Inheritance of 3-Hydroxytyramine in Sugarbeet; a Phenolic Compound Associated with Cercospora Leaf Spot Resistance[1]

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A phenolic compound (3-hydroxytyramine) present in sugarbeet leaves, *Beta vulgaris* L., is related to resistance to *Cercospora beticola* Sacc. (3).[3] Oxidized 3-hydroxytyramine is toxic to *C. beticola* in pure culture (3). There is usually an increase in the 3-hydroxytyramine content of leaves as a result of leaf spot infection, particularly in susceptible genotypes (authors' unpublished 1968 data). Harrison *et al.* (2) reported that 3-hydroxytyramine increased in response to artificial injury. The exact relationship of 3-hydroxytyramine and Cercospora resistance has not been established. Resistance might depend partly on the ability of the plant to provide oxidized 3-hydroxytyramine or some other inhibitory compound upon injury or other stimulation rather than entirely on the original 3-hydroxytyramine concentration. However, it appears that the inherent concentration of 3-hydroxytyramine is important, since correlations of 3-hydroxytyramine content with leaf-spot-resistance ratings show that most resistant varieties and genotypes, under both diseased and disease free conditions, contain more 3-hydroxytyramine than susceptible types (3, 5).

Even though the role of 3-hydroxytyramine in Cercospora resistance has not been defined, we made a genetic analysis of this character to determine the genetic differences between two inbred lines of sugarbeet. We used the partitioning method of genetic analysis, Powers *et al.* (8). Obviously, the more that is known about the genetics of a quantitative character, the more direct the approach to breeding or modification of the character. When the precise role of 3-hydroxytyramine in Cercospora resistance is established, information about the inheritance of 3-hydroxytyramine may be important in selecting and breeding for resistant genotypes.

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[3] Numbers in parentheses refer to literature cited.

Methods and Materials

The experiment was conducted under leaf spot free conditions at the Colorado State University Agronomy Research Center in the summer of 1967. We used two inbred lines of sugarbeet, 52-305 (very high 3-hydroxytyramine) and 52-407 (relatively low 3-hydroxytyramine). Both were long term inbreds; each had been inbred the equivalent of at least 10 generations of selfing. In previous leaf spot tests 52-305 and 52-407 were rated as moderately resistant and susceptible, respectively. We used both 52-305 male fertile (MF) and its cytoplasmic male sterile (CMS) equivalent to develop the experimental populations. Inbred 52-305 CMS resulted from six generations of backcrossing where 52-305 was the recurrent parent. We developed F_1, F_2, and first backcross generations to 52-305 and 52-407. Hence, there were three segregating generations (F_2, B_1P_1, and B_1P_2) and three non-segregating generations (P_1, P_2, and F_1) in the experiment. Twenty F_1 plants were interpollinated for production of the F_2 generation. Twenty F_1 plants were used in developing each backcross generation. The experimental design was a randomized complete block with 40 replications. We used 20-foot single row plots with 22 inches between rows, and about 10 inches between plants within a row. A common competitor of relatively low vigor was grown between plot rows. Leaves for 3-hydroxytyramine determinations were harvested on August 2 and 3, the approximate time of the growing season when, according to Harrison *et al.* (2), 3-hydroxytyramine content should be maximized. Six plants of each population in each replication were individually sampled. The four youngest fully expanded leaves on each plant were quick frozen at the field using dry ice. These samples were stored at —29 C until analyzed. The determination of 3-hydroxytyramine content is a colorimetric process and has been described by Harrison *et al.* (2). 3-hydroxytyramine was measured as mg/100 g of frozen leaf. There were 240 individual plant determinations in each population. More plants of each population would have been desirable. However, the time and expense of making 3-hydroxytyramine determinations limited our study.

The partitioning method of genetic analysis as used in this study, is a means of qualitating a quantitative character with the possibility, then, of determining gene number and type of gene action conditioning the character. This approach to the genetic analysis of quantitative characters is not new; it was implied in Nilsson-Ehle's analysis of triplicate factors in wheat kernel color in 1911. It was further developed by other investigators but was brought to its most advanced form by Powers *et al.* (8) and Powers (7).

