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A STANDARDIZED SYSTEM OF NOMENCLATURE FOR GENES GOVERNING CHARACTERS OF OATS
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Crops Research Division
Agricultural Research Service
A STANDARDIZED SYSTEM OF NOMENCLATURE FOR GENES GOVERNING CHARACTERS OF OATS

By M. D. Simons, F. J. Zillinsky, and N. F. Jensen

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 The views of numerous individuals were obtained through correspondence, and several unpublished reports of committees on gene nomenclature in other crop species were also consulted.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.

2 Respectively, pathologist, Crops Research Division, ARS, U.S. Department of Agriculture, Stationed at Iowa State University of Science and Technology, Ames; head, Cereal Crops Section, Research Branch, Canada Department of Agriculture, Ottawa; and professor of Plant Breeding, Department of Plant Breeding, Cornell University, Ithaca, N.Y.
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.


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 \textit{A. sativa}, and between \textit{A. fatua} and \textit{A. sterilis}.


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".


Froier
Stanton
Wakabayashi
Zillinsky
Thomas
Hayes
Ko
Schafer
Jones
Henning
Florell
Norton
al.
Wiggans
Jones
Dyck
"Ko
c-2.
H.
f-3.
f-2.
Eg-1.
dw-3.
dw-2.
Dw-4.
Eg-2.

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".
H. Norton (1907). Gene, with variable dominance, conditioning hulllessness and multiflorous spikelet. Von Tschermak (1910), Gaines (1917), Zinn and Surface
(1917), Caporn (1918), Love and McRostie (1919), Reed (1925), Lebedeff (1930), Chou (1932).


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-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).


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).


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 (1918), 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 (1918), 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-5. Fraser (1919). Second gene for yellow lemma color. Designated "y".


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


Lf-1. Finkner et al. (1954). Dominant gene for lemma fluorescence under UV. Designated "F_1".

Lf-2. Finkner et al. (1954). Second dominant gene for lemma fluorescence under UV. Designated "F_2".

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).


I-Pc-1. Dietz and Murphy (1930). Dominant gene inhibiting Pc-1. Designated "I_1".


I-Pc-3. Cochran et al. (1945). Dominant gene in Richland-Fulghum that inhibits Pc-3. Designated "C_3".


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


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-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-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.


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


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), Upadhayya and Baker (1962a).


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), Upadhayya 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), Upadhayya and Baker (1960), Upadhayya and Baker (1962a) designated it "G".


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.


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).


U-4. Barney (1924). Dominant gene for resistance to loose smut, caused by Ustilago avenae (Pers.) Rostr., in Black Mesoag. 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).


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

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

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

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

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


Reed and Stanton (1937). Gene for resistance to U. kollerl in Rossman.

Reed and Stanton (1937). Gene for resistance to U. kollerl in Danish.


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

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

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

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

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.
AKERMAN, Å.
1922. Untersuchungern über eine in direktem Sonnenlichte nicht lebensfägige Sippe von

1948. Genetiska undersökningar av den svarta skalfiguren hos havre. [Genetic analyses on

_________ and BADER, M.
1937. Über Kreuzungen zwischen Avena sativa und Avena fatua und einige Untersuchungen
über Fatuoiden. [Hybrids between A. sativa and A. fatua and some investigations on

_________ and FRÖIER, K.

AAMODT, O. S., JOHNSON, L. P. V., and MANSON, J. M.
1934. Natural and artificial hybridization of Avena sativa with A. fatua and its relation to the

ANDERSEN, S.
Plkult., Meddel. 68: 179. (Original not seen. Abs. in Plant Breeding Abs. 32: 322. 1962.)

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BAKER, E. P.

BARNEY, A. F.
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BARTLETT, H. H.

BROWNING, J. A., and FREY, K. J.
Plant Dis. Rptr. 48: 768-771.

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1964. Crosses of six monosomics in Avena sativa L. with varieties, species, and chloro-
phyll mutants. Crop Sci. 4: 589-593.

