CROPS RESEARCH

ARS 34-85
December 1966

A STANDARDIZED SYSTEM OF NOMENCLATURE FOR GENES GOVERNING CHARACTERS OF OATS

Agricultural Research Service
U.S. DEPARTMENT OF AGRICULTURE

CONTENTS

Prepared by
Crops Research Division
Agricultural Research Service

A STANDARDIZED SYSTEM OF NOMENCLATURE FOR GENES GOVERNING CHARACTERS OF OATS[1]

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INTRODUCTION

Preliminary investigation showed that genes governing characters of oats (used here to include all diploid, tetraploid, and hexaploid species of Avena) had been named in haphazard fashion. There have been no rules and only few suggestions to guide investigators in this field; and, more important, there has not been a centralized organization for collecting and clearing information. The symbols that have been used often bear little relationship to the characters concerned, and completely different symbols have sometimes been assigned to genes governing the expression of the same character. Even more confusing, the same symbols have occasionally been assigned to genes for entirely different characters. The study of the genetics of oats is now proceeding at an increasingly rapid pace and, without some kind of system, can be expected to lead to ever greater confusion. With these thoughts in mind, the chairman of the National Oat Conference, assigned the authors as a committee the task of developing a standardized system of nomenclature for genes governing characters of oats.

Standardized systems of genetic nomenclature have been established for corn (Emerson, Beadle, and Fraser, 1935), barley (Robertson, Wiebe, and Immer, 1941), and wheat (Ausemus, Harrington, Reitz, and Worzella, 1946). These were studied in detail by this committee. The views of numerous individuals were obtained through correspondence, and several unpublished reports of committees on gene nomenclature in other crop species were also consulted.

RULES FOR SYMBOLIZATION OF GENES IN OATS

The report of a committee (Tanaka et al. 1957) appointed by the Permanent International Committee for Genetics Congresses was particularly applicable. This report established a set of rules to be used as a guide by persons in all branches of biology interested in genetic nomenclature. These rules are actually rather general and, in some places, suggest more than one alternative. The rules established for genes in oats, given below, were adapted from the international rules, the alternatives and modifications being based on the specific problems and needs involved in the study of oat genetics:

1. Symbols of genes, derived from the English name of the character involved or from the Latin name of the pathogen in the case of a disease reaction, will be written in Roman letters. The symbol for a dominant gene will begin with a capital letter, and the symbol for a recessive gene with a small letter. Each symbol will be short, suggestive, differ from all other symbols used for oat genes, and contain not more than one capital letter.

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[1] Cooperative investigation between the Agricultural Research Service, U.S. Department of Agriculture, and the Iowa Agriculture and Home Economics Experiment Station; Journal Paper No. J-5425, Project No. 1176, of the Iowa Station.

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2. Two or more genes governing expression of the same character or otherwise conditioning phenotypically similar effects will be designated by a common basic symbol. This will be construed to mean that all genes governing reaction to a specific disease organism, without reference to races of the pathogen or varieties of the host, will have the same basic symbol. Within a basic symbol, nonallelic loci will be distinguished by an Arabic numeral on the same line after a hyphen following the basic symbol. The first locus to be discovered for a character will be understood to bear the number 1. The second locus for the character, if and when it is discovered, will be designated number 2. Members of allelic series will be distinguished by small letters following immediately after the locus number. The letters a and b will be understood to refer to the original allele-pair first discovered.

3. Inhibitors, suppressors, enhancers, lethals, and sterility genes will be designated, respectively, by the symbols I, Su, En, L, and S, or i, su, en *l*. and s if they are recessive, followed by a hyphen and the symbol of the gene affected.

4. No "wild type" will be recognized, and genes occurring in diploid, tetraploid, and hexaploid species will be included in a single system.

5. Linkage groups and corresponding chromosomes will be designated by Arabic numerals.

6. Genic formulas will be written as fractions, with the maternal alleles written as numerators. Each fraction will correspond to a single linkage group. Different linkage groups written in numerical sequence are separated by semicolons. Symbols of unlocated genes will be placed within parentheses at the end of the formula. In euploids and aneuploids, the gene symbols will be repeated as many times as there are homologous loci.

7. Symbols of extra-chromosomal factors will be enclosed within brackets and will precede the formula.

8. Chromosomal aberrations will be indicated by abbreviations: Df for deficiency, Dp for duplication, In for inversion, T for translocation, and Tp for transposition.

9. The zygotic number of chromosomes will be indicated by 2n, the gametic number by n, and the basic number by x.

GENES IN OATS AND SYMBOLS ASSIGNED TO THEM

A survey was made of the literature reporting genetic studies of oats; also, an attempt was made to determine which reports duplicated the discoveries of earlier investigators. This often necessitated making more or less arbitrary decisions. In general, for genes governing disease reaction, a gene reported by second and subsequent investigators in the same or obviously related varieties was assumed to be the same as the first gene reported in that variety for that character, unless there was some evidence that a different gene actually was involved. Genes conditioning the same basic character, but reported from unrelated varieties, were assumed to be neither identical nor allelic unless there was some reason for so thinking.