Our application of the partitioning method requires approximately normal frequency distributions of non-segregating populations (environmental distributions). In an attempt to normalize the data three scale changes were tested. Since estimates of environmental variance without replication variability were used to develop theoretic frequency distributions, replication variability was adjusted out of the data using methods described by Powers *et al.* (9).

In using the partitioning method of genetic analysis, it is necessary to develop a genetic hypothesis or model. and, from this model, calculate theoretic means, genetic variances, and frequency distributions for the segregating generations. Differences from obtained means, genetic variances, and frequency distributions are then tested for significance. Any preliminary information available from the data which might help in formulating a more accurate genetic model is most useful.

Results

Since approximate normality of environmental distributions is an assumption basic to the use of the partitioning method (as applied in this study), the obtained frequency distributions of the non-segregating populations were tested for normality. All populations except 52-407 (P_2) had chi square P values of less than 0.01 and were not normally distributed. All populations were skewed significantly to the left and all had significant positive kurtosis. These tests of third and fourth moment statistics are considered to be quite sensitive tests for deviations from normality.

Three scale changes were tested, namely, square root, 4th root, and $\log_{10}$ of 3-hydroxytyramine. The square root transformation removed all positive kurtosis from all populations but did not remove the positive skewness from any of the populations. The $\log_{10}$ transformation removed the positive kurtosis from three populations and removed all skewness to the left, but introduced, at the same time, skewness to the right in four populations. The fourth root transformation removed all skewness and kurtosis from all non-segregating populations. This transformation resulted in normal distributions of all populations when the calculated normal and obtained distributions were compared by chi square tests (Table 1). After adjustment to remove replication effects and transformation to 4th root (Table 1), the data had no significant third and fourth moment statistics. The chi square tests for normality gave an acceptably high P value (indicating normality) for all distributions except the F_2 where $0.02<P<0.05$. Genetic segregation in the F_2 was

Table 1.—Skewness, kurtosis (g_1 and g_2 statistics), and chi square tests for normality of $\sqrt[4]{\text{3-hydroxytyramine}}$ on both unadjusted and adjusted (to remove replication effect) data.

Population	Third moment (g_1)		Fourth moment (g_2)		Chi square for normality
	Skewed right	Skewed left	Positive kurtosis	Negative kurtosis	P value between
Unadjusted 4th root data:					
52-305 CMS (P_1)	—0.114		0.166		0.90 - 0.80
52-407 MF (P_2)		0.199		—0.451	0.30 - 0.20
F_1	—0.099		0.114		0.50 - 0.30
F_2	—0.112			—0.260	0.98 - 0.95
B_1P_1		0.021		—0.339	0.95 - 0.90
B_1P_2		0.134		—0.650*	0.20 - 0.10
4th root data adjusted for replication:					
P_1		0.236		—0.096	0.50 - 0.30
P_2	—0.114			—0.522	0.99 - 0.98
F_1		0.139	0.072		0.20 - 0.10
F_2		0.328		—0.417	0.05 - 0.02
B_1P_1		0.059	0.019		0.30 - 0.20
B_1P_2		0.258		—.390	0.50 - 0.30

*denotes significance at the 5% level.

assumed to have caused the slight deviation from normality. However, it is not necessary that the genetic variability be of any certain form. The adjusted 4th root data for P_1, P_2, and F_1 were distributed normally; this permitted use of the partitioning method.

Means and variances for the transformed data are shown in Table 2. The total within plot variances did not include variability due to replications. In order to estimate genetic variances necessary in the partitioning method, an estimate of environmental variance was needed first. A comparison of total within plot variances for the non-segregating populations showed that they were not homogeneous; the means and variances were related. Hence, the mean of their total within plot variances

Table 2.—Means, variances (total, total within plot, and genetic), and heritabilities for $\sqrt[4]{\text{3-hydroxytyramine}}$. Environmental variance estimated by regression equation $\hat{Y}$ —0.60668 + 0.453159X — 0.063671X²

