OATS: A STANDARDIZED SYSTEM OF NOMENCLATURE FOR GENES AND CHROMOSOMES AND CATALOG OF GENES GOVERNING CHARACTERS

IN COOPERATION WITH IOWA AGRICULTURE AND HOME ECONOMICS EXPERIMENT STATION
On January 24, 1978, four USDA agencies—Agricultural Research Service (ARS), Cooperative State Research Service (CSRS), Extension Service (ES), and the National Agricultural Library (NAL)—merged to become a new organization, the Science and Education Administration (SEA), U.S. Department of Agriculture.

This publication was prepared by the Science and Education Administration's Federal Research staff, which was formerly the Agricultural Research Service.
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Washington, D.C.  
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INTRODUCTION

In 1966, a set of rules for symbolization of genes governing characters in oats and a list of genes were published by a committee that had been assigned this task (Simons, Zillinsky, and Jensen, 1966). This publication emphasized the need for a unified system, and noted that standardized systems of genetic nomenclature had been established for corn (Emerson, Beadle, and Fraser, 1935), barley (Robertson, Wiebe, and Immer, 1941), and wheat (Ausemus, Harrington, Reitz, and Worzella, 1946). These and other reports on gene nomenclature were studied before the rules for oat genes were adapted from the guide by the International Committee on Genetic Symbols and Nomenclature (Tanaka et al., 1957).

Experience has shown that this system is basically sound. It can be used with ease by people who are interested in the facets of basic and applied genetics of oats. Since publication of the original set of rules and list of genes, however, several errors have been noted, and many new genes have been discovered. Evidence shows that the rules needed certain minor revisions and some expansion to cover additional aspects of oat genetics. Also, new or revised systems, reflecting new developments and concepts in the field, have been published or proposed for wheat (Kimber and Sears, 1968; McIntosh, 1973), and for barley (Ramage, 1972).

With these thoughts in mind, a second committee, consisting of the authors, was formed, and this publication is the result of their work.


2Simons, Plant pathologist, Agricultural Research Service, U.S. Department of Agriculture, Ames, Iowa 50011; Martens and MacKenzie, plant pathologist and cerealist, Research Branch, Agriculture Canada, Winnipeg, Manitoba, Canada; Nishiyama, professor emeritus, Faculty of Agriculture, Kyoto University, Kyoto 606, Japan; Sadanaga, geneticist, Agricultural Research Service, U.S. Department of Agriculture, Ames, Iowa 50011; Sebesta, plant pathologist, Research Institute of Crop Production, Prague, Czechoslovakia; and Thomas, geneticist, Welsh Plant Breeding Station, University College of Wales, Aberystwyth, United Kingdom.

3Authors and year of publication (italic) in parentheses refer to References, p. 24.
RULES FOR SYMBOLIZING GENES AND CHROMOSOMES OF OATS

The rules for oat gene nomenclature published in 1966 (Simons, Zillinsky, and Jensen, 1966) were based on the report (Tanaka et al., 1957) of a committee appointed by the Permanent International Committee for Genetics Congresses. The general rules established by the Tanaka Committee remain as the basic guide to the rules for oat gene nomenclature. Rules for nomenclature of aneuploids in wheat were established by Kimber and Sears (1968). A summary of the most applicable of these rules, adapted for oats, also is included in our list of rules. (The original publication should be consulted when additional details or examples of the symbolization of aneuploids are needed.)

1. Symbols of genes, derived from the English name of the character involved or from the Latin name of the organism, as in the case of reaction to a pathogen or insect, will be written in Roman letters. The basic symbol will begin with a capital letter, and, with the exception of genes for reaction to living organisms, this symbol will stand for the dominant allele where only two alleles, one dominant, are known. A recessive allele may be indicated by starting the symbol with a small letter. Symbols for all alleles for reaction to living organisms will start with a capital letter (see rule 2). Each symbol will be short, suggestive, different from all other symbols used for oat genes, and contain not more than one capital letter.

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 means that all genes governing reaction to a specific disease organism, without reference to races of the pathogen or cultivars of the host, will have the same basic symbol. Similarly, all genes governing electrophoretically detectable isozymes of the same enzyme 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 discovered for a character will be assigned the number 1, the second 2, and so on. Members of allelic series will be distinguished by small letters that follow immediately after the locus number. The letters a and b will refer to the original allele-pair. For alleles governing reaction to living organisms, the letter a will refer to the allele conditioning resistance and b for the allele conditioning susceptibility, regardless of dominance relationships.

3. Genes identified by use of aneuploids will be given symbols on the same basis as any other genes. Descriptions of such genes will indicate that they were identified by means of aneuploidy and include monoeffect and nullieffect, where appropriate.

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

5. No “wild type” will be recognized as a standard type, and genes occurring in diploid, tetraploid, and hexaploid species will be included in a single system.

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

7. 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.

