Cst	C Cst	CstCst

1 normal (CC) : 2 heterozygous steriloids (C Cst) : 1 true-breeding steriloid (CstCst).

The heterozygous steriloids, as mentioned previously, have a heteromorphic pair of chromosomes, one of which has a median attachment (the Cst chromosome) and the other a sub-median attachment (the C chromosome). The breeding behaviour is precisely like that of the "A" series fatuoids, although in the meiotic chromosomes of the latter no one has yet

published any figures showing heteromorphism.

Nishiyama (1933) states: "The mutation theory does always hold its validity, until the more substantial evidence is given for the aberration hypothesis." He thinks that the "A" series fatuoids owe their origin to gene mutation, and the other series to chromosome aberration. Evidence presented in this thesis on the cytogenetics of heterozygous steriloids indicates that they are of the "A" series type in breeding behaviour, but that chromosome aberration is involved.

Recent work by Painter (1934) and others on the salivary gland chromosomes in Drosophila has shown that any so-called point mutation which has been studied in detail has proved to be, not a point mutation in the commonly accepted sense of the term, but a change in segments of chromosomes, albeit very minute in some cases. This current evidence reverses the statement of Nishiyama quoted above;

Seeds from one heterozygous fatuoid type plant from a segregating sub-fatuoid have been sown and the progeny were normals, heterozygous fatuoids and fatuoids. In this case it is apparent that a pollen grain bearing a normal C chromosome fertilized an egg cell with the Cf fatuoid chromosome to give a heterozygous fatuoid. The breeding behaviour of this heterozygous fatuoid from a segregating sub-fatuoid is further proof that these segregating sub-fatuoids have a

Cf chromosome.

Sufficient segregating steriloids and sub-fatuoids are available to indicate that the occurrence of heterozygous types among their progeny by self-fertilization is not to be expected. It appears that they can arise only through hybridization. The obvious natural hybrids have been discussed previously. With regard to the fatuoid complex they behave as do the heterozygous types which are not obviously natural hybrids, the only difference being that the latter display segregation for other vegetative characteristics while the former do not. It seems, then, that these heterozygous types are also natural hybrids but since they are the result of crossing with Kanota types the criterion of hybridity (segregation of other characteristics)is not available to the classifier.

The aberrant types just reviewed may be brought together here:

(i) in the Banner strain.

$\frac{C}{C}$ = normal A. sativa (42 chromosomes)

$\frac{C-}{C-}$ = asynaptic "normal" (42 (?) chromosomes)

$\frac{Cst}{Cst}$ = steriloid (42 chromosomes)

$\underline{\underline{Cst}}$ = steriloid (41 chromosomes)

$\underline{\underline{C}}$ = heterozygous fatuoid (41 chromosomes)

$\underline{\underline{=}}$ = fatuoid (40 chromosomes) - asynaptic

$\underline{\underline{frag.}}$ = fatuoid (40 chromosomes + fragment) - synaptic

(ii) in the Kanota strain.

$\frac{C}{C}$ = normal A. byzantina (42 chromosomes)

$\frac{C}{Cf}$ = heterozygous fatuoid (42 chromosomes, including a heteromorphic bivalent)

$\frac{C}{Cst}$ = heterozygous steriloid (42 chromosomes, including a heteromorphic bivalent)

$\frac{C}{Csf}$ = heterozygous sub-fatuoid (42 chromosomes, including a heteromorphic bivalent)

$\frac{Cst}{Cst}$ = true-breeding steriloid (42 chromosomes)

$\frac{Csf}{Csf}$ = true-breeding sub-fatuoid (42 chromosomes)

$\frac{Cst}{Cf}$ = segregating steriloid (42 chromosomes, including a heteromorphic bivalent)

$\frac{Csf}{Cf}$ = segregating sub-fatuoid (42 chromosomes, including a heteromorphic bivalent)

$\frac{Cf}{Cf}$ = fatuoid (42 chromosomes, including a terminally attached bivalent) - synaptic, but almost sterile.

Nishiyama (1933) constructed a tentative map of the C chromosome. He suggested that the longer arm carries genes responsible for the synapsis of the meiotic chromosomes and the shorter genes for the cultivated characters of the grain.

While the present work does not show the order of the genes for the sativa suppressors of factors for the various aberrant characteristics the cytological observations indicate that they are borne on the longer arm of the C chromosome and the genes responsible for normal meiosis on the shorter arm. The two arms have the following proportionate length in the different "C" chromosomes reported herein (see text fig. 9):

C ca. 1 : 1.6

Cst ca. 1 : 1.1

Csf ca. 1 : 0.2

Cf ca. 1 : 0.0

Since one arm of the "C" chromosome in all types is about the same length as the shorter arm of the normal C chromosome, it is simpler to assume losses of a segment of the longer arm to account for the aberrant types. This, of course, implies that the sativa genes are borne on the longer arm and that losses of sativa suppressors of genes for the aberrant characteristics result in the new phenotypes.

In "A" series fatuoids the Cf chromosome approximates the length of the normal C, in fatuoids from steriloids and sub-fatuoids it is a short terminally attached chromosome and in the 40-chromosome fatuoids it is lacking altogether.

The Cst chromosome has lost sativa suppressors of genes for the steriloid characteristics, the Csf chromosome the sativa suppressors of genes for the sub-fatuoid characteristics and the Cf chromosome those of both. This interpretation may have to be extended to include further suppressors of fatuoid genes since the fatuoid complex may not necessarily be the sum of the steriloid and sub-fatuoid gene-complexes, but this latter is the simpler assumption and fits all the present data. This interpretation implies, of course, that the steriloid and sub-fatuoid types are the result of a break within the sativa gene-complex.

Since "B" series heterozygous fatuoids usually produce only a few fatuoids and these 40-chromosome, dwarf, asynaptic and sterile, and on the other hand, since segregating steriloids and sub-fatuoids of the Kanota strain produce many fatuoids and these 42-chromosome, mostly dwarf, synaptic but sterile, it appears that there must be fertility factors apart from genes for synapsis.

Origin of the aberrant steriloid and sub-fatuoid types in the present material.

