# THE ROLE OF MAJOR GENES IN THE EVOLUTION OF ECONOMIC CHARACTERS

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CONTENTS



## Introduction

The early Mendelians devoted much attention to the more obvious plant and animal characters, such as pigmentation, dwarfing and the like, and a wealth of information confirming Mendel's work was soon built up. The next stage—the application of genetics to economic ends—disclosed difficulties, and experience has shown that many economic characters do not give simple Mendelian ratios in hybridization. As a result of this, modern thought tends more and more to stress the importance of minor genes. This school of thought has been developed by Fisher (1930) and 'Student' (1933, 1934), and extended by Mather (1943*a*, *b*) with his polygene concept. The polygene school holds that quantitative characters are normally built up by the combined effect of a number of minor genes brought together in response to selection pressure. A second school, led by the Russian academician Lysenko, maintains that the whole Mendelian outlook is fallacious. Lysenko's (1946) theories, however, take so little account of established fact and scientific proof that for the purposes of this paper they may safely be disregarded. Studies of the response of commercial cottons to blackarm* attack (Knight & Clouston, 1939; Knight, 1944, 1946) have indicated that major genes are more important in the synthesis of resistant types than might have been supposed. From this a theory has been developed, and is set forth in this paper, of the significance of major genes in adaptive response.

## Definitions

The terms 'major' and 'minor' genes are used throughout this paper as a convenient shorthand—clearly each must merge into the other so that no hard and fast line can be drawn between them. Major gene is used in the sense of a gene which, as compared with its allele (or weakest allele), produces a large effect. A minor gene, on the other hand, when compared with its allele produces an insignificant effect. Obviously there must be all degrees of gene 'size' from these major genes to the minor genes—a normal curve of gene 'size'—but it is the genes which produce large differences that are of outstanding

* Blackarm is a rain-borne disease caused by *Bacterium* (*Xanthomonas*) *malvacearum*.

interest to the applied geneticist. It is the aim of this paper to examine the adaptive significance of major genes, but it must be borne in mind that major genes are not a distinct class but are only one extreme of a frequency curve of gene size.

'Preadaptation' is used to mean the existence of a character which renders an organism potentially able to make use of a changed environment or to extend its original environmental limits.

Two other terms require definition: modifier and character. 'Modifier' is used in the sense of a gene which alters the expression of a major gene but which has no effect on its allele. A 'modifier complex' is thus an assemblage of minor genes, all of which affect the expression of the same major gene. The term 'character' has been used throughout this paper to mean a readily recognizable phenotypic effect or difference. Thus the large phenotypic effect associated with a major gene as compared with its allele would be a 'character', whereas the minute difference produced by a single minor gene would not be so described, though the cumulative action of a group of such genes would be said to control a 'character'.

Gene 'size' throughout this paper refers to the magnitude of the effect of a gene.

## THE RELATIONSHIP BETWEEN GENE SIZE AND SELECTIVE VALUE

Consider the response of an organism to changing selective forces. It is a reasonable assumption that the variability present prior to the change in selective forces was effectively neutral. It by no means follows, however, that the gene differences present will all be of equal value under the new conditions. For instance, on the introduction of blackarm disease into a cotton-growing country, resistance genes previously of neutral effect become genes of very varying magnitudes. Areas where the crop already contains a proportion of plants carrying the strong resistance gene $\mathbf{B}_2$ will be 'sifted' by the selection pressure of the disease so that an increasing proportion of $\mathbf{B}_2$ plants will survive. This has, in fact, happened in many parts of India and Africa with the result that many of the Indian and African cottons carry a large proportion of $\mathbf{B}_2$ plants. In the areas climatically most suited to the spread of this disease the continued selection pressure of blackarm will give a premium to plants showing a greater resistance than $\mathbf{B}_2$ and so will be built up a modifier complex enhancing the strength of $\mathbf{B}_2$. Minor genes with additive effect *vis-à-vis* $\mathbf{B}_2$ will also be added until in the areas most subject to the disease something approaching immunity will be evolved.

At the other end of the scale will come areas in which the commercial cotton crop contains no plants carrying $\mathbf{B}_2$ or other strong resistance factors. The subjection of such an area to a series of yearly blackarm attacks may either eliminate the crop, as happened with the very susceptible Egyptian type crop in much of the U.S. cotton belt, or it will by its steady selection pressure favour plants carrying minor genes governing resistance. If a number of such genes exists in the crop there will be built up *slowly* a measure of resistance sufficient to control the disease under the conditions obtaining in that particular area. Minor gene resistance of this type is found in certain American Upland cottons—notably in Deltapine 14, Rowden B6 and to a lesser extent in some of the Stoneville types. Harland (1934) built up resistance of this type by constant selection in Sea Island, and although this resistance was not at first recognized by Knight & Clouston (1941) the presence of a weak resistance has since been confirmed by the writer.