In the case of genes for characters other than disease reaction, genes governing expression of the same character were assumed to be identical if they were reported from the same species, unless there was some reason to think otherwise. Avena sterilis and A. byzantina were assumed to be a single species in this regard.

The genes, or loci, recognized are shown below, listed alphabetically according to their symbols. The reference given after the symbol is usually, but not always, the earliest reported discovery of the gene on the basis of the committee's investigations. References listed after the description report additional studies of the gene or report work with genes that are now tentatively regarded as the same gene. It is possible that future investigations or a more critical examination of existing data may show that some of these reports actually dealt with distinct genes. When this occurs, such genes will be assigned their own numbers.

A-1. Norton (1907). Gene conditioning awnedness in A. sativa. Dominance variable. Nilsson-Ehle (1914), Zinn and Surface (1917), Fraser (1919), Cotner (1929), Shaw and Bose (1933), De Villiers (1935), Torrie (1939), Ko et al. (1946), Coffman (1964).

A-2. Zade (1912). Gene conditioning awnedness in A. fatua. Dominance variable. Surface (1916), Love and Fraser (1917), Wilds (1917), Love and Craig (1918b), Von Tschermak (1929), Philp (1933), Aamodt et al. (1934).

A-3. Wiggans (1918). Gene conditioning awnedness in A. sterilis and A. byzantina. Dominance variable. Reed and Stanton (1925), Von Tschermak (1929), Coffman (1964).

A-4. Henning (1924). Gene conditioning strong awns on the primary kernel in Early New Market. Recessive. Designated "w".

A-5. Henning (1924). Gene conditioning awnlessness on the secondary kernel in Early New Market. Dominant. Designated "S".

A-6. Von Tschermak (1929). Second gene conditioning awnedness in A. fatua.

A-7. Von Tschermak (1929). Second gene conditioning awnedness in A. sterilis [and A. byzantina]. Coffman (1964).

A-8. Johnson (1933). Dominant gene for strong awns in Black Mesdag. Designated "S".

A-9. Johnson (1933). Gene for intermediate awns in Victory. Designated "T".

A-10. Tang (1938). Gene conditioning strong awns in California and Washington strains of A. sterilis macrocarpa. Designated "A1".

I-A-10. Tang (1938). Gene in Early Ripe that inhibits A-10 and A-11.

A-11. Tang (1938). Gene conditioning strong awns in Washington strain of A. sterilis macrocarpa. Designted "A2".

A-12. Coffman (1964). Second gene conditioning awnedness in A. sativa.

A-13. Coffman (1964). Third gene conditioning awnedness in A. sativa.

A-14. Coffman (1964). Third gene conditioning awnedness in A. byzantina [and A. sterilis].

ap. Tang (1938). Recessive gene conditioning awn pubescence in strains of A. sterilis macrocarpa.

B. Mackie (1928). Partly dominant gene for resistance to blast in Kanota.

Ba-1. Surface (1916). A generally partly dominant gene conditioning the "cultivated" type of basal articulation of the primary floret in crosses with species and varieties having "wild" type basal articulation. Wilds (1917), Wiggans (1918), Love and Craig (1918b), Fraser (1919), Henning (1924), Goulden (1926), Von Tschermak (1929), Ma (1933), Philp (1933), Shaw and Bose (1933), Aamodt et al. (1934), Middleton (1938), Tang (1938), Hayes et al. (1939), Torrie (1939), Ko et al. (1946), Kehr and Hayes (1950), Day (1963), Coffman (1964).

Ba-2. Von Tschermak (1929). Second gene conditioning "cultivated" type of basal articulation of the primary floret in crosses between A. sativa, and between A. fatua and A. sterilis.

Ba-3. Ko et al. (1946). Gene conditioning "cultivated" type of basal articulation of the primary floret in SD334, complementary with Ba-4.

Ba-4. Ko et al. (1946). Gene conditioning "cultivated" type of basal articulation of the primary floret in SD334, complementary with Ba-3.

Ba-5. Jones (1940). Dominant gene conditioning "wild" type basal articulation of the primary floret in diploid and tetraploid species of Avena. Designated "X".

Ba-6. Jones (1940). Second dominant gene conditioning "wild" type basal articulation of the primary floret in diploid and tetraploid species of Avena. Designated "Y".

Cda-1. Nishiyama (1934). Complementary gene conditioning, with Cda-2, chlorophyll deficiency-albino in diploid oats. Designated "G".

Cda-2. Nishiyama (1934). Complementary gene conditioning, with Cda-1, chlorophyll deficiency-albino in diploid oats. Designated "C".

cda-3. Smith (1938). Recessive gene for chlorophyll deficiency-albino found in progeny of Victoria.