Unfortunately the chromosome constitution of the Kanota heterozygous fatuoid which gave rise to the steriloid-sub-fatuoid chimaera is not known. It belonged to a "C" series strain. It will be recalled that the progeny from the chimaera were steriloid, sub-fatuoid and fatuoid plants, each characterized by a differently modified C chromosome. If the chimaera were a 43-chromosome plant of the constitution CCfCf the new types might have originated through a translocation of part of the C chromosome to one of the Cf chromosomes, followed by somatic segregation to give the chimaerical constitution. That is, one Cf chromosome would split, the C and the other Cf would by translocation be converted to C- and Cf+ which equal respectively Cst and Csf. These then segregate to give CstCf and CsfCf cells. The fatuoids could arise from either of these by normal meiotic segregation and recombination.

Stern (1933) has demonstrated somatic crossing-over and segregation in Drosophila. He found that crossing-over occurs at the four-strand stage between two strands only, that somatic segregation follows somatic crossing-over and that somatic segregation may lead to somatic reduction. If the chimaera in question were a 42-chromosome plant of the

constitution CCf and somatic crossing-over between the C and Cf chromosomes occurred early in the development of the chimaera, the normal C chromosome must have been eliminated from the germ tract since no normals or heterozygous fatuoids occurred in the progeny. Somatic crossing-over at the 4-strand stage would, in this case, have produced daughter chromosomes C, Cst, Csf and Cf.

If either of these explanations is correct, steriloid and sub-fatuoid are complementary types. This will be tested by crossing a true-breeding steriloid with a true-breeding sub-fatuoid. The F_1 should be heterozygous fatuoid in phenotype since a plant of the constitution CstCsf is heterozygous for each of the suppressors of steriloid and sub-fatuoid. The F_2 should be true-breeding steriloids and sub-fatuoids and heterozygous fatuoids; fatuoids and normals would be expected only if crossing-over occurs between the +st and +sf loci.

The Banner strain, 24-20, was of the "A" series type, but it gave rise to "C" and "B" series heterozygous fatuoids in succeeding generations. The heterozygous fatuoid plant 25-35 (ex 24-20) was of the "A" series type and 26-350 (ex 25-35) was of the "C" series type. The latter gave rise to a typical "B" series plant, 27-438, the heterozygous fatuoid which numbered two steriloids among its progeny. The heterozygous fatuoids of succeeding generations from 27-438 were

modified "B" series plants with 41 chromosomes, giving very few normals, some heterozygous fatuoids and many fatuoids like the "B" series strain obtained by Nishiyama (1933) from this original "A" strain. Plant 29-80 (ex 28-171 ex 27-438) gave at least one synaptic fatuoid with 41 chromosomes plus a fragment chromosome and one sib heterozygous fatuoid which segregated two asynaptic "normals". The foregoing is enough to indicate that chromosome aberration is common in this strain.

Two 41-chromosome steriloids occurred among the progeny of the "B" series (presumably 41-chromosome) heterozygous fatuoid 27-438, the pedigree of which has been outlined above. It is known that the unpaired chromosome in heterozygous fatuoids of the "B" series is a whole C chromosome; it is also known that heterozygous steriloids (Kanota) carry a whole C chromosome and that its pairing partner is the shorter Cst chromosome with median attachment. It seems logical to infer that the unpaired chromosome in segregating 41-chromosome Banner steriloids is also the Cst chromosome and that it has arisen through somatic loss late in ontogeny of that segment of the C chromosome which bears sativa suppressors of the factors necessary for the expression of the steriloid complex.

While the possibility of a hybrid origin of the two steriloids cannot be ruled out entirely, the fact that only two have arisen in the thousands of Banner plants grown in this Department in ten years and their occurrence in one family makes the mutation hypothesis much the more plausible. Furthermore, in the progeny of these steriloids there was no segregation for other vegetative characteristics, all segregates being typical Banner oats except for the steriloid and fatuoid complexes. Also, apart from the unpaired chromosome meiosis is completely normal in these steriloids, whereas in hybrids, even of varietal crosses, of oats and wheat irregularities are frequent.

It will be noticed that throughout this discussion no mention has been made of the loci of genes for the expression of fatuoid, sub-fatuoid and steriloid characteristics. Surface (1916) concluded that the so-called fatua characters comprised one linkage group. Nishiyama (1933) criticizing this, wrote: "If all the cultivated genes are completely linked the fatua or fatuoid characters may show a pseudo-linkage, although they are separately carried on different chromosomes." Philp (1935) disagrees with Nishiyama, however, for he says: " . . . factors which are not carried by pairing chromosomes cannot show linkage or pseudo-linkage. The differences between two races or

species can only be expressed in terms of differences between pairing chromosomes and the analysis of hybrids can never expect to provide a means of telling the differences between non-pairing chromosomes.

"In the F_2 of *sativa-fatua* hybrids normal meiosis and vigour is the rule and thus it must be concluded that *A. fatua* has a chromosome carrying factors for meiosis which pairs with the C chromosome of *A. sativa*. An alternative would be to suppose that shift occurred owing to intraspecific pairing but there is no evidence to show that *A. sativa* has another pair of chromosomes (in addition to the C chromosomes) carrying factors for meiosis. Since the chromosome of *A. fatua* is similar in this respect to the C chromosome it seems logical to suppose that it also carries a group of factors for the *fatua* characters."

It might be pointed out, however, that in the mutant types from normal oat strains there is only one pair of C chromosomes and there is no evidence demanding the conclusion that *A. sativa* carries a *fatua* or fatuoid chromosome. Rather is the fatuoid phenotype allowed expression when *sativa* inhibitors are lacking. Thus the fatuoid genes may exhibit a pseudo-linkage as Nishiyama has remarked.

In the same paper Philp states that "the C chromosome may be regarded as the key chromosome for the evolution of the

species for the following reasons. First, it carries genes affecting meiosis (hence fertility) and vigour; secondly, it carries genes controlling the principal characters by which the species is differentiated - the *sativa* complex; and thirdly, no crossing-over occurs within the *sativa* complex in hybrids with *A. fatua* (Surface, 1916; Philp, 1933)."