Minor genes are not all of equal value—clearly there must be a gradation from the

minor type to the major type. Thus, in blackarm resistance, the genes $B_2$ and $B_3$ are both strong and each confers a high degree of resistance. The Asiatic gene $B_4$ is slightly weaker than either $B_2$ or $B_3$ and the American Upland gene $B_1$ is so weak that although its phenotype can be clearly recognized on some backgrounds it is lost on others (Knight, 1947). Other weak but identifiable resistance genes have been found in a *punctatum* cotton from Kufra oasis and in another *punctatum* of unknown origin. In addition, unidentifiable minor genes and modifiers also occur in blackarm resistance.

### On the occurrence of preadaptation

In this consideration of the selective effect of a new disease it has been assumed that various resistance genes were already in existence although the crop had not previously been subjected to the disease. Experience has shown that preadaptation of this kind is not uncommon.

Such preadaptation is particularly well illustrated in the resistance of cotton (*Gossypium*) to blackarm disease. *Gossypium* is, in origin, a xerophytic or semi-xerophytic genus and *Bact. malvacearum* is a rain-borne disease. If, therefore, resistance has developed in 'response' to the selective effect of blackarm working in the rain areas in which cultivated cottons are grown, one would expect the wild species of *Gossypium* to be susceptible. An examination of these wild species,* however, shows *G. anomalum* to be almost immune. *G. somalense*, *G. klotzschianum* and its var. *davidsonii*, *G. armourianum*, *G. raimondii* and *G. aridum* are all resistant, whilst *G. harknessii* contains both resistant and susceptible types. *G. stocksii*, *G. sturtii*, *G. thurberi* and *G. tomentosum* are all susceptible.

An ecological examination of the wild *Gossypiums* shows that resistance to blackarm has not been developed under a selection pressure due to the presence of the disease. *G. anomalum*, which is almost immune to the disease, is found on the southern fringes of the Sahara across the width of Africa. The range of *G. somalense* overlaps that of *G. anomalum*, but the species is, if anything, rather more xerophytic. It, also, is resistant to blackarm. The New World species *G. klotzschianum*, *G. klotzschianum* var. *davidsonii*, *G. armourianum*, *G. raimondii*, *G. aridum* and *G. harknessii* are all from arid areas where they would not be subject to blackarm attack. Furthermore, there would be no survival of disease debris under such conditions, and seed infection would be impossible because of the length of time these wild seeds take to germinate. Since, by the very nature of these eco-climatic conditions, the possibility of the presence of blackarm disease is excluded, it is logical to suppose that the advent of this disease in the genus *Gossypium* was subsequent to the establishment of cotton as a cultivated crop in more mesophytic environments.

Further examples of preadaptation in *Gossypium* can be cited. Thus marked resistance to the virus disease Leaf Curl is found in some, but by no means in all, Sea Island cottons; it is also found in certain American Upland cottons from India and in some of the diploid *Gossypiums*, notably in *G. thurberi*, *G. armourianum*, *G. aridum* and *G. raimondii* from the New World and *G. stocksii* from Sind. Since Leaf Curl disease has been reported only from the Sudan and Nigeria, the resistance occurring in types of non-African origin is clearly preadaptational. Moreover, marked resistance to the Egyptian and Pink bollworms (*Earias insulana* and *Platyedra gossypiella*) has been found in *Gossypium thurberi* and *G. armourianum*. Both these bollworms are of Old World origin and their introduction

* In this connexion it should be noted that most of these wild *Gossypiums* had come from single plants.

into the New World was, in terms of evolution, very recent, so that the resistance shown by these two New World *Gossypiums* was not evolved under selection pressure from these pests.

### A THEORY OF THE GENETIC NATURE OF PREADAPTATION

Where a character has been built up under selection, one of the following genetic situations will have arisen:

(1) A single gene, giving full expression to the character, established in the population.

(2) One or more major genes of lesser size established and intensified up to the level of full expression by the addition of cumulative minor genes and modifiers.

(3) An accumulation of minor genes all of small individual effect.

Obviously situation (1) will be established more quickly than number (2), provided a gene of the necessary magnitude exists. Similarly, situation (2) will be established more easily and quickly than (3) if any suitable major genes are present in the population. It follows that characters that are fully developed at their first appearance are most unlikely to depend on genetic situation (3) and may be expected in most cases to be governed by situation (1). On the other hand, adaptations developed slowly over a considerable period in response to selection pressure will, in general, be governed by a number of minor genes. In fact, the rate at which a character is established under selection may be used as an index of the probable size of the genes involved. *Preadaptational characters involving major differences will thus be typically controlled by one or a few large genes*, whereas adaptations which arise in 'response' to an existing selection pressure may be controlled either by major or minor genes or both, according to the size of the genes available.