8. Symbols of extrachromosomal factors will be enclosed within brackets and will precede the formula.

9. Chromosomal aberrations will be indicated by abbreviations: Df for deficiency, Dp for duplication, In for inversion, T for translocation, and Tp for transposition. A summary of rules specifically for nomenclature of aneuploids are:
   a. Complete chromosomes will be designated by Arabic numerals. This rule will apply to chromosomes paired in homologous bivalents, trivalents or other multivalents, and also in the unpaired monosomic condition.
   b. Telocentric chromosomes will be designated by the letter t, whether or not the telocentric chromosome is involved in a pairing configuration. If the telocentric chromosome is paired with a complete chromosome, the telocentric designation will precede the complete chromosome designation.
   c. Isochromosomes will be designated by the small letter i, whether or not the isochromosome is involved in a pairing configuration.
   d. Nullisomics will be indicated by the symbol O, followed by the symbols indicating which chromosome is absent.
   e. The ability of chromosomes to pair will be indicated by superscripts following the chromosome symbols. For example, a euploid plant of *Avena sativa* will be designated as 21", and a monosomic deficiency as 20" + 1'.
   f. At the time this is being written, there is no consensus among different investigators regarding numbering schemes for the chromosome complement. Therefore, rules on designation of chromosomes are being delayed until a standard numbering system can be devised.

10. The zygotic number of chromosomes will be indicated by 2n, the gametic number by n, and the basic number by x.
OAT GENES AND ASSIGNED SYMBOLS

A survey of literature reporting genetic studies of oats attempted 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 cultivars was assumed the same as the gene first reported in that cultivar for that character unless a different gene was evident. Genes conditioning the same basic character, but reported from unrelated cultivars, were assumed to be neither identical nor allelic unless there were some reason.

For genes of characters other than disease reaction, genes governing expression of the same character were assumed identical if they were reported from oats having the same genome constitution, unless there were some reason to think otherwise.

The genes, or loci, recognized are listed alphabetically by symbol. The reference given after each symbol is usually, but not always, the earliest reported discovery of the gene based on the committee's investigations. References listed after the description report additional studies of the gene or pertain to genes now tentatively regarded as the same gene. 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. Descriptions, symbols, and pages on which they appear follow:

Awnedness (A) .................................................. 5
Awn pubescence (Ap) ....................................... 6
Basal articulation (Ba) ...................................... 6
Blast (Bl) ......................................................... 7
Chlorophyll deficiency-albino (Cda) ....................... 7
Chlorophyll deficiency-chlorina (Cdc) ..................... 7
Chlorophyll deficiency-lutescens (Cdl) .................... 7
Chlorophyll deficiency-netting (Cdn) ...................... 8
Chlorophyll deficiency-stripe (Cds) ......................... 8
Chlorophyll deficiency-albovirescens (Cdv) .............. 8
Ditylenchus dipsaci reaction (Dd) .......................... 8
Dwarfness (Dw) ............................................... 8
Esterase (E) .................................................... 8
Erysiphe graminis reaction (Eg) .............................. 8
Floret disjunction (Fd) .................................... 9
Floret development (Fl) ..................................... 9
Fatuoid (Ft) ................................................... 9
Gametophyte (Gf) ............................................ 9
Giantism (Gi) .................................................. 9
<table>
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<th>Gene Symbol</th>
<th>Reference and description</th>
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<td>A-1. Norton (1907). Gene conditioning awnedness in cultivated hexaploid oats. Dominance and expressivity variable. Nilsson-Ehle (1914), Surface (1916), Zinn and Surface (1917), Love and Fraser (1917), Wilds (1917), Love and Craig (1918b), Fraser (1919), Henning (1924), Reed and Stanton (1925), Cotner (1929), Tschermak (1929), Shaw and Bose (1933), Philp (1933), Johnson (1933), Aamodt et al. (1934), De Villiers (1935), Tang (1938), Torrie (1939), Ko et al. (1946), Coffman (1964).</td>
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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), Coffman et al. (1925), Goulden (1926), 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-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, complementary with Ba-6. 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, complementary with Ba-5. Designated "Y."

Ba-7. Nishiyama (1973). Gene for wild type of basal articulation of
primary and secondary florets, in progeny of *A. barbata* x *A. strigosa*. Designated "W."

**Ba-8.** Nishiyama (1973). Complementary gene conditioning, with ba-7b, basifracture of the secondary floret, hypostatic to Ba-7, in progeny of *A. barbata* x *A. strigosa*. Designated "B."

**Ba-9.** Nishiyama (1933). Dominant gene for the cultivated type of basal articulation of the primary and secondary floret. Discovered by monosomic analysis.

**B1-1.** Mackie (1928). Partly dominant gene for resistance to blast in Kanota.

**cda-1.** Nishiyama (1941). Gene for chlorophyll deficiency-albino, linked to ma-1 and L-1, in progeny of *A. barbata* x *A. strigosa*. Designated "al," "G," and "C." Nishiyama (1934).

**cda-2.** Nishiyama (1957). Gene for chlorophyll deficiency-albino, linked to Lp-9 and Le-12, in progeny of *A. barbata* x *A. strigosa*.

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


**cdc-4.** Nishiyama (1957). Recessive gene for yellow plant color, in progeny of *A. barbata* x *A. strigosa*.


**cdl-3.** Froier (1946). Gene conditioning chlorophyll deficiency-
lutescens in the variety Swedish. Designated “Ls.” Åkerman (1922).