Philp thinks "that in every known respect the evolution of the C chromosome appears to be analogous to that of the sex chromosomes which have a group of special genes located on them and have differential pairing segments." This is a thought-provoking statement. The expression of sex in Drosophila is due to the balance set up between the X chromosomes and the autosomes. The X chromosome carries genes for femaleness; its pairing partner, the Y chromosome, does not carry genes for the alternative character, maleness. The situation in the mutant fatuoid (and speltoid) forms is analogous in that the *sativa* phenotype is due to the balance between *sativa* genes on the C chromosomes and the remainder of the genotype. The aberrant phenotypes are due to changes in the balance brought about by losses of *sativa* genes. The *fatua* genes *may* be on the C chromosomes of *A. fatua*; they may be possessed in common on another chromosome of *A. fatua* and *A. sativa*, or they may be distributed throughout the

genotype apart from the C chromosome of A. sativa; all we know definitely as yet is the location of the sativa inhibitors of fatua or fatuoid genes.

VII Conclusions.

The conclusions may be summarized as follows:

1. Changes in the C chromosome are primarily responsible for the various aberrant types: fatuoid, heterozygous fatuoid, true-breeding steriloid, segregating steriloid, heterozygous steriloid, true-breeding sub-fatuoid, segregating sub-fatuoid and heterozygous sub-fatuoid.

2. The aberrant types are due to losses of different sativa factors epistatic to fatuoid, sub-fatuoid, or steriloid factors.

3. Nothing is definitely known of the loci of the fatuoid, sub-fatuoid or steriloid genes: there is no clear evidence that the fatuoid genes are carried on the B chromosome, or the steriloid genes on the A chromosome (contra Philp, 1933).

4. The sativa complex is carried on the longer arm of the C chromosome and genes, the presence of which are necessary for normal meiosis, on the shorter arm (contra Nishiyama, 1933). In addition to the sativa complex the longer arm may bear genes for fertility.

5a. The *sativa* phenotype is due to the balance between the *sativa* genes on the C chromosome and the remainder of the genic complement.

b. Aberrant phenotypes are due to a change in this balance, brought about through loss of *sativa* genes.

6. The Kanota aberrant steriloid and sub-fatuoid types arose through somatic aberration followed by somatic segregation; the heterozygous steriloids and heterozygous sub-fatuoids arose through natural crossing.

7. The Banner steriloids most probably arose through somatic loss of a segment of the C chromosome bearing suppressors of steriloid genes.

8. Synaptic fatuoids (40 + fragment) and asynaptic "normals" (Banner) have arisen through chromosome aberration.

VIII Summary.

In the Banner (*Avena sativa*) and Kanota (*A. byzantina*) oat strains studied, the aberrant types are correlated with distinctive cytological conditions as exhibited by the meiotic chromosomes. The modified chromosome aberration theory of Huskins is adequate as a general explanation of all types, but the cause of the changes in phenotype can be referred to differences in the C chromosome alone. It is unnecessary to refer then to mutation of several genes of the *sativa* complex which would entail a very high degree of coincidence.

The simultaneous origin of the aberrant steriloid and sub-fatuoid types in a Kanota chimaera, associated with readily observable changes in the C chromosome substantiates the chromosome aberration theory. The changes in this case are probably due to somatic crossing-over accompanied by segregation. In the case of the Banner steriloid where the original form was monosomic, the change may have been due to simple fragmentation. Additional evidence of fragmentation is found in the exceptional synaptic fatuoids and asynaptic "normals" from 41-chromosome Banner heterozygous fatuoids.

The C chromosome in *A. sativa* bears the factors +sub-fatuoid, +steriloid, +sterility on the longer arm and +asynapsis and sterility on the shorter arm. It is suggested that the *sativa* phenotype is produced by the balance set up between *sativa* genes on the C chromosome and

the remainder of the genotype. The aberrant phenotypes are then due to changes in this balance brought about through losses of segments of the C chromosome bearing sativa suppressors of genes for fatuoid and steriloid characteristics. The simplest interpretation of the present data is that fatuoid is the sum of sub-fatuoid and steriloid and that these types arose through a break within the sativa complex.

IX Acknowledgements.

The writer wishes to record his indebtedness to Dr. C. Leonard Huskins for placing research material at his disposal and for helpful and stimulating criticism throughout the course of this investigation; and to the National Research Council of Canada for the award of a scholarship.

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XI. Description of Plates.

Plates I and II: Spikelets of the oat types described on pp. 36 - 40. (*ca.* natural size)

Plate I.

Fig. 1. Normal Banner.

Fig. 2. Heterozygous fatuoid.

Fig. 3. Steriloid.

Fig. 4. Fatuoid (sterile) from heterozygous fatuoid.

Fig. 5. The same, from steriloid.

Fig. 6. *Avena fatua*.

Plate II - Kanota.

Fig. 7. Normal Kanota.

Figs 8, 9, and 10. Heterozygous types.

Fig. 11. Steriloid.

Fig. 12. Sub-fatuoid.

Fig. 13. Fatuoid from heterozygous fatuoid.

Fig. 14. The same, from steriloid.

Plates III - VI: Seeds of the oat types described on pp. 36 - 40. (magnification *ca.* 2 x)

Plate III - Banner.

Fig. 15. Normal Banner.

Fig. 16. Heterozygous fatuoid.

Fig. 17. Steriloid.

Plate IV.

Fig. 18. Fatuoid from heterozygous fatuoid (Banner).

Fig. 19. The same, from steriloid (Banner).

Fig. 20. Avena fatua.

Plate V - Kanota.

Fig. 21. Normal Kanota.

Figs 22, 23, and 24. Heterozygous types.

Plate VI - Kanota.

Fig. 25. Steriloid.

Fig. 26. Sub-fatuoid.

Fig. 27. Fatuoid from segregating steriloid.

Fig. 28. The same, from segregating sub-fatuoid.

Plates VII and VIII - Photomicrographs.

Plate VII.

1. Diakinesis in a pollen mother cell of a 41-chromosome Banner steriloid. (cf. text fig. 2)
2. The same.
3. Metaphase in an exceptional fertile Banner fatuoid with 40 chromosomes and a fragment chromosome (20 II + fragment I).
4. Anaphase in the same plant.

   3 and 4 illustrate the effect of the fragment on meiosis.
5. Meiosis in an asynaptic "Normal" Banner plant. See page 42.

Plate VIII.

6. First metaphase in a pollen mother cell of a Kanota fatuoid from a segregating sub-fatuoid. Note the terminally-attached C_f chromosome pair. See p. 50.