Population	Mean	Total variance	Total w/in plot variance	Genetic variance	Heritability (h^2)
52-305 CMS (P_1)	3.005±0.035	0.2958	0.1988		
52-407 MF (P_2)	2.216±0.020	0.0964	0.0352		
F_1	2.719±0.031	0.2337	0.1673		
F_2	2.859±0.037	0.3363	0.2187	0.0502	0.23
B_1P_1	2.837±0.038	0.3441	0.2031	0.0366	0.18
B_1P_2	2.340±0.028	0.1901	0.1231	0.0180	0.15

could not be used as an estimate of environmental variance. Expanding on methods of Powers (6), the quadratic regression equation of within plot variances on plot means for the three non-segregating populations was developed. The multiple correlation coefficient was 0.31 with 117 degrees of freedom. It was significant at the 1% level. Therefore, from the quadratic regression equation, $\hat{Y} = -0.60668 + 0.453159X - 0.063671X^2$, developed from plot means and total within plot variances of 4th root 3-hydroxytyramine data for the three non-segregating populations, the environmental variance for any mean within the range of the P_1 and P_2 means could be estimated. The environmental variances for the three segregating populations (F_2, B_1P_1, and B_1P_2) were estimated using this equation together with the F_2, B_1P_1, or B_1P_2 mean. These estimates were then subtracted from the total within plot variances to estimate the genetic variances for the three segregating populations. These estimates of genetic variance include both additive and non-additive genetic variance, and provide estimates of broad sense heritability (h^2) as well as a necessary comparison with theoretic genetic variances in the partitioning method. Heritabilities (h^2) for the F_2, B_1P_1, and B_1P_2 in Table 2 were quite low, ranging from 0.23 to 0.15, which indicated that environment was contributing the greatest amount to the total variance. This added to the difficulty of making an accurate genetic analysis.

Preliminary study of means, genetic variances, and frequency distributions indicated that the inheritance of 3-hydroxytyramine in this case was not simple. The mid-parent value, $\frac{P_1 + P_2}{2}$, was 2.610 while the F_1 was 2.719, indicating partial dominance for high 3-hydroxytyramine. Also $\frac{F_1 + P_2}{2} = 2.468$ while $B_1P_2 = 2.340$, indicating partial dominance for low 3-hydroxytyramine. At the same time $\frac{F_1 + P_1}{2} = 2.862$ while $B_1P_1 = 2.837$; this close comparison indicated no dominance. The F_2 was 2.859 which was higher than the F_1. This difference can most logically be explained by epistasis; it probably did not result from dominance, linkage, or transgressive segregation.

Preliminary consideration of number of major genes, or groups of closely linked genes which tend to act as a single gene, indicated that there was probably more than one gene involved. However, in applying the partitioning method of genetic analysis the logical approach is to start with the simplest

possible genetic hypothesis. In this case this would be one locus (P_1 = AA, P_2 = aa), no dominance, with each A allele adding 0.3945 to the P_2 mean of 2.216. Even though it was likely that P_1 and P_2 were genetically different at more than one locus with respect to 3-hydroxytyramine, this 1-gene model was tested for exemplary purposes (Table 3). Since in Table 3 the F_2 and B_1P_2 theoretic and obtained means differ significantly, as do their theoretic and obtained genetic variances, the 1-gene hypothesis must be rejected. The F_2 statistics are likely more critical than the backcrosses; however, the theoretic and obtained statistics for all segregating populations must not differ significantly. Further, the theoretic frequency distributions for the F_2, B_1P_1, and B_1P_2 can be calculated and compared with the obtained distributions. In the case of the 1-gene hypothesis in Table 3 there was no need to compare frequency distributions, since the hypothesis had been rejected already on the basis of means and variances.

We tested other 1-gene models which included varying degrees of dominance. All such models were rejected. Genetic penetrance or expressivity were not useful modifications. We concluded that the difference between P_1 and P_2 could not be explained by the genetic hypothesis of one effective factor difference. We then tested more complex genetic models. Models involving two and three loci with linkage of varying intensity,

Table 3.—Theoretic means and genetic variances for $\sqrt[3]{\text{3-hydroxytyramine}}$. Genetic hypothesis: P_1 = AA, P_2 = aa, no dominance