Fisher (1930) considers that the range of selective intensity in which a factor is effectively neutral is very small. He concluded that the genes contributing to the genetic variance of a species must therefore be of small individual effect. Fisher was considering the situation in which a species is steadily modified by slow adaptive response to a slowly changing environment. Under man's selection, however, changes in selection pressure are often sudden and drastic and the conditions postulated by Fisher do not, therefore, hold. Such drastic environmental changes may greatly alter the magnitude of gene effects. Thus, to return to the blackarm example, genes conferring resistance are, so far as is known, of no effect in the absence of the disease and therefore fulfil Fisher's requirement for genes which will contribute to the variability of the species. As has been shown above, however, these genes, under the impact of blackarm, become genes of very varying and often very considerable effect.

Diseases tend to be of greater importance in the closely planted monocultures of modern crop production than in the sparser conditions more typical of wild growth. The advent of a new disease in a cultivated crop represents a sudden environmental change of the type under discussion and one might, therefore, expect to find preadaptation and, consequently, major gene control predominating in the genetics of disease resistance. A survey of recent plant-breeding literature shows the facts to be in accordance with this expectation, as is demonstrated by the list (in Appendix 1) of thirty-three crop plants in which major gene resistance to eighty-four pests and diseases has been demonstrated.

In view of this importance of major genes in the control of disease resistance, the writer made a survey of recent literature to ascertain whether major genes are of equal

importance in the control of other economic characters. The results of this survey, tabulated in Appendix 2, show that in thirty-eight plants some 160 economic characters have so far been found to be wholly or partly under major gene control. These economic characters can be divided into four classes according to the evolutionary road by which major gene control became established.

(1) Those characters valued by man which have no selective value, either positive or negative, in the wild state. Such characters are preadaptational and therefore their control by one or a few major genes is not unexpected.

(2) Those characters which would have negative selection value in the wild species but which are valued by man. The survival of major genes governing characters of this type can reasonably be attributed to man having noticed the occasional rare mutation and propagated it for his own ends.

(3) Certain characters which by their very nature must be operated by a switch gene mechanism. These are the characters in which no intermediate stage between presence and absence is possible so that a slow building up of minor genes is out of the question, although they can be added after the main gene has brought the trait into being.

(4) Characters in which all degrees of expression are of value in appropriate environments. Such characters are usually manifested in the wild state as a cline developed in response to a continuously varying environmental factor, such as rainfall. The genetic control of such a cline would typically be multifactorial and it might be supposed that the individual genes would be small in effect. In actual fact, however, investigation of such characters in the genetically more uniform populations studied by the plant breeder has frequently shown that some of the genes involved are of large effect.

The application of this grouping to the characters listed in Appendix 2 shows that 49% of them belong to group (1), 28% to group (4), 19% to group (2), and only 4% to group (3). Although this grouping is arbitrary in certain cases, this fault is not likely materially to affect the totals in the four groups, and it is interesting to note the preponderance of group (1) traits—the preadaptational characters. It is also interesting to note the relative unimportance of group (3). It thus seems probable that preadaptation has played an important role not only in the evolution of disease resistance but also in the evolution of economic characters in general.

## Discussion

It is clear from the lists in Appendices 1 and 2 that major genes are more common in the control of economic characters than current genetic ideas would lead one to suppose. It is not suggested that control by a single powerful major gene unaccompanied by minor genes is likely to be a common genetic situation in many economic characters. Genes which by themselves give complete expression to a character appear to be rare; hence in economic characters, man will have added, by constant selection towards the maximum expression, a complex of minor genes. Thus, particularly where a character would have been of value in the wild state, a common form of genetic control is likely to be by one or two large genes accompanied by a number of minor genes and modifiers. This type of control will be particularly common where both alleles governing a character have been of value under different environmental conditions in the wild state.