7. Early anaphase of the first division in a segregating sub-fatuoid (Kanota). The C_{sf} - C_f chromosome pair have separated prematurely.

8. Early metaphase in a segregating steriloid(Kanota). The C_{st} - C_f chromosome pair lies at the right.

9. Metaphase in Normal Kanota. (cf. text fig. 7.)

10. Illustrating multivalent configurations common in natural hybrids (Kanota heterozygous types). (cf. text fig. 8a)

PLATE I

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PLATE II

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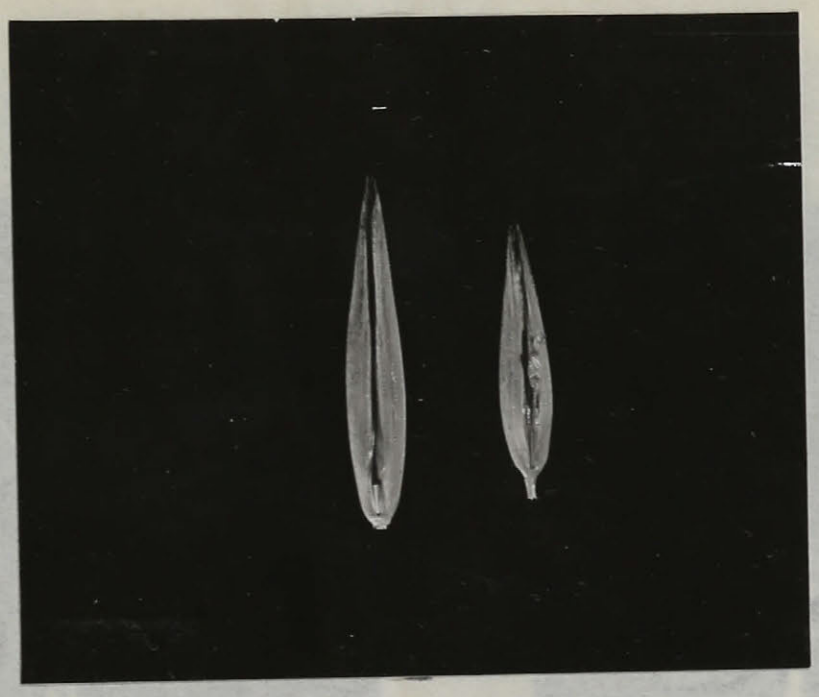
PLATE III



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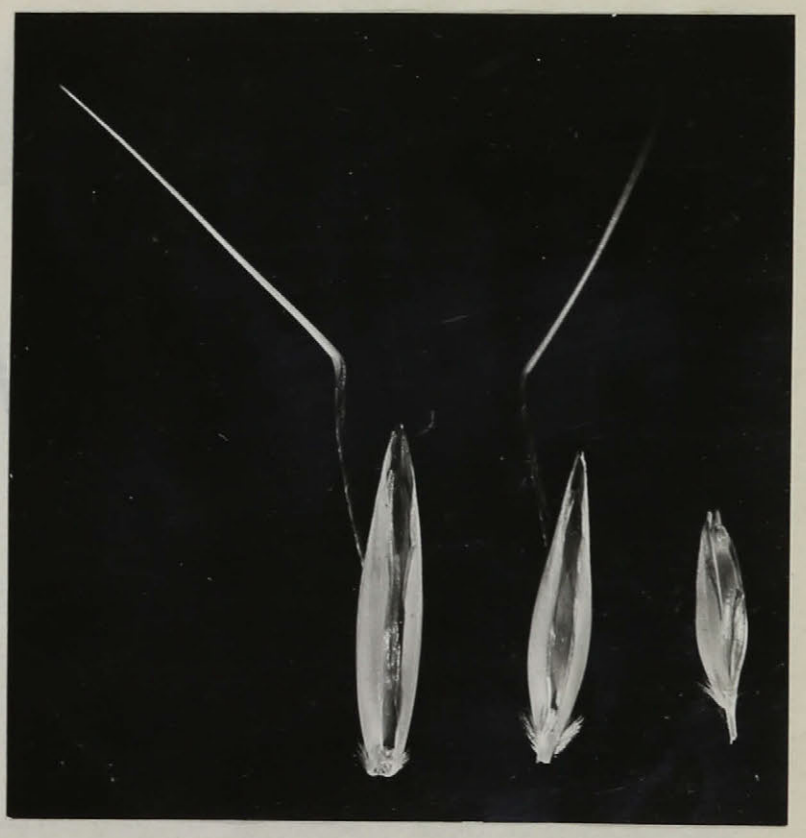

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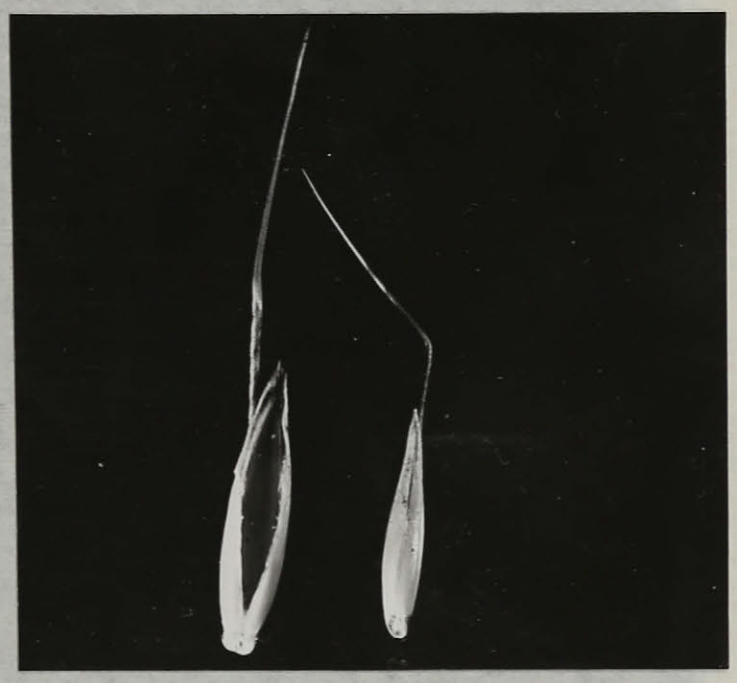
PLATE IV