Cdc-1. Åkerman and Fröier (1941). Gene conditioning chlorophyll deficiency-chlorina in Golden Rain. Designated "Chlor 1". Fröier (1946), Morey and Earhart (1952).

Cdc-2. Åkerman and Fröier (1941). Gene conditioning chlorophyll deficiency-chlorina in Golden Rain. Designated "Chlor 2". Froier (1946).

Cdc-3. Åkerman and Fröier (1941). Gene conditioning chlorophyll deficiency-chlorina in Golden Rain. Designated "Chlor 3". Froier (1946).

Cd*l*-1. Froier (1946). Gene conditioning chlorophyll deficiency-lutescens in Novahavre. Designated "Lp". Åkerman (1922).

Cd*l*-2. Froier (1946). Second gene conditioning chlorophyll deficiency-lutescens in Novahavre. Designated "Ln". Åkerman (1922).

Cd*l*-3. Froier (1946). Gene conditioning chlorophyll deficiency-lutescens in the variety Swedish. Designated "Ls". Åkerman (1922).

Cds-1. Coffman et al. (1925). Gene conditioning chlorophyll deficiency-stripe (yellow) in Burt.

Cds-2. Coffman et al. (1925). Second gene conditioning chlorphyll deficiency-stripe (yellow) in Burt.

Cds-3. Coffman et al. (1925). Third gene conditioning chlorophyll deficiency-stripe (yellow) in Burt.

Cdv. Froier (1948). Gene conditioning chlorophyll deficiency-albovirescens.

Dd. Griffiths and Holden (1957). Dominant gene carried by Gray Winter for resistance to the stem eelworm, Ditylenchus dipsaci Kühn.

ds-1. Dyck and Rajhathy (1965). Recessive gene conditioning desynapsis during meiosis in A. strigosa.

ds-2. Thomas and Rajhathy (1966). Recessive gene conditioning desynapsis during meiosis in tetraploid oats. Designated ds_2.

Dw-1. Stanton (1923). Gene for dwarfness in progeny of Winter Turf x Sixty Day. Designated "D". Cotner (1929), Florell (1931), Litzenberger (1949b).

dw-2. Lewis (1926). Recessive complementary gene conditioning, with dw-3, dwarfism in progeny of Albion. Designated "da".

dw-3. Lewis (1926). Recessive complementary gene conditioning with dw-2, dwarfism in progeny of Red Rustproof. Designated "dr".

Dw-4. Cotner (1929). Gene for dwarfness in progeny of crosses between hexaploid oats. Florell (1931), Litzenberger (1949b).

Eg-1. Jones and Griffiths (1952). Gene for resistance to Erysiphe graminis DC. in Cc4146.

Eg-2. Jones and Griffiths (1952). Gene for resistance to Erysiphe graminis DC. in A. strigosa.

Fd-1. Wiggans (1918). Gene for A. sterilis type of secondary floret disjunction in crosses with A. sativa and A. fatua. Henning (1924), Cotner (1929), Von Tschermak (1929), Florell (1931), Ma (1933), Torrie (1939), Hayes et al. (1939), Ko et al. (1946), Coffman (1964).

Fd-2. Florell (1931). Second gene for A. sterilis type of secondary floret disjunction in crosses with A. sativa and A. fatua. Hayes et al. (1939), Coffman (1964).

Fd-3. Ko et al. (1946). Complementary gene conditioning, with Fd-4, A. byzantina type of secondary floret disjunction.

Fd-4. Ko et al. (1946). Complementary gene conditioning, with Fd-3, A. byzantina type of secondary floret disjunction.

Gc-1. Wakabayashi (1921). Gene for dark glume color, dominant over red, in Black Tartarian. Gaines (1924).

Gc-2. Schafer (1923). Gene for red glume color, dominant over white, in Red Rustproof.

Gi-1. Zhegalov (1920). Dominant gene for giantism in A. orientalis.

gi-2. Zillinsky (1959). Recessive gene ("monster") for giantism in progeny of Clintland-Garry x Laurel-Klein 69B.

H. Norton (1907). Gene, with variable dominance, conditioning hull-lessness and multiflorous spikelet. Von Tschermak (1910), Gaines (1917), Zinn and Surface

(1917), Caporn (1918), Love and McRostie (1919), Reed (1925), Lebedeff (1930), Chou (1932).

Ha-1. Andersen (1961). Gene in Grise de Houdan for resistance to the cereal root eelworm, *Heterodera avenae* Woll.

Ha-2. Andersen (1961). Dominant gene in certain American oat varieties for resistance to the cereal root eelworm, *Heterodera avenae* Woll.

Hv. Murphy and Meehan (1946). Dominant gene in Victoria for susceptibility to Victoria blight, caused by *Helminthosporium victoriae* Meehan and Murphy. May be pleiotropic or closely linked to Pc-2. Litzenberger (1949a), Finkner (1953), Welsh et al. (1954).