Even if a character is basically controlled by major genes it may yet be too complex to be analysed genetically. This applies to compound 'characters' which are the result

of interaction of several characters, and hence it applies to many of the so-called 'quantitative' characters. Thus, for example, 'ginning out-turn'* in cotton is of great economic importance. One would not, however, expect it to show 'simple' inheritance because ginning out-turn is dependent on (1) density of population of lint hairs on the seed coat, (2) length of the lint hairs, (3) weight of the lint hairs per unit length (this, itself, is dependent on (*a*) degree of thickening of the hair and (*b*) hair diameter), and (4) size of seed. Furthermore, ginning out-turn has been of value to man ever since cotton became a commercial crop, so that over and above any larger differences due to major genes, one would expect man's selection to have built up a considerable complex of minor and modifying genes. Where two cotton types share several, but not all, of the characters which together determine ginning out-turn, then, on intercrossing them, one might expect to obtain some semblance of a Mendelian ratio. This probably explains the bimodal curves obtained by O'Kelly & Hull (1930) in some of their ginning out-turn inheritance studies, in which they found that 'where segregation was sufficiently clear-cut to give definite indications, it appeared that lint percentage was controlled by a single factor pair'. They also noted that 'in crosses between species and crosses between upland strains where the percentage differences were narrow, it was not possible to determine definitely the nature of the segregation'. This is in accord with the theory given above: in the interspecies crosses there would have been too many variables for major gene segregation to be evident, whereas in the upland crosses involving small percentage differences, the major genes were presumably the same in each parent and the differences would have been due to modifiers and/or minor genes.

By their very nature, many valuable crop characters are likely to have complex inheritance, but such 'blending' inheritance is not *necessarily* entirely polygenic. For breeding purposes an attempt should be made to reduce complex characters to their integral components in the hope that these can be examined separately. Even in the more complex characters there is still reasonable hope of finding key genes governing inheritance, provided the complexity can be reduced to its component interacting characters.

In the field of disease resistance, plant breeders might with benefit consider the implications of preadaptation. Often the local crop carries no major genes governing resistance to a particular disease. For example, major genes governing resistance to leaf curl virus do not appear to be present in Sakel cotton. Thus the NT2 variety of Sakel was originally fully susceptible to this virus disease. By constant selection, however, it was rendered more and more resistant until the later substrains approached immunity. S. H. Evelyn's (unpublished) work on breeding for leaf curl resistance in Domains Sakel cotton, again illustrates the synthesis of polygenic resistance to the disease. On the other hand, Sea Island and other imported types grown in the Sudan have differed markedly in their leaf curl susceptibility, some being highly susceptible and others almost immune. Since leaf curl is unknown outside Africa this resistance is presumably preadaptational. Such types might thus be expected to provide a useful source of major resistance genes for future work. Furthermore, a major gene governing a preadaptational character is unlikely to be accompanied by any minor gene or modifier complex so that it should not normally lose appreciably in strength when transferred to other varieties and species.

To the plant breeder engaged in hybridization, major genes are of greater value than minor genes because they are far more easily handled. Modern genetics tends to stress

* The percentage of cotton lint produced by a given weight of seed cotton.

the minor gene outlook, but any overstressing of this is likely to be to the detriment of applied genetics. Breeders who have followed the minor gene school of thought might be disinclined to examine their material in sufficient detail. Plant breeders are often content with the so-called 'practical' outlook, whereby their immediate problem is settled without any genetical information being acquired which would help others to deal with similar problems. If a more wholehearted attempt at genetical analysis were made as a routine practice, it is possible that many more cases of major gene inheritance of economic characters would be discovered. In intraspecific hybridization, two or three major genes each showing partial dominance, even if unaccompanied by minor genes and modifying factors, will often present all the appearances of polygenic inheritance. In interspecific hybridization, characters not infrequently show blending inheritance in $F_2$ yet segregate clearly in backcross generations after the genetic background of one or other parental species has been re-established in the hybrid stock. In the absence of detailed genetical analysis there is considerable danger of misinterpretation of such situations in terms of minor genes.

## Summary

It is considered that preadaptation is not uncommon in economic characters. Notable examples are the resistance of many wild xerophytic species of *Gossypium* to the rain-borne disease *Bact. malvacearum*, the resistance of several cottons of non-African origin to the virus disease leaf curl (a disease unknown outside the Sudan and Nigeria), and the marked resistance of certain New World species to the Egyptian and Pink bollworms.

It is argued that preadaptational characters involving major differences will typically be found to be controlled by one or a few large genes, whereas adaptations which arise in 'response' to an existing selection pressure may be controlled either by major or minor genes, or both, according to the strength of the genes available.

Preadaptation is thought to have played a major part in the evolution of disease and pest resistance and to this is attributed the predominance of major gene control in resistance. In this connexion a list is given of thirty-three crop plants in which major gene resistance to eighty-four pests and diseases has been demonstrated.

Major gene control is shown to be of considerable importance in a large number of economic characters other than disease resistance and a list is given of thirty-eight plants in which 160 economic characters have been found to be wholly or partly under major gene control. Approximately 50% of these characters are classified as preadaptational. It is suggested that major genes are more common in the control of economic characters than is generally supposed. Control by a single powerful major gene, unaccompanied by minor genes, is unlikely to be common. Single genes alone rarely give complete expression to a character, so that man's constant selection towards the maximum expression of each economic character will have added a complex of minor and modifying genes to the original strong gene.