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1959. Analysis of genes conditioning resistance of oat varieties to races of Puccinia
St. Paul. 1959.]

CHOU, C. Y.
1932. A study of the inheritance of hull character and resistance to loose smut in oats from
a cross between A. sativa var. Smut Resistant and A. nuda var. Hullless 407a1-18. [Ph.D.
Thesis, Cornell Univ., Ithaca, N.Y.]
COCHRAN, G. W., JOHNSTON, C. O., HEYNE, E. G., and HANSING, E. D.

COFFMAN, F. A.

PARKER, J. H., and QUISENBERRY, K. S.


COTNER, J. B.

CRAIGMILES, J. P.


DAVIES, D. W., and JONES, E. T.

DAY, A. D.

DE VILLIERS, P. J. R.

DIETZ, S. M.

MURPHY, H. C.

DYCK, P. L., and RAJHATHY, T.

ZILLINSKY, F. J.

ZILLINSKY, F. J.

EMERSON, R. A., BEADLE, G. W., and FRASER, A. C.

FEDEROVA, N.

FINKNER, R. E., ATKINS, R. E., and MURPHY, H. C.

WEST, D. W.


HAYES, H. K., GRIFFEE, F., STEVENSON, F. J., and LUNDEN, A. P.

MOORE, M. B., and STAKMAN, E. C.

HENNING, L. J.

JOHNSON, L. P. V.

JONES, E. T.

1940. A comparison of the segregation of wild versus normal or cultivated base in the grain of diploid, tetraploid, and hexaploid species of oats. Genetica 22: 419-434.

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HAYES, H. K., MOORE, M. B., and STAKMAN, E. C.

KO, S. Y., TORRLE, J. H., and DICKSON, J. G.

KOO, K. S., MOORE, M. B., MYERS, W. M., and ROBERTS, B. J.


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LEWIS, R. D.
LITZENBERGER, S. C.


LOVE, H. H., and CRAIG, W. T.
1918a. Small grain investigations. J. Hered. 9: 67-76.
and CRAIG, W. T.
and FRASER, A. C.
and MC ROSTIE, G. P.

MA, P. C.

MC KENZIE, R. I. H.
and FLEISCHMANN, G.
and GREEN, G. J.
and GREEN, G. J.

MACKIE, W. W.

MARSHALL, H. G., and MYERS, W. M.

MEURMAN, OLAVID.

MIDDLETON, G. K.

MOREY, D. D., and EARHART, R. W.

MURPHY, H. C., and COFFMAN, F. A.
and MEEHAN, FRANCES.
and STANTON, T. R., and STEVENS, H.
MURPHY, H. C., ZILLINSKY, F. J., SIMONS, M. D., and GRINDELAND, R.

MYERS, W. M., KOO, F. K. S., MOORE, M. B., and ROBERTS, B. J.

NICOLAISEN, W.

NILSSON/EHLE, H.


NISHIYAMA, I.

NORTON, J. B.

ODLAND, T. E.

OSLER, R. D., and HAYES, H. K.

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REED, G. M.


REED, G. M.


and STANTON, T. R.


and STANTON, T. R.


and STANTON, T. R.


RIVERS, G. M.

ROBB, W.

ROBERTSON, D. W., WIEBE, G. A., and IMMER, F. R.

ROSENSTIEL, K. V.

RU, S. H.

SADANAGA, K., MURPHY, H. C., and GRINDELAND, R.

SANDERSON, K. E.

SCHAFER, E. G.

SCHATTENBERG, H.

SHAW, F. J. F., and BOSE, R. D.

SIMONS, M. D.

and MURPHY, H. C.
SIMONS, M. D., SADANAGA, K., and MURPHY, H. C.

SMITH, D. C.


STANTON, T. R.


SURFACE, F. M.

TANAKA, Y. (COMMITTEE CHAIRMAN) et al.

TANG, S. Y.

THOMAS, H., and RAJHATHY, T.

TORRIE, J. H.

TSCHERMAK, E. von.


UPADHYAYA, Y. M., and BAKER, E. P.


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