Kp-1. Nilsson-Ehle (1908). Gene for long hairs (kernel pubescence) at base of kernel. Zinn and Surface (1917).

Kp-2. Nilsson-Ehle (1908). Gene for short hairs (kernel pubescence) at base of kernel. Zinn and Surface (1917).

Kp-3. Zade (1912). Gene for heavy kernel pubescence in *A. fatua*. Wilds (1917), Von Tschermak (1929), Jones (1930), Federova (1930), Philp (1933), Aamodt et al. (1934).

Kp-4. Wiggans (1918). Gene for kernel pubescence in Red Texas. Fraser (1919), Schafer (1923), Henning (1924), Cotner (1929), Shaw and Bose (1933), Ma (1933), Tang (1938), Middleton (1938), Torrie (1939), Hayes et al. (1939), Ko et al. (1946), Litzenberger (1949b), Kehr and Hayes (1950), Craigmiles (1952), Coffman (1964).

Kp-5. Wiggans (1918). Second gene for kernel pubescence in Red Texas. Shaw and Bose (1933), Tang (1938).

Kp-6. Henning (1924). Gene for dense kernel pubescence in *A. sterilis nigra*.

Kp-7. Federova (1930). Gene modifying kernel pubescence. Designated "m_1". Ma (1933).

Kp-8. Federova (1930). Second gene modifying kernel pubescence. Ma (1933).

Kp-9. Ko et al. (1946). Complementary gene conditioning, with Kp-10, kernel pubescence in cross between Bond and SD334.

Kp-10. Ko et al. (1946). Complementary gene conditioning, with Kp-9, kernel pubescence in cross between Bond and SD334.

L-1. Nilsson-Ehle (1909). Dominant gene for presence of ligule in open panicle oats. Designated "L_1". Love and Craig (1918a), Garber (1922), Meurman (1927), Odland (1928), Åkerman and Mühlow (1933).

L-2. Nilsson-Ehle (1909). Second dominant gene for presence of ligule in open panicle oats. Designated "L_2". Love and Craig (1918a), Garber (1922), Meurman (1927), Odland (1928), Åkerman and Mühlow (1933).

L-3. Nilsson-Ehle (1909). Third dominant gene for presence of ligule in open panicle oats. Designated "L_3". Meurman (1927), Åkerman and Mühlow (1933).

L-4. Nilsson-Ehle (1909). Fourth dominant gene for presence of ligule in open panicle oats. Designated "L_4".

Lc-1. Wilson (1904). Incompletely dominant gene for black or dark lemma color. Norton (1907), Wilson (1907), Nilsson-Ehle (1909), Surface (1916), Zinn and Surface (1917), Wilds (1917), Love and Craig (1918b), Caporn (1918), Gaines (1924), Quisenberry (1926), Meurman (1927), Garber and Quisenberry (1928), Hayes et al. (1928), Odland (1928), Federova (1930), Welsh (1931), Florell (1931), Robb (1932), Ru (1933), Johnson (1933), Philp (1933), Ma (1933), Aamodt et al. (1934), De Villiers (1935), Åkerman and Bader (1937), Tang (1938), Middleton (1938), Patel (1941), Åkerman (1948), Coffman (1964).

Lc-2. Nilsson-Ehle (1909). Gene for gray lemma color expressed only in the absence of black. Designated "Gr". Surface (1916), Wilds (1917), Love and Craig (1918b), Caporn (1918), Henning (1924), Meurman (1927), Federova (1930), Welsh (1931), Robb (1932), Johnson (1933), Philp (1933), Ma (1933), Aamodt et al. (1934), Coffman (1964).

Lc-3. Nilsson-Ehle (1909). Second gene for black lemma color. Designated "S_2". Robb (1932), Åkerman (1948), Coffman (1964).

Lc-4. Nilsson-Ehle (1909). Gene for yellow lemma color. Designated "G". Surface (1916), Wilds (1917), Love and Craig (1918b), Fraser (1919), Cotner (1929), Ma (1933), Torrie (1939), Coffman (1964).

Lc-5. Fraser (1919). Second gene for yellow lemma color. Designated "y".

Lc-6. Fraser (1919). Gene for red lemma color. Designated "R". Henning (1924), Cotner (1929), Torrie (1939), Ko et al. (1946), Coffman (1964).

Lc-7. Meurman (1927). Gene that intensifies gray lemma color in the presence of Lc-2. Designated "Z".

Lc-8. Cotner (1929). Second gene for red lemma color. Coffman (1964).

Lc-9. Welsh (1931). Dominant gene for white lemma color.

Lc-10. Welsh (1931). Second dominant gene for white lemma color.

Lc-11. Ko et al. (1946). Complementary gene conditioning, with Lc-12, white to yellowish lemma color.

Lc-12. Ko et al. (1946). Complementary gene conditioning, with Lc-11, white to yellowish lemma color.

Lf-1. Finkner et al. (1954). Dominant gene for lemma fluorescence under UV. Designated "F".