By their very nature, many valuable crop characters are likely to have complex inheritance, but such 'blending' inheritance is not necessarily entirely polygenic. For breeding purposes an attempt should be made to reduce complex characters to their integral components to facilitate genetic analysis.

It is a pleasure to record my gratitude to Mr J. B. Hutchinson for his constructive criticisms and helpful discussions.

## APPENDIX 1

*Diseases and pests, resistance to which is controlled, at least in part, by major genes*

*Wheat*

*Puccinia graminis tritici* (Melchers & Parker, 1922)
*P. glumarum* (Biffen, 1905)
*P. triticina* (Waterhouse, 1930)
*P. rubigo-vera tritici* (Mains, 1934)
*Tilletia tritici* (Briggs, 1931)
*T. levis* (Gaines & Smith, 1933)
*Ustilago tritici* (Tingey, 1934)
*Urocystis tritici* (Shen, 1934)
*Erysiphe graminis* (Waterhouse, 1930)
Mosaic (yellow) (Miyake, 1938)
Mosaic (green) (Miyake, 1938)
*Chlorops pumilionis* (Jasnowski, 1938)
*Cephus cinctus* (Putnam, 1942)

*Barley*

*Puccinia graminis tritici* (Powers & Hines, 1933)
*P. simplex* (Straib, 1937)
*Ustilago nuda* (Nahmmacher, 1931)
*Erysiphe graminis hordei* (Dietz & Murphy, 1930*a*)
*Helminthosporium* (Kuckuck, 1930)

*Oats*

*Puccinia coronata avenae* (Dietz & Murphy, 1930*b*)
*P. graminis avenae* (Waterhouse, 1930)
*Ustilago avenae* (Reed, 1932)
*U. levis* (Reed, 1932)

*Rice*

*Helminthosporium oryzae* (Nagai & Hara, 1930)
*Piricularia oryzae* (Ramiah & Ramaswami, 1936)
*Cercospora oryzae* (Nakamori, 1936)

*Setaria*

*Ustilago crameri* (Yu, 1942)

*Sorghum*

*Sphacelotheca cruenta* (Marcy, 1937)
*S. sorghi* (Marcy, 1937)
*Pythium* root rot (Bowman, Martin, Melchers & Parker, 1937)
(?) Chinch Bug (Dahms, 1943)

*Maize*

*Puccinia sorghi* (Mains, 1931)
*Gibberella saubinetii* (where due to non-sugary gene) (Senn, 1932)
*Bact. stewartii* (Wellhausen, 1937)
(?) *Ustilago zeae* (Saboe & Hayes, 1941)
*Helminthosporium maydis* (Ullstrup, 1941)

*Cotton*

*Bact. malvacearum* (Knight & Clouston, 1939)
Jassid (Knight, unpublished)

*Potato*

*Synchytrium endobioticum* (Salaman & Lesley, 1923)
*Phytophthora infestans* (Black, 1945)
*Actinomyces scabies* (Krantz & Eide, 1941)
Virus A (Cockerham, 1943)
Virus B (Cockerham, 1943)
Virus C (Cockerham, 1943)
Virus X (Cockerham, 1943)

*Apple*

(?) Apple cedar rust (Moore, 1940)

*Capsicum*

Tobacco mosaic virus (Holmes, 1937)

*Sunflower*

*Homoesoma nebulella* (Plaček, 1936)
*Orobanche* (Plaček, 1936)

*Tobacco*

Tobacco-mosaic (Holmes, 1938)

*Gram* (Cicer)

*Fusarium* wilt (Ayyar & Iyer, 1936)

*Pea* (Pisum)

*Fusarium orthoceras* var. *pisi* (Wade, Zaumeyer & Harter, 1938)
*F. oxysporum f. pisi* race 2 (Walker, Delwiche & Hare, 1944)
Mildew (Harland, 1946)

*Cowpea* (Vigna sinense)

Powdery mildew (Dundas, 1939)

*Beans* (Vigna sesquipedalis)

Powdery mildew (Dundas, 1939)

*Beans* (Phaseolus)

*Colletotrichum Lindemuthianum* (Burkholder, 1918)
*Uromyces appendiculatus* (Wingard, 1933)
Mosaic (Pierce & Walker, 1933)
Alfalfa mosaic (Wade & Zaumeyer, 1940)
*Erysiphe polygoni* (Dundas, 1939)
*Heterodera marioni* (Barrons, 1940)

*Soya bean*

*Empoasca fabae* (Johnson & Hollowell, 1935)