Pop.	Genotype	Genotype mean (X)	Genotype frequency (F)	X^2
F_2	AA	3.0050	0.25	9.0300
	Aa	2.6105	0.50	6.8147
	aa	2.2160	0.25	4.9107
	$\Sigma(XF) = 2.6105$		theoretic mean = 2.61	**
	$\Sigma(X^2F) = 6.8925$		obtained mean = 2.86	
	$CF = [\Sigma(\bar{X}F)]^2 = 6.8147$		theoretic genetic variance = 0.0778	**
	$\Sigma x^2/1 = 0.0778$		obtained genetic variance = 0.0502	
B_1P_1	AA	3.0050	0.50	9.0300
	Aa	2.6105	0.50	6.8147
	$\Sigma(\bar{X}F) = 2.8078$		theoretic mean = 2.81	
	$\Sigma(X^2F) = 7.9223$		obtained mean = 2.84	
	$CF = [\Sigma(XF)]^2 = 7.8836$		theoretic genetic variance = 0.0387	
	$\Sigma x^2/1 = 0.0387$		obtained genetic variance = 0.0366	
B_1P_2	Aa	2.6105	0.50	6.8147
	aa	2.2160	0.50	4.9107
	$\Sigma(\bar{X}F) = 2.4133$		theoretic mean = 2.41	**
	$\Sigma(X^2F) = 5.8627$		obtained mean = 2.34	
	$CF = [\Sigma(XF)]^2 = 5.8240$		theoretic genetic variance = 0.0387	**
	$\Sigma x^2/1 = 0.0387$		obtained genetic variance = 0.0180	

**denotes a significant difference (1%) between the paired means or variances.

isodirectional and nonisodirectional effects, and various epistatic effects were all inadequate to describe the obtained data. In general our greatest difficulty in developing a genetic model that described the obtained data was due to the rather high F_2 mean and low B_1P_2 mean. Models that included linkage tended to give genetic variances which were higher than obtained genetic variances. Linkage of any combination and intensity resulted in the model providing a poorer fit to the obtained means, genetic variances, and frequency distributions.

Table 4.—Genotypes in the F_2 of a 4-gene model together with their means (based on the model) for $\sqrt[4]{\text{3-hydroxytyramine}}$ and expected frequencies

Genotype	Mean	Frequency	Genotype	Mean	Frequency
AABBCCDD[1]	3.0050	0.00390625	AaBbCcDd[1, 2]	2.7190	0.06250000
AABBCCDd[1]	3.0050	0.00781250	AaBbCcdd[2]	2.7190	0.03125000
AABBCCdd	2.9750	0.00390625	AaBbccDD	2.9500	0.01562500
AABBCcDD[1]	3.0050	0.00781250	AaBbccDd[2]	2.5250	0.03125000
AABBCcDd[1]	3.0050	0.01562500	AaBbccdd[2]	2.5250	0.01562500
AABBCcdd	2.9750	0.00781250	AabbCCDD	2.9600	0.00781250
AABBccDD	2.9700	0.00390625	AabbCCDd	2.9550	0.01562500
AABBccDd	2.9700	0.00781250	AabbCCdd	2.9550	0.00781250
AABBccdd	2.9650	0.00390625	AabbCcDD	2.9500	0.01562500
AABbCCDD[1]	3.0000	0.00781250	AabbCcDd[2]	2.4750	0.03125000
AABbCCDd[1]	3.0000	0.01562500	AabbCcdd[2]	2.4750	0.01562500
AABbCCdd	2.9700	0.00781250	AabbccDD	2.9500	0.00781250
AABbCcDD[1]	3.0000	0.01562500	AabbccDd[2]	2.3500	0.01562500
AABbCcDd[1]	3.0000	0.03125000	Aabbccdd[2]	2.3500	0.00781250
AABbCcdd	2.9700	0.01562500	aaBBCCDD	2.9600	0.00390625
AABbccDD	2.9650	0.00781250	aaBBCCDd	2.9600	0.00781250
AABbccDd	2.9650	0.01562500	aaBBCCdd	2.9550	0.00390625
AABbccdd	2.9600	0.00781250	aaBBCcDD	2.9450	0.00781250
AAbbCCDD	2.9700	0.00390625	aaBBCcDd	2.9450	0.01562500
AAbbCCDd	2.9700	0.00781250	aaBBCcdd	2.9400	0.00781250
AAbbCCdd	2.9650	0.00390625	aaBBccDD	2.9350	0.00390625
AAbbCcDD	2.9700	0.00781250	aaBBccDd	2.9350	0.00781250
AAbbCcDd	2.9700	0.01562500	aaBBccdd	2.9300	0.00390625
AAbbCcdd	2.9650	0.00781250	aaBbCCDD	2.9600	0.00781250
AAbbccDD	2.9600	0.00390625	aaBbCCDd	2.9550	0.01562500
AAbbccDd	2.9600	0.00781250	aaBbCCdd	2.9550	0.00781250
AAbbccdd	2.9550	0.00390625	aaBbCcDD	2.9500	0.01562500
AaBBCCDD[1]	2.9950	0.00781250	aaBbCcDd[2]	2.3400	0.03125000
AaBBCCDd[1]	2.9950	0.01562500	aaBbCcdd[2]	2.3440	0.01562500
AaBBCCdd	2.9700	0.00781250	aaBbccDD	2.9500	0.00781250
AaBBCcDD[1]	2.7000	0.01562500	aaBbccDd[2]	2.3100	0.01562500
AaBBCcDd[1]	2.7000	0.03125000	aaBbccdd[2]	2.3100	0.00781250
AaBBCcdd	2.9500	0.01562500	aabbCCDD	2.9350	0.00390625
AaBBccDD	2.9700	0.00781250	aabbCCDd	2.9000	0.00781250
AaBBccDd	2.9700	0.01562500	aabbCCdd	2.9000	0.00390625
AaBBccdd	2.9650	0.00781250	aabbCcDD	2.9500	0.00781250
AaBbCCDD[1]	2.5000	0.01562500	aabbCcDd[2]	2.3000	0.01562500
AaBbCCDd[1]	2.5000	0.03125000	aabbCcdd[2]	2.3000	0.00781250
AaBbCCdd	2.9500	0.01562500	aabbccDD	2.9500	0.00390625
AaBbCcDD[1]	2.7190	0.03125000	aabbccDd[2]	2.2160	0.00781250
			aabbccdd[2]	2.2160	0.00390625