Lf-2. Finkner et al. (1954). Second dominant gene for lemma fluorescence under UV. Designated "L".

Lp-1. Bartlett (1916). Dominant gene for lemma pubescence in *A. fatua*. Surface (1916), Wilds (1917), Love and Craig (1918b), Federova (1930), Florell (1931), Ma (1933), Philp (1933), Aamodt et al. (1934), De Villiers (1935).

Lp-2. Bartlett (1916). Gene inhibiting expression of Lp-1. Love and Craig (1918b), Federova (1930), Ma (1933), De Villiers (1935).

Lp-3. Wilds (1917). Second dominant gene for lemma pubescence in *A. fatua*. Philp (1933).

Lp-4. Cotner (1929). Gene for lemma pubescence in *A. sterilis*. Tang (1938).

Lp-5. Cotner (1929). Second gene for lemma pubescence in *A. sterilis*. Tang (1938).

Lp-6. Tang (1938). Third gene for lemma pubescence in *A. sterilis*.

Lw. Nilsson-Ehle (1908). Gene for lemma waxiness. Meurman (1927).

Np. Florell (1931). Gene for nodal pubescence in *A. sterilis* and *A. byzantina*. Litzenberger (1949a), Craigmiles (1952).

Pc-1. Davies and Jones (1927). Dominant gene for resistance to crown rust, caused by *Puccinia coronata* Cda. var. *avenae* Fraser and Led., in Red Rustproof. Designated "S" by Dietz and Murphy (1930).

I-Pc-1. Dietz and Murphy (1930). Dominant gene inhibiting Pc-1. Designated "I".

Pc-2. Murphy et al. (1937). Partially dominant gene for resistance to *P. coronata* race 1 in Victoria. Is pleiotropic or closely linked with Hv. Waterhouse (1939), Weetman (1942), Cochran et al. (1945), Murphy and Meehan (1946), Litzenberger (1949b), Vallega (1951), Finkner (1953), Griffiths (1953), Finkner (1954), Welsh et al. (1954), Simons (1956), Craigmiles (1956), Chang (1959), Chang and Sadanaga (1964).

Pc-3. Hayes et al. (1939). Complementary gene conditioning, with Pc-4, resistance to *P. coronata* in Bond. Torrie (1939), Weetman (1942), Cochran et al. (1945), Ko et al. (1946), Litzenberger (1949b), Kehr and Hayes (1950), Griffiths (1953).

I-Pc-3. Cochran et al. (1945). Dominant gene in Richland-Fulghum that inhibits Pc-3. Designated "C".

Pc-4. Hayes et al. (1939). Complementary gene conditioning, with Pc-3, resistance to *P. coronata* in Bond. Torrie (1939), Weetman (1942), Cochran et al. (1945), Ko et al. (1946), Litzenberger (1949b), Kehr and Hayes (1950), Griffiths (1953).

I-Pc-4. Cochran et al. (1945). Dominant gene in Richland-Fulghum that inhibits Pc-4. Designated "D".

Pc-3c. Weetman (1942). Complementary gene conditioning, with Pc-4c, resistance to some isolates of P. coronata race 1 in Ukraine. Upadhyaya and Baker (1962b).

Pc-4c. Weetman (1942). Complementary gene conditioning, with Pc-3c, resistance to some isolates of P. coronata race 1 in Ukraine. Upadhyaya and Baker (1962b).

Pc-5. Litzenberger (1949a). Dominant gene for resistance to P. coronata races 1 and 45 in Landhafer. Kehr and Hayes (1950), Finkner (1954), Simons and Murphy (1954), Baker (1955), Craigmiles (1956), Chang (1959), Rivers (1959), Upadhyaya and Baker (1962b), Chang and Sadanaga (1964).

Pc-6. Litzenberger (1949b). Dominant gene for resistance to P. coronata races 1 and 45 in Santa Fe. Designated "S". Osler and Hayes (1953) designated it "S"; Finkner (1954) designated it "M_1"; Simons and Murphy (1954), Baker (1955), Finkner et al. (1955), Craigmiles (1956), and Chang (1959) designated it "M". Upadhyaya and Baker (1962b).

Pc-7. Osler and Hayes (1953). Complementary gene conditioning, with Pc-8, resistance to P. coronata races 45 and 57 in Santa Fe. Designated "D".

Pc-8. Osler and Hayes (1953). Complementary gene conditioning, with Pc-7, resistance to P. coronata races 45 and 57 in Santa Fe. Designated "D".

Pc-6c. Finkner (1954). Dominant gene for resistance to P. coronata race 57 in Ukraine. Designated "M" and is linked to Pc-9. Finkner et al. (1955), Sanderson (1960), Upadhyaya and Baker (1962b).

Pc-9. Finkner (1954). Second dominant gene for resistance to P. coronata race 57 in Ukraine. Designated "U" and is linked to Pc-6c. Finkner et al. (1955), Sanderson (1960), Upadhyaya and Baker (1962b).