*Tomato*

*Cladosporium fulvum* (Guba, 1936)
*Fusarium* wilt (Bohn & Tucker, 1940)
*Septoria lycopersici* (Andrus & Reynard, 1945)
*Alternaria solani* (Reynard & Andrus, 1945)

*Ground nuts*

Leaf-spot fungus (1) (Higgins, 1938)
Leaf-spot fungus (2) (Higgins, 1938)

*Onion*

*Colletotrichum circinans* (Rieman, 1930, 1931)

*Cabbage*

*Fusarium conglutinans* (Walker, 1930)

*Brussels sprouts*

*Fusarium* (Blank & Walker, 1933)

*Kohlrabi*

*Fusarium* (Blank & Walker, 1933)

*Flax*

*Melampsora lini* (Henry, 1930)
*Colletotrichum lini* (Houston & Stanford, 1945)

*Beet*

Curly-top (Owen & Abegg, 1938)

*Cucumber*

*Cladosporium cucumerinum* (Bailey, 1939)
Mosaic (Shifriss, Myers & Chupp, 1942)

*Lettuce*

*Brenia lactucae* (Jagger & Whitaker, 1940)
*Erysiphe cichoracearum* (Whitaker & Pryor, 1941)
Brown Blight (Jagger, 1940)

*Water melon*

*Colletotrichum lagenarium* (Poole, 1944)

*Melon*

*Erysiphe cichoracearum* (Jagger, Whitaker & Porter, 1938)

*Cajanus*

(?) Wilt (Shaw, 1936)

*Grape*

*Phylloxera vitifoliae* (Riehm, 1940)
(?) *Peronospora viticola* (Husfeld, 1930)

## APPENDIX 2

*Economic characters which are controlled, at least in part, by major genes*

| Group* | Character |
|---|---|
| | *Wheat* |
| 4 | Awn length (see Sansome, 1939) |
| 2 | Awns, absence of (see Sansome, 1939) |
| 1 | Ear density and shape (see Sansome, 1939) |
| 1 | Grain colour (see Sansome, 1939) |
| 2 | Non-shattering (Miczynski, 1930) |
| 1 | Brittleness (Raum, 1931) |
| 1 | Waxy endosperm (Watkins & Cory, 1931) |
| 4 | Earliness (Crescini, 1933) |
| 3 | Spring versus winter habit (see Sansome, 1939) |
| 4 | Grain weight (Jasnowski, 1935) |
| 4 | Grains per spikelet (Jasnowski, 1935) |
| 4 | Spikelets per ear (Jasnowski, 1935) |
| 1 | Absence of sprouting in ear (Åkerman, 1936) |
| 1 | (?) Gluten quality (Worzella, 1934) |
| 1 | Hollow versus solid stem (Platt, Darroch & Kemp, 1941) |

* See p. 374.

| Group | Character |
|---|---|
| | *Barley* |
| 4 | Rows per ear (Huber, 1931) |
| 1 | Hulled versus naked (Huber, 1931) |
| 1 | Grain colour (see Sansome, 1939) |
| 2 | Absence of dormancy (Moormann, 1942) |
| 4 | Earliness (Barbacki, 1930) |
| 1 | Starchy versus glutinous (Kasiwada, 1930) |
| 4 | Plant height (Swenson & Wells, 1944) |
| 4 | Rachis internode number (Swenson & Wells, 1944) |
| 2 | Non-barbed awns (Huber, 1931) |
| 1 | Ear density (see Sansome, 1939) |
| 1 | Ear length (Huber, 1931) |
| 1 | Ear shape (Huber, 1931) |
| 1 | Branched ears (Huber, 1931) |
| 3 | Spring versus winter habit (see Sansome, 1939) |
| 2 | Brittle awns (Tavčar, 1939) |
| 2 | Non-brittle rachis (Johnson & Åberg, 1943) |
| 2 | Awnless (see Sansome, 1939) |
| | *Oats* |
| 2 | Awnless (Florell, 1931) |
| 1 | Hulled versus hull-less (Love & McRostie, 1919) |
| 2 | Absence of dormancy (Moormann, 1942) |
| 4 | Earliness (Shaw & Bose, 1933) |
| 1 | Grain colour (see Sansome, 1939) |
| | *Rice* |
| 2 | Awnless (Jones, 1933) |
| 4 | Awn length (Mitra & Ganguli, 1932) |
| 4 | Earliness (Jones, 1933) |
| 1 | Glume length (Jones, 1933) |
| 1 | Compact versus spreading habit (Ramiah, 1930) |
| 1 | Grain colour (Ramiah, 1930) |
| 1 | Glutinous versus starchy (Ramiah, Jobitharaj & Mudaliar, 1931) |
| 1 | Long versus short kernels (Ramiah *et al.* 1931) |
| 1 | Lax versus dense panicle (Ramiah *et al.* 1931) |
| 1 | Brittleness (Jones, 1933) |
| 2 | Non-shattering (Jones, 1933) |
| 4 | Erect, or prostrate tillers (Ting, 1933) |
| 4 | Early short type (Miyazawa, 1934) |
| 1 | Lodging versus non-lodging (Ramiah & Dharmalingam, 1934) |
| 1 | Perfume (Kadam & Patankar, 1938) |
| 4 | Floating habit (Ramiah & Ramaswami, 1941) |
| | *Sorghum* |
| 4 | Normal versus dwarf (early) (Sieglinger, 1932) |
| 4 | Tall versus normal (Karper, 1932) |
| 1 | Pearly versus chalky grain (Ayyangar, Vijiaraghavan, Ayyar & Rao, 193 |
| 1 | Waxy endosperm (Karper, 1933) |
| 1 | Awns versus awnless (Sieglinger, Swanson & Martin, 1934). |
| 1 | Pithy versus juicy stalks (Ayyangar, Ayyar, Rao & Nambiar, 1936) |
| 1 | Insipid versus sweet stalks (Ayyangar *et al.* 1936) |
| 4 | Short early versus tall late (Ayyangar, Ayyar & Nambiar, 1937) |
| 1 | Twin seeded versus single seeded spikelets (Stephens & Quinby, 1938) |