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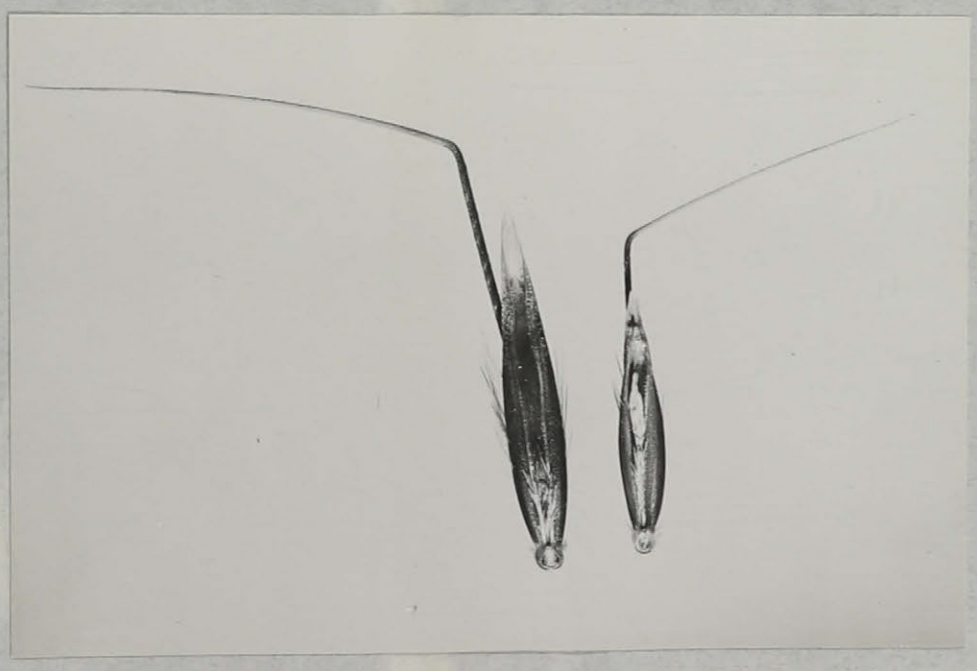

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PLATE V

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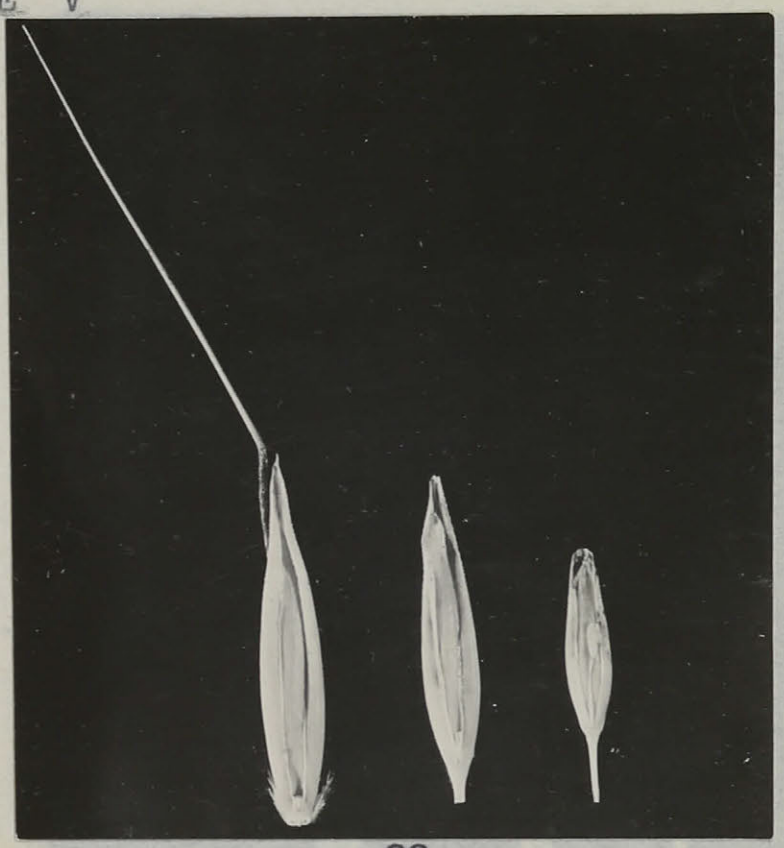

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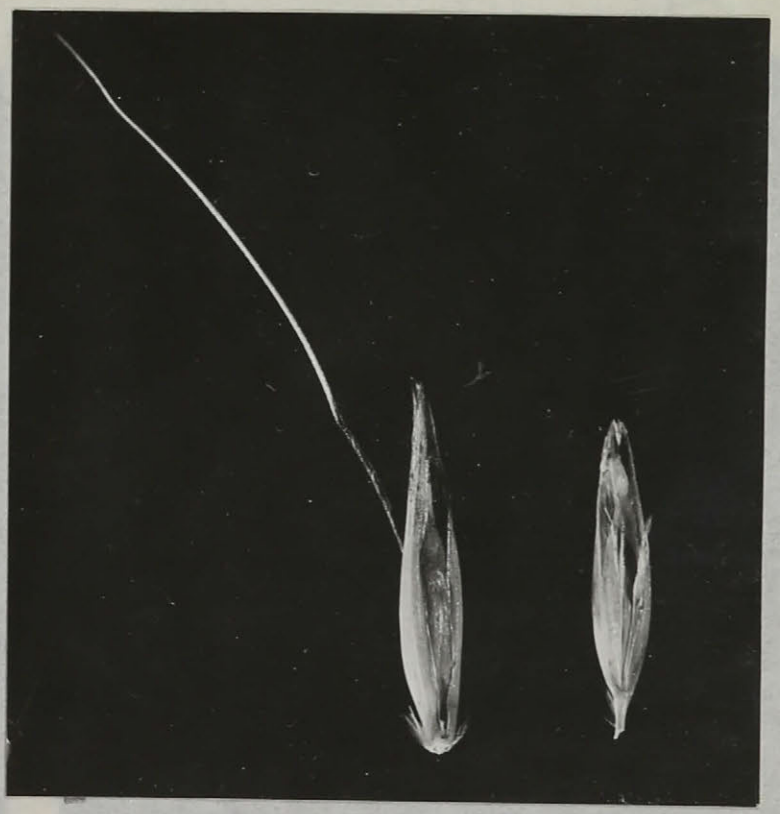