Pc-10. Finkner (1954). Dominant gene for resistance to P. coronata race 57 in Klein 69B. Designated "K". Sanderson (1960).

I-Pc-10. Finkner (1954). Dominant gene in Clinton inhibiting Pc-10. Designated "I_k".

Pc-6d. Finkner (1954). Dominant gene for resistance to P. coronata race 57 in Trispernia and Anthony-Bond x Boone. Designated "M_2". Simons and Murphy (1954), Baker (1955), Craigmiles (1956), Upadhyaya and Baker (1962b).

Pc-2c. Finkner (1954). Gene for resistance to P. coronata race 57 in Anthony-Bond x Boone. Designated "V_1" and is not associated with susceptibility to Helminthosporium victoriae.

Pc-11. Welsh et al. (1954). Dominant gene for resistance to P. coronata races 1 and 45 in Victoria. Waterhouse (1939), Vallega (1951), Chang and Sadanaga (1964).

Pc-12. Welsh et al. (1954). Second dominant gene for susceptibility to P. coronata races 1 and 45 in Victoria.

Pc-9c. Simons and Murphy (1954). Gene for resistance to P. coronata races 45 and 101 in a derivative of Santa Fe. It is linked to Pc-6. Finkner et al. (1955) designated it "U", and Chang (1959) used the same symbol.

Pc-13. Finkner et al. (1955). Dominant gene for resistance to P. coronata race 109 in Clinton. Designated "A". Chang (1959) designated it "b^R".

I-Pc-13. Chang (1959). Dominant gene inhibiting Pc-13, in Gopher and other lines.

Pc-14. Simons (1956). Dominant gene for resistance to P. coronata race 202 in Ascencao. Designated "E". Chang (1959).

I-Pc-14. Chang (1959). Dominant gene inhibiting Pc-14, in Gopher and other lines. Designated "I_E".

Pc-15. Murphy et al. (1958). Dominant gene for resistance to P. coronata races 202 and 258 in the diploid Saia. Simons et al. (1959), Marshall and Myers (1961), Dyck and Zillinsky (1962), Dyck and Zillinsky (1963).

Pc-16. Murphy et al. (1958). Second dominant gene for resistance to P. coronata races 202 and 258 in the diploid Saia. Marshall and Myers (1961).

Pc-17. Murphy et al. (1958). Third dominant gene for resistance to P. coronata races 202 and 258 in the diploid Saia.

Pc-18. Simons et al. (1959). Dominant gene for resistance to P. coronata races 205, 216, and 264 in the diploid Glabrota. Marshall and Myers (1961).

Pc-19. Simons et al. (1959). Dominant gene for resistance to P. coronata races 205, 227, and 264 in the diploid C.I. 3815. Marshall and Myers (1961).

Pc-20. Simons et al. (1959). Gene for resistance to P. coronata races 202, 203, 205, 216, and 264 in the tetraploid C.I. 7233.

Pc-13c. Chang (1959). Gene for susceptibility to P. coronata race 258 in De Argelia and Dom Pedrito. Designated "B".

Pc-13d. Chang (1959). Second gene for susceptibility to P. coronata race 258 in De Argelia and Dom Pedrito. Designated "b".

Pc-21. Chang (1959). Gene for resistance to P. coronata races 203, 216, and 258 in Santa Fe. Designated "S".

Pc-22. McKenzie (1961). Incompletely dominant gene for resistance to P. coronata races 264, 279, and 290 in Ceirch dubach.

Pc-23. Dyck and Zillinsky (1963). Gene for resistance to P. coronata race 264 (but not race 294) in the diploid C.D. 3820.

Pc-24. Upadhyaya and Baker (1960). Complementary dominant gene conditioning resistance, with Pc-25, to P. coronata races 203, 226, 230, 237, and 286 in Garry. Designated "Vc_a". Upadhyaya and Baker (1962b).

Pc-25. Upadhyaya and Baker (1960). Complementary dominant gene conditioning resistance, with Pc-24, to P. coronata races 203, 226, 230, 237, and 286 in Garry. Designated "Vc_b". Upadhyaya and Baker (1962b).

Pc-26. Upadhyaya and Baker (1960). Gene for resistance to P. coronata in Garry. Designated "Vc_2". Upadhyaya and Baker (1962b).

I-Pc-26. Upadhyaya and Baker (1960). Gene inhibiting Pc-26. Designated "IVc_2".

Pc-27. Upadhyaya and Baker (1960). Gene for adult resistance to P. coronata in Garry. Designated "Vc_1". Upadhyaya and Baker (1962b).

Pc-28. Upadhyaya and Baker (1960). Second gene for adult resistance to P. coronata in Garry. Designated "Vc_3". Upadhyaya and Baker (1962b).

Pc-29. Marshall and Myers (1961). Second gene for resistance to P. coronata race 216 in the diploid Glabrota.