| Group | Character |
|---|---|
| 2 | Cleistogamy (Ayyangar & Ponnaiya, 1939*b*) |
| 1 | (?) Absence of cyanogenetic glucosides (Franzke, Puhr & Hume, 1939) |
| 4 | Time of floral initiation and maturity date (Quinby & Karper, 1945) |
| | *Sudan grass* (Sorghum Sudanense) |
| 4 | Tillers versus tillerless (Ayyangar & Ponnaiya, 1939*a*) |
| 4 | Tillers all flowering together versus un-uniform flowering (Ayyangar & Ponnaiya, 1939*a*) |
| 4 | Long versus short awn (Ayyangar & Ponnaiya, 1939*a*) |
| | *Maize* |
| 1 | Endosperm colour (see Hayes & Immer, 1942) |
| 1 | Aleurone colour (see Hayes & Immer, 1942) |
| 1 | Endosperm nature (see Hayes & Immer, 1942) |
| 1 | Pericarp and cob colour (see Hayes & Immer, 1942) |
| 1 | Vitamin A content (Hauge & Trost, 1930) |
| 1 | (?) Nicotinic acid (Mather & Barton-Wright, 1946) |
| 1 | Rows per cob (Tavčar, 1935) |
| | *Rye* |
| 1 | Brittleness (Lada, 1933) |
| | *Pennisetum* |
| 4 | Bristly versus non-bristly (Ayyangar & Hariharan, 1936) |
| 1 | Sugary versus starchy grains (Patel, 1941) |
| | *Setaria* |
| 4 | Bristle length (Ayyangar, Narayanan & Rao, 1933) |
| 1 | Dense versus lax (Ayyangar & Sarma, 1933) |
| | *Panicum* |
| 1 | Grain colour (Ayyangar & Rao, 1938) |
| | *Cotton* |
| 1 | Long versus short sympodia (Kearney, 1930) |
| 1 | Normal versus 'cluster' (Thadani, 1923) |
| 4 | Degree of boll dehiscence (Abraham, 1934) |
| 1 | Lint colour (Harland, 1939) |
| 1 | Lint percentage (O'Kelly & Hull, 1930) |
| 1 | Presence or absence of lint (Afzal & Hutchinson, 1933) |
| 1 | 'Expansive' lint (Hutchinson, Silow & Stephens, 1947) |
| 4 | 5-loc boll (Anonymous, 1944–5) |
| | *Capsicum* |
| 2 | Absence of pungency (Deshpande, 1935) |
| 4 | (?) Number of locules (Miller & Fineman, 1938) |
| | *Tobacco* |
| 1 | Nicotine content (Hackbarth & Sengbusch, 1935) |
| 4 | Incompatibility and fertility (East, 1932) |
| | *Blackberry* |
| 2 | Thornlessness (Stene & Odland, 1938) |
| | *Sweet clover* (Melilotus) |
| 1 | Coumarin content (Stevenson & White, 1940) |
| | *White clover* (Trifolium) |
| 1 | Absence of cyanogenetic glucoside (Corkill, 1942) |