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PLATE VI

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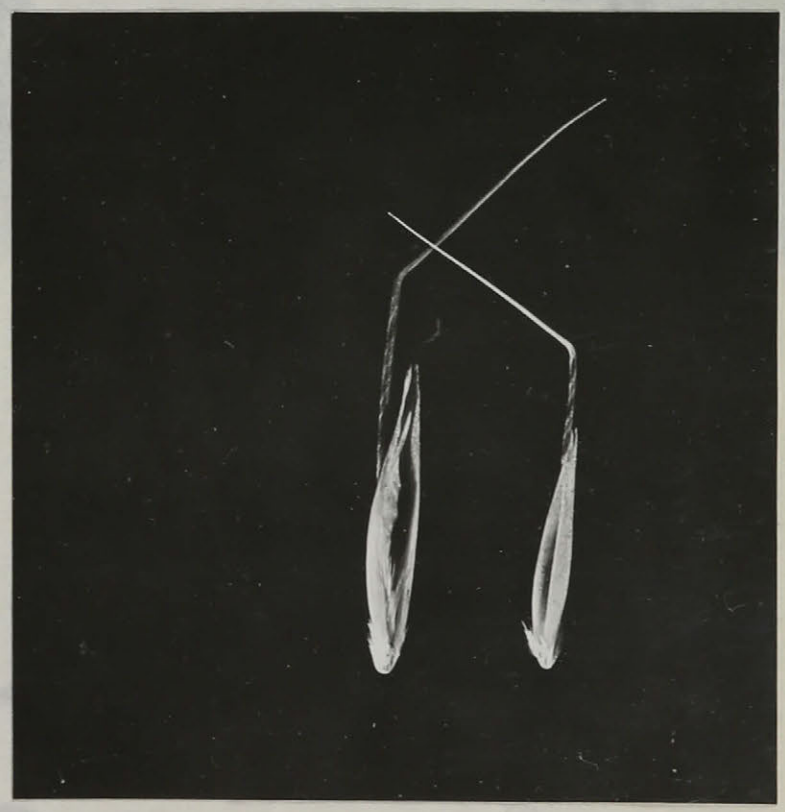

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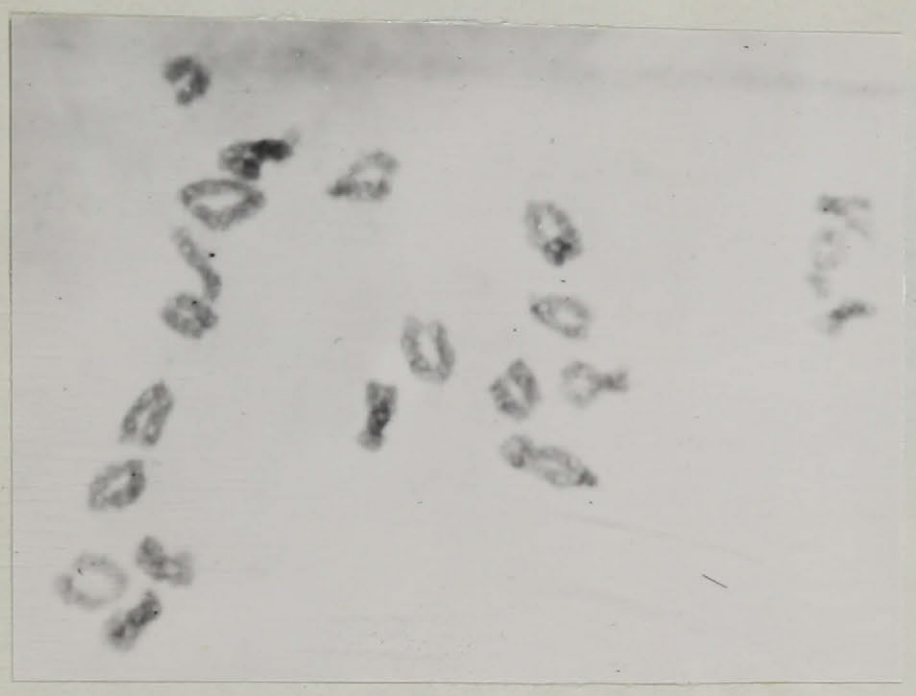
PLATE VII - Photomicrographs.



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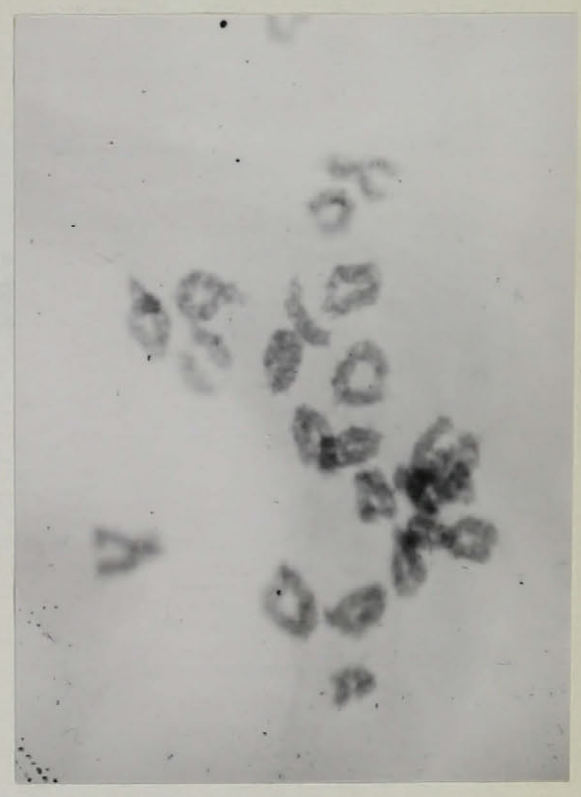