1 = genotypes which also appear in B_1P_1
2 = genotypes which also appear in B_1P_2

Four-gene models were inadequate without epistasis. One 4-gene model, including complex epistatic combinations, was developed that satisfactorily described the obtained means and genetic variability. The 81 genotypes in the F_2 of this 4-gene model, with their frequencies and means, are listed in Table 4; the B_1P_1 and B_1P_2 genotypes are designated. The frequency of each of the 16 backcross genotypes in the B_1P_1 and B_1P_2 was 0.0625. Table 4 shows that this 4-gene model included many complex interactions, some of which are difficult to justify on the basis of known types of genetic interaction, although it is not genetically impossible that such interactions could exist. The obtained means and variances served as guides in specifying the interactions; hence the model was "made to fit" the obtained data. However 2- and 3-gene models could not be made to fit regardless of the number and type of interactions injected into the model. Table 5 lists the obtained and theoretic values for means and genetic variances developed from the genetic model in Table 4. It also shows goodness-of-fit chi square P values for obtained and theoretic frequency distributions. The means, genetic variances, and frequency distributions calculated from the 4-gene model in Table 4 were not significantly different than the obtained statistics of the F_2, B_1P_1, and B_1P_2. Therefore, the 4-gene model in Table 4 was adequate to describe the obtained results of the three segregating populations. This 4-gene model with complex interactions was the simplest genetic explanation for the inheritance of 3-hydroxytyramine in these populations. However, considering the complexity of the genetic interactions in the model, it is likely that 3-hydroxytyramine in this case was conditioned by more than four genes, with somewhat less complex interactions than were necessary in the 4-gene model. Unfortunately, the number of individuals in each population (240) was not large enough to permit accurate testing of genetic models with more than four genes.