Dw-5. Nishiyama (1957). Recessive gene for dwarfness, in progeny of *A. barbata* x *A. strigosa*.


4Personal communication.

Eg-4. Thomas, Leggett, and Jones (1975). Gene for resistance to all currently prevalent races of *Erysiphe graminis* in *Avena barbata* Cc4897. Was transferred to *A. sativa* Manod.


fl-1. Dyck (1968). Recessive gene affecting the normal development of florets of *A. strigosa* spikelets are completely lacking on plants of this mutant.


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


Hv-1. 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).


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5Personal communication.


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).


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), Wakahayashi (1921), 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), Philip (1933), Ma (1933), Aamodt et al. (1934), De Villiers (1935), Åkerman and Bader (1937), Tang (1938), Middleton (1938), Patel (1941), Åkerman (1948), Coffman (1964).

Wilds (1917), Love and Craig (1918b), Caporn (1918), Henning (1924), Meurman (1927), Federova (1930), Welsh (1931), Robb (1932), Johnson (1933), Philip (1933), Ma (1933), Aamodt et al. (1934), Coffman (1964).


Lc-10. Ko et al. (1946). Complementary gene conditioning, with Lc-11, white to yellowish lemma color.


Lf-1. Finkner, Murphy, Atkins, and West (1954). Dominant gene for lemma fluorescence under UV. Designated “F.” May be associated with lemma color.


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


Ma-1. Nishiyama (1941). Dominant gene for early maturity, linked to Cda-1 and 1-1, in progeny of A. barbata x A. strigosa. Designated "Re."


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


Pc-4. Hayes *et al.* (1939). Complementary gene conditioning, with Pc-3, resistance to *P. coronata* in Bond. Torrie (1939), Weet-
man (1942), Cochran et al. (1945), Ko et al. (1946), Litzenberger (1949b), Kehr and Hayes (1950), Griffiths (1953), Šebesta (1976).


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


**Pc-37.** Dyck (1966). Dominant gene for resistance to crown rust race 294 carried by diploid C.D. 7994.

**Pc-38.** Fleischmann and McKenzie (1968). Gene carried by *A. sterilis*

\(^6\)Personal communication.
CW491-4 for resistance to crown rust races 264, 290, 295, 332, and 446—susceptible to race 202.


Pc-42. Fleischmann and McKenzie (1968). Gene carried by *A. sterilis* F83 for resistance to crown rust race 332 but not 264, 290, 295, or 446.


Pc-46. Fleischmann et al. (1971a). Dominant gene for resistance to *P. coronata* races 239, 264, 290, 326, 330, and 332 in *A. sterilis* F290 collected in Israel. Allelic or closely linked to Pc-50.


Pc-49. Fleischmann et al. (1971b.). Dominant gene for resistance to *P. coronata* races 216, 326, 330, 332, and 446 in *A. sterilis* F158 collected in Israel. Subsequent pathological and genetic data (McKenzie and Martens, unpublished) indicate Pc-49 is identical to Pc-40.


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*Personal communication.*


Pc-54. McKenzie and Martens (1976).^ Incompletely dominant gene for resistance to cultures of *P. coronata* races 239, 264, and 295 in *A. sterilis* CAV 1830 and CAV 1832 collected in Turkey. Allelic or closely linked to Pc-35.


Pg-1. Garber (1921). Dominant gene for resistance to stem rust, caused by *Puccinia graminis* Pers. f. sp. *avenae* Eriks, 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). Griffey (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

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^Personal communication.
“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). Allelic or closely linked with Pg-1 and Pg-8.


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.”


conferring seedling resistance (which changes to moderate susceptibility in the adult plant stage) to a wide range of \textit{P. graminis} races. Derived from C.I. 8250 (cv. Kyto from Yugoslavia). Independent of the Pg-2, Pg-4, and Pg-9 loci.


Pg-14. MacKey and Mattsson (1972). Dominant or semidominant gene, carried by S 81 and other lines, that confers resistance to Swedish oat stem rust isolate Leijerstam 6AB 26-59. Designated “N.”


Psc-2. Cheng and Roane (1968). Gene in Dubois, linked to Psc-3, for resistance to halo blight (\textit{Pseudomonas coronafaciens}).

Psc-3. Cheng and Roane (1968). Gene in Victorgrain, linked to Psc-2, for resistance to halo blight (\textit{Pseudomonas coronafaciens}).


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


Px-4c. Smith (1972). Inactive allele for peroxidase in certain oat cultivars. Designated “PXa.”


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).


Tg-1. Gardenhire (1964). Dominant gene for resistance to Toxoptera graminium Rond. (Greenbug) in Russian 77.


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), Cherewick and McKenzie (1969).


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


**CATALOGING AND SYMBOLIZING GENES DISCOVERED IN THE FUTURE**

We propose that we continue to serve as a committee, and that we catalog genes in oats that are discovered and reported in the future. Investigators wishing to have new genes cataloged would send pertinent data to a member of the committee. The committee will then see that the symbols assigned do not duplicate previous symbols. We further propose that new genes be listed annually in the *Oat Newsletter.*
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