Pc-30. Marshall and Myers (1961). Second gene for resistance to P. coronata races 216 and 276 in the diploid C.I. 3815.

Pc-31. Marshall and Myers (1961). Dominant gene for resistance to P. coronata races 203, 216, and 276 in the diploid C.I. 4746.

Pc-32. Marshall and Myers (1961). Dominant gene for resistance to P. coronata races 203, 216, and 276 in the diploid Ceirch Llwyd.

Pc-33. Marshall and Myers (1961). Second dominant gene for resistance to P. coronata race 216 in the diploid Ceirch Llwyd.

Pc-34. McKenzie and Fleischmann (1964). Gene for resistance to P. coronata races 203, 205, 264, 276, and 279 in D-60.

Pc-35. McKenzie and Fleischmann (1964). Gene for resistance to P. coronata races 203, 205, 264, 276, and 279 in D-137.

Pg-1. Garber (1921). Dominant gene for resistance to stem rust, caused by Puccinia graminis Pers. f. sp. avenae Erikss, and E.Henn., races 1, 2, 5, 8, 8A, 9, 10, and 11 in White Russian. Designated "S" by Dietz (1928) and "D" by Murphy and Coffman (1961). Griffee (1922), Hayes et al. (1928), Smith (1934), Cochran et al. (1945), Kehr et al. (1950), Myers et al. (1955), Koo et al. (1955), Koo et al. (1956), McKenzie and Green (1962), Upadhyaya and Baker (1962a).

I-Pg-1. Dietz (1928). Dominant gene inhibiting the expression of Pg-1 and Pg-2, in Burt.

Pg-2. Dietz (1928). Dominant gene for resistance to P. graminis races 1, 2, 3, 5, 7, 7A, and 12 in Green Russian. Designated "A" by Welsh and Johnson (1954). Welsh (1931), Gordon and Welsh (1932), Smith (1934), Torrie (1939), Litzenberger (1949b), Myers et al. (1955), Koo et al. (1955), Baker (1955), Koo et al. (1956), McKenzie and Green (1962), Upadhyaya and Baker (1962a).

Pg-3. Waterhouse (1930). Dominant gene for resistance to P. graminis races 1, 3, 4, and 11 in Joanette. Designated "E" by Welsh and Green (1958). Welsh (1931), Gordon and Welsh (1932), Welsh and Johnson (1951), McKenzie and Green (1962).

Pg-4. Welsh and Johnson (1954). Dominant gene for resistance to P. graminis races 1, 2, 3, 4, 5, 6, 7, 8, 10, 11, 12, and 13 in RL1225 (derived from Hajira). Designated "B". Litzenberger (1949b), Welsh and Johnson (1951), Baker (1955), Upadhyaya and Baker (1960), McKenzie and Green (1962).

Pg-5. Welsh and Johnson (1954). Dominant gene for resistance to certain races of P. graminis in RL1225 (derived from Hajira). Designated "C" and may be the same as Pg-4. Litzenberger (1949b), Welsh and Johnson (1951), Baker (1955), Upadhyaya and Baker (1960), Upadhyaya and Baker (1962a) designated it "G".

Pg-6. Zillinsky et al. (1956). Dominant gene for resistance to P. graminis races 6, 7, 7A, and 8 in the diploid CD3820. Sadanaga et al. (1960), Dyck and zillinsky (1962).

Pg-7. Zillinsky et al. (1956). Second dominant gene for resistance to P. graminis races 6, 7, 7A, and 8 in the diploid CD3820.

pg-8. Browning and Frey (1959). Recessive gene for resistance to P. graminis races 1, 2, 6, 6A, 7, 7A, 8, 8A, 10, 13, and 13A. Designated "F" by Welsh et al. (1961). Browning and Frey (1962) suggested it may be allelic with Pg-1 or Pg-2.

pg-9. McKenzie and Green (1965). Recessive gene for resistance to P. graminis races 6F and 6AF in C.I. 4529 and other lines. May be allelic with Pg-3. Designated "H".

Pg-10. Pavek and Myers (1965). Dominant gene for mesothetic reaction to P. graminis race 13A in C.I. 1575 and other lines. Designated "G".

psc. Griffiths (1961). Partially recessive gene in Cc4146 for resistance to halo blight, caused by Pseudomonas coronafaciens (Elliott) Stevens.

Pt-1. Nilsson-Ehle (1909). Gene for open panicle type. Designated "A_1". Norton (1907), Wakabayashi (1921), Garber (1922), Quisenberry (1926), Odland (1928), Coffman (1964).

Pt-2. Nilsson-Ehle (1909). Second gene for open panicle type. Designated "A_2". Quisenberry (1926), Coffman (1964).

Pt-3. Nilsson-Ehle. (1909). Third gene for open panicle type. Designated "A_3". Coffman (1964).