| Group | Character |
|---|---|
| | *Raspberry* |
| 1 | Fruit colour (Crane & Lawrence, 1931) |
| | *Coconut* |
| 1 | Makapuno versus normal (Torres, 1937) |
| | *Peach* |
| 1 | Melting flesh (Bailey & French, 1932) |
| 1 | Clingstone versus freestone (Bailey & French, 1932) |
| | *Grape* |
| 1 | Early branching (Stummer & Frimmel, 1932) |
| 1 | Colour of juice (Branas, Bernon & Levadoux, 1938) |
| | *Pisum* |
| 4 | Height (Mendel) |
| 2 | Thickness of pod-wall (Nilsson, 1932) |
| | *Beans* (Vicia) |
| 4 | Number of seeds per pod (Sirks, 1931) |
| 4 | Internode number (Sirks, 1931) |
| 4 | Stem length (Sirks, 1931) |
| | *Beans* (Phaseolus) |
| 2 | 'Stringless' pod (Prakken, 1934) |
| 4 | Climbing versus bush habit (Schreiber, 1932) |
| 2 | Absence of parchment in pod wall (Prakken, 1934) |
| | *Soya bean* |
| 1 | Seed coat colour (Woodworth, 1932) |
| 1 | Pubescence (Woodworth, 1932) |
| 4 | Lateness associated with tall habit (Woodworth, 1932) |
| 4 | Seeds per pod (Takahashi, 1934) |
| | *Tomato* |
| 4 | Tall versus dwarf (MacArthur, 1931) |
| 1 | Smooth versus peach skin (MacArthur, 1931) |
| 1 | Fruit colour (MacArthur, 1931) |
| 4 | Internode length (MacArthur, 1931) |
| 1 | Self-pruning habit (MacArthur, 1931) |
| 1 | Smooth versus fasciated fruit (MacArthur, 1931) |
| 4 | Uniform ripening (Anonymous, 1942) |
| 2 | Determinate versus indeterminate habit (Currence, 1932) |
| 4 | Number of locules (Yeager, 1937) |
| 1 | Fruit shape (Yeager, 1937) |
| | *Ground nuts* |
| 4 | Late versus early (Patel, John & Seshadri, 1936) |
| 1 | Long versus short seed (Hull, 1937) |
| 4 | Growth habit (Valencia—Spanish-Runner) (Hull, 1937) |
| | *Carrot* |
| 1 | Root shape (Frimmel & Lauche, 1938) |
| | *Sesamum* |
| 4 | Branching (Nohara, 1933) |
| 4 | Extra pods in leaf axil (Langham, 1945) |
| 1 | Calcium oxalate in seed (Nohara, 1933) |

| Group | Character |
|---|---|
| | *Beet* |
| 3 | Annual versus biennial (Munerati, 1931) |
| 2 | Self-fertility (Owen, 1938) |
| | *Strawberry* |
| 1 | Red versus white fruit (Fedorova, 1935) |
| 2 | Ever-bearing versus normal (Fedorova, 1935) |
| | *Lettuce* |
| 1 | Leaf lobing (Durst, 1930) |
| 2 | Non-prickly midribs (Durst, 1930) |
| 3 | Photoperiodism (Bremer & Grana, 1935) |
| 3 | Heading versus rosetting (Bremer & Grana, 1935) |
| | *Cucumber* (Cucumis sativus) |
| 2 | Fine spines versus coarse (Poole, 1944) |
| 2 | Few versus numerous spines (Poole, 1944) |
| 2 | Absence of spines (Poole, 1944) |
| 2 | Determinate versus indeterminate growth (Poole, 1944) |
| 1 | Fruit colour (Poole, 1944) |
| 4 | Tall versus short (Poole, 1944) |
| 2 | Tender skin (Poole, 1944) |
| 1 | Smooth versus rough fruit (Poole, 1944) |
| | *Cucurbita pepo* |
| 1 | Fruit shape (Sinnott & Hammond, 1930) |
| | *Watermelon* (Citrullus vulgaris) |
| 1 | Smooth versus furrowed fruit (Poole, 1944) |
| 4 | Seed weight (Weetman, 1937) |
| 1 | Elongate versus spherical fruit (Weetman, 1937) |
| 1 | Flesh colour (Porter, 1937) |
| 2 | Non-explosive rind (Poole, 1944) |
| 2 | Tender rind (Porter, 1937) |
| | *Celery* |
| 1 | Pithy versus non-pithy (Jones, 1932; Emsweller, 1932) |
| | *Lupins* |
| 2 | Alkaloid-free (Hackbarth & Sengbusch, 1934) |
| 1 | Soft versus hard skin (Sengbusch, 1938) |
| 2 | Non-splitting pods (Hackbarth, 1938) |
| 1 | White 'grain' (Hackbarth, 1938) |
| | *Yeast* |
| 3 | Ability to ferment sugar (Spiegelmann, Lindegren & Lindegren, 1945) |

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