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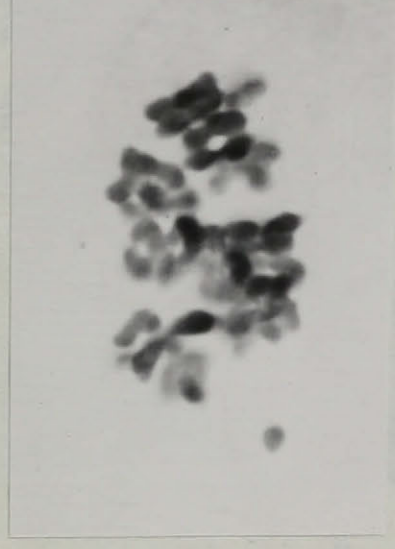

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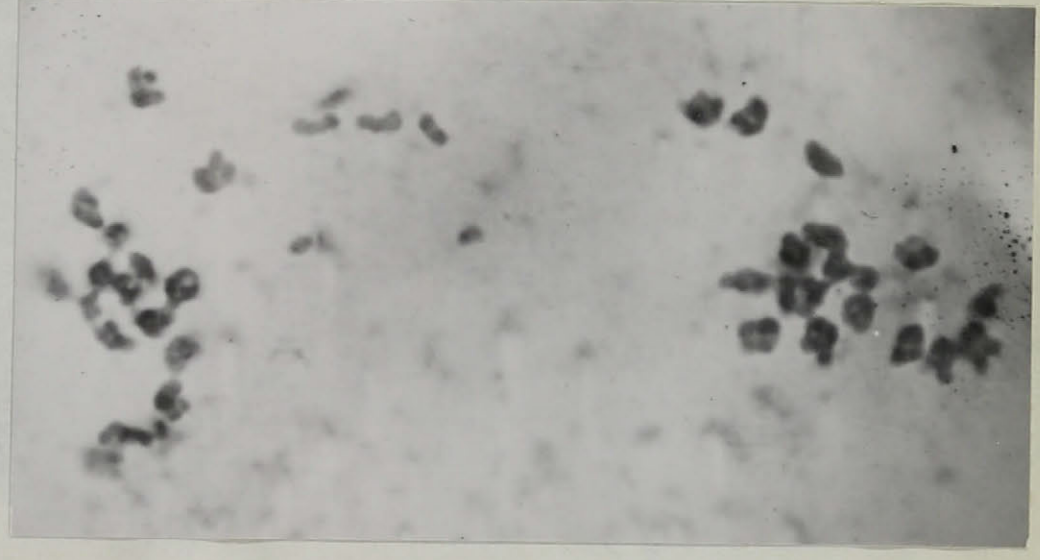

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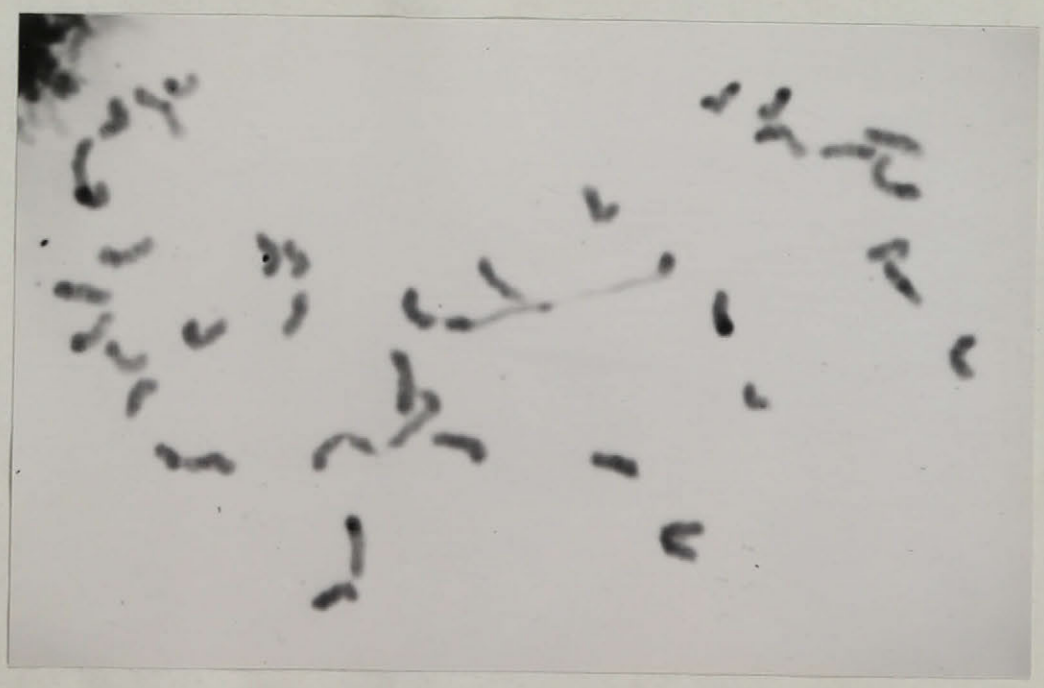

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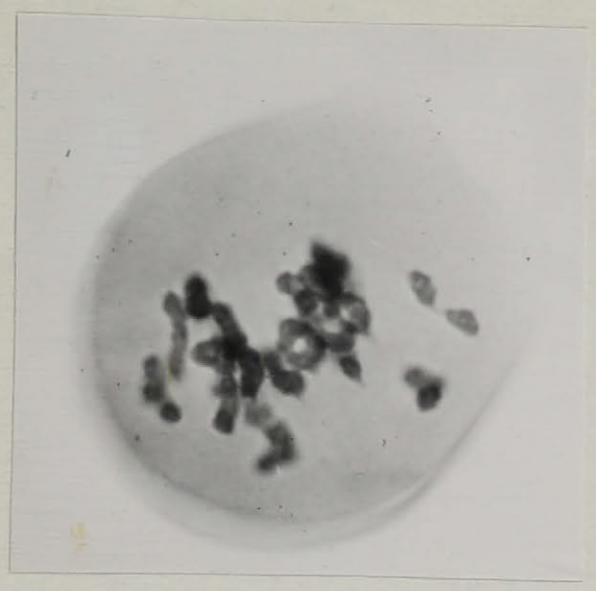
PLATE VIII - Photomicrographs.