Pt-4. Patterson et al. (1959). Dominant gene for dense or cluster panicle type in Milford.

rp-1. Henning (1924). Recessive gene for rachilla pubescence. Odland (1928), Hayes et al. (1928), Ma (1933), Philp (1933), Aamodt et al. (1934), Tang (1938).

Rp-2. Johnson (1933). Dominant gene for long, abundant rachilla pubescence. Designated "N". Coffman (1964).

Rp-3. Johnson (1933). Gene conditioning short rachilla pubescence, hypostatic to Rp-2. Designated "F".

Rp-4. Coffman (1964). Second dominant gene for long, abundant rachilla pubescence.

sc. Pridham (1916). Recessive gene for pinkish straw color in Algerian.

U-1. Wakabayashi (1921). Dominant gene for resistance to covered smut, caused by Ustilago kolleri Wille., in Red Rustproof. Gaines (1925), Nicolaisen (1931), Schattenberg (1934).

U-2. Wakabayashi (1921). Third dominant gene for resistance to U. kolleri in Red Rustproof. Gaines (1925), Nicolaisen (1931), Schattenberg (1934).

U-3. Wakabayashi (1921). Third dominant gene for resistance to U. kolleri in Red Rustproof. Gaines (1925), Schattenberg (1934).

U-4. Barney (1924). Dominant gene for resistance to loose smut, caused by Ustilago avenae (Pers.) Rostr., in Black Mesdag. Reed (1925), Reed (1928), Garber et al. (1928), Garber et al. (1929), Rosenstiel (1929), Nicolaisen (1931), Johnson (1933), Stanton et al. (1934), Reed (1934), Schattenberg (1934), Reed (1935), Reed (1941).

U-5. Barney (1924). Dominant gene for resistance to U. avenae in Fulghum. Cochran et al. (1945).

U-6. Barney (1924). Second dominant gene for resistance to U. avenae in Fulghum.

U-7. Barney (1924). Dominant gene for resistance to U. avenae in Burt.

U-8. Barney (1924). Second dominant gene for resistance to U. avenae in Burt.

U-9. Barney (1924). Dominant gene for intermediate reaction to U. avenae in Golden Rain.

U-10. Reed (1928). Dominant gene for resistance to U. kolleri in Early Gothland. Reed (1931), Reed and Stanton (1937).

U-11. Reed (1928). Dominant gene for resistance to U. avenae in Monarch. Reed (1931), Schattenberg (1934), Reed (1941).

U-12. Gaines and Smith (1929). Gene for resistance to U. kolleri in Markton. Coffman et al. (1931), Schattenberg (1934), Reed and Stanton (1938).

U-13. Gaines and Smith (1929). Second gene for resistance to U. kolleri in Markton. Coffman et al. (1931), Schattenberg (1934), Reed and Stanton (1938).

U-14. Nicolaisen (1931). Dominant gene for resistance to U. avenae in Black Mesdag. Johnson (1933), Schattenberg (1934).

U-15. Nicolaisen (1931). Second dominant gene for resistance to U. avenae in Black Mesdag. Schattenberg (1934).

U-16. Schattenberg (1934). Dominant gene for resistance to U. avenae in Markton. Reed and Stanton (1938).

U-17. Schattenberg (1934). Gene for resistance to U. avenae in Gopher.

U-18. Reed and Stanton (1937). Gene for resistance to U. kolleri in Rossman.

U-19. Reed and Stanton (1937). Gene for resistance to U. kolleri in Danish.

U-20. Reed and Stanton (1937). Gene for resistance to U. kolleri in Scottish Chief.

U-21. Reed and Stanton (1937). Gene for resistance to U. kolleri in Seizure.

U-22. Hayes et al. (1939). Gene for resistance to U. avenae and U. kolleri in Bond. Torrie (1939), Cochran et al. (1945).

U-23. Torrie (1939). Gene for partial resistance to U. avenae and U. kolleri in Bond.

U-24. Torrie (1939). Gene for resistance to U. avenae and U. Kolleri in Victoria. Patel (1941), Cochran et al. (1945).

U-25. Torrie (1939). Gene for partial resistance to U. avenae and U. kolleri in Victoria.

U-26. Reed (1942). Gene for resistance to Ustilago species in Navarro.

U-27. Reed (1942). Gene for resistance to Ustilago species in Navarro.

U-28. Reed (1942). Gene for resistance to Ustilago species in Navarro.

U-29. Reed (1942). Gene for resistance to Ustilago species in Navarro.

U-30. Reed (1942). Gene for resistance to Ustilago species in Navarro.

SYMBOLIZATION OF GENES DISCOVERED IN THE FUTURE

It is proposed that the National Oat Conference serve as a center for assigning symbols to genes in oats that might be discovered and reported in the future. Investigators wishing to have symbols assigned to new genes would send pertinent data regarding the genes to the Chairman of the National Oat Conference. If there was reasonably good evidence indicating that the genes differed from any previously reported, the Chairman, or persons designated by him, would then assign symbols according to the rules outlined above.

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