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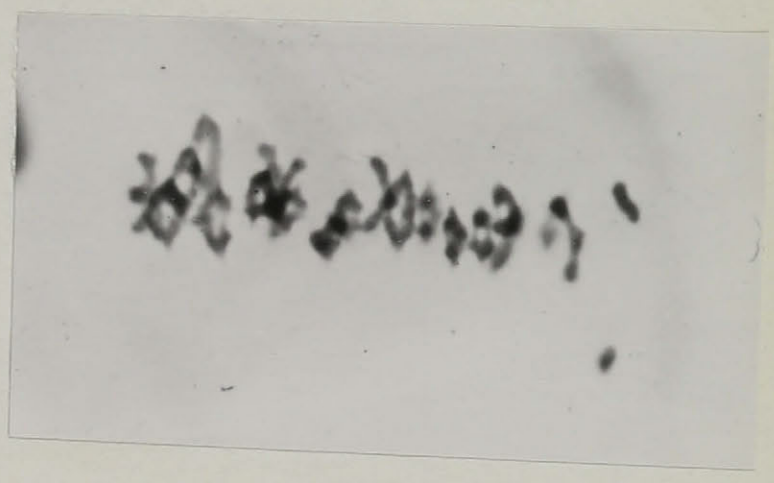

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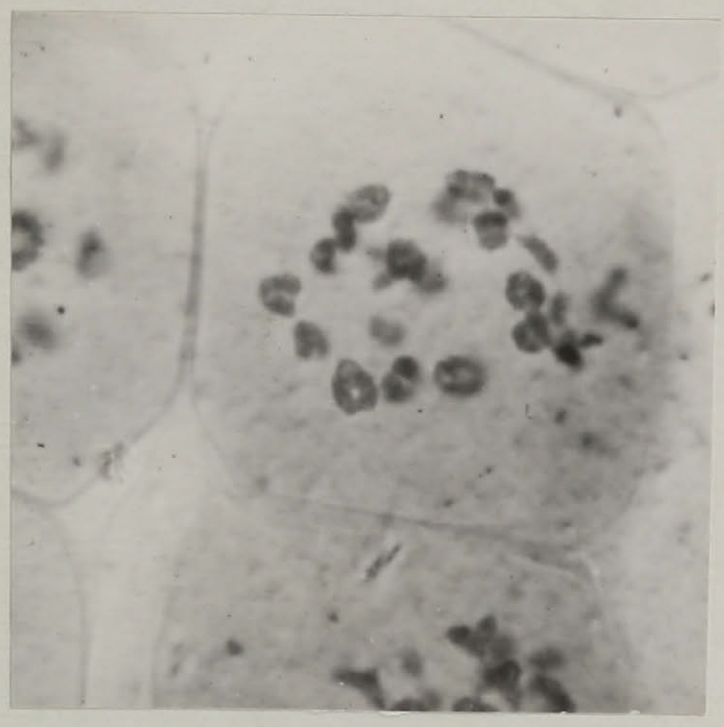

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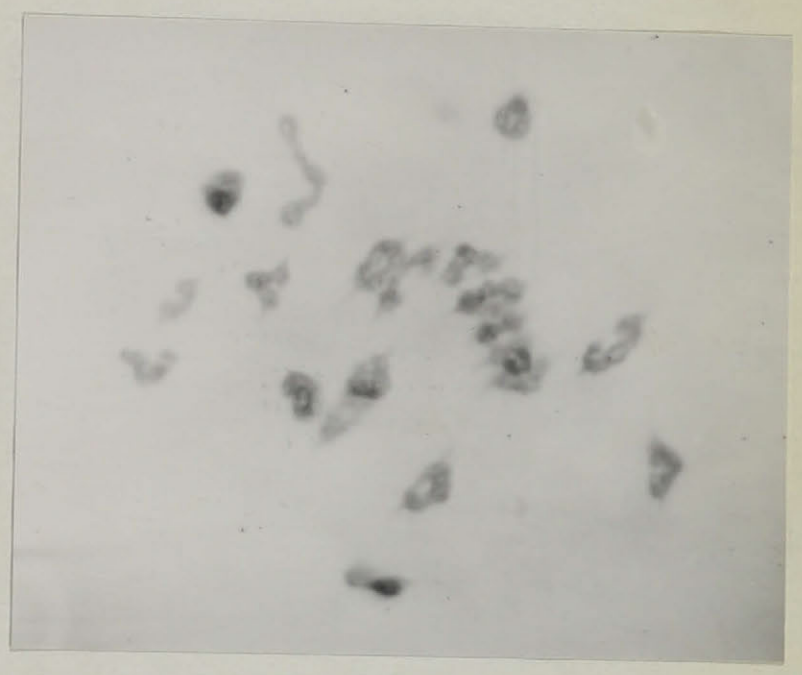

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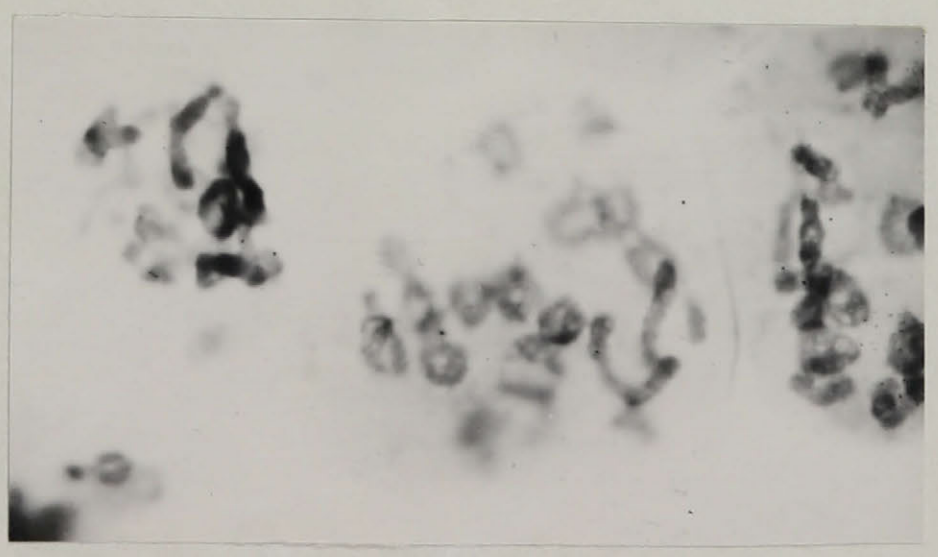

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