Table 5.—Comparison of $\sqrt{3\text{-hydroxytyramine}}$ obtained and theoretic means, genetic variances, and frequency distributions for the F_2, B_1P_1, and B_1P_2 (theoretic values based on 4-gene model in Table 4)

Pop.	Mean		Genetic variance		Freq. dist. chi square P values
	Theoretic	Obtained	Theoretic	Obtained	
F_2	2.77	2.86	0.059	0.050	$0.05<P<0.10$
B_1P_1	2.87	2.84	0.037	0.037	$0.80<P<0.90$
B_1P_2	2.41	2.34	0.023	0.018	$0.50<P<0.70$

99% confidence intervals on obtained means:
F_2 $2.77< \bar{X} < 2.95$
B_1P_1 $2.75< \bar{X} < 2.92$
B_1P_2 $2.28< \bar{X} < 2.41$

Other methods of estimating gene number have been developed. A common method is Castle's formula (1) which has very limiting assumptions: 1) isogenic parents; 2) genes with large effect all in one parent; 3) equal effect of all genes with no dominance or epistasis; and 4) no linkage. Violation of any of these assumptions results in the estimated gene number being smaller than the actual number. In our case all the assumptions may have been violated. The gene number was nonetheless estimated as:

$$\frac{(\bar{X}_{P_1} - \bar{X}_{P_2})^2}{8(s^2_{F_2} - s^2_{F_1})} = \frac{(3.005 - 2.216)^2}{8(0.2187 - 0.1673)} = 1.514$$

Hence, two genes was a minimal estimate of gene number conditioning 3-hydroxytyramine in this study. This supports the estimate of four or more genes by using the partitioning method, but it provides little useful information by itself.

Discussion

This study is part of a general investigation of the biochemical nature of Cercospora leaf spot resistance in sugarbeet. In spite of considerable domestic and foreign breeding research effort, adequate leaf spot resistance for all beet growing areas has not been developed and incorporated into commercial varieties. Under disease conditions mass selection combined with motherline breeding, inbreeding, and formation of synthetic varieties and hybrids has resulted in a level of resistance that is adequate for marginal leaf spot areas, but inadequate for complete protection in primary leaf spot environments. Identification of resistant genotypes has been difficult. It is possible that information on the biochemical basis of leaf spot resistance may lead to methods of accurately identifying and isolating resistant genotypes, preferably under disease free conditions, which can then be incorporated or transferred into commercial varieties and hybrids.

At present, only a general relationship between 3-hydroxytyramine content and leaf spot resistance has been established. However, genetic information about 3-hydroxytyramine is expected to be useful in the over-all research program.

It is apparent from this study and others (4) that 3-hydroxytyramine content is characterized by high environmental variance. This is indicated in the present study by low heritability in the F_2 which was a product of two homozygous inbreds with quite different means. This detracts from the precision of inheritance studies, but in this study there was sufficient genetic

variability to allow a genetic analysis by the partitioning method. In a genetic analysis such as this, it is always best to use parents that are not greatly different, with the likelihood that their gene differences for the characteristic of interest might be few, unless there is specific interest in particular parental populations. P_1 and P_2 in this study had quite different 3-hydroxytyramine means, in spite of the fact that P_1 was rated as only moderately resistant and P_2 as susceptible. However, even greater mean differences exist among other inbreds which have been inventoried for 3-hydroxytyramine content. Choice of more similar parents may have resulted in inadequate genetic variance for reliable partitioning of the segregating populations.

Genotype by environment interaction is a factor which has not been considered. It is conceivable that our genetic conclusions could have been different under another environment, although this is not common where only a few major genes are involved.

In our opinion the genetic results from this partitioning method of genetic analysis are valid. By using this analysis method it was impossible to find any model with less than four genes which fit the obtained F_2, B_1P_1, and B_1P_2 data regardless of dominance, distribution of dominant genes among parents, linkage, penetrance, and epistasis included in the model. Hence, we concluded that the F_2, B_1P_1, and B_1P_2 generations were segregating for four or more effective genetic factors. We consider it probable that more than four were involved because of the complex interactions necessary to make the 4-gene model describe the obtained data. Thus the inheritance of 3-hydroxytyramine was relatively complex. This does not exclude the possibility that it may be conditioned by fewer genes in other cases.

The two parental inbreds in this study are of no direct interest in breeding for leaf spot resistance, and we do not know how genetically typical they are with respect to 3-hydroxytyramine content. So it is not logical to make conclusions about all sugarbeets based on these two genotypes. But it is certain that 3-hydroxytyramine content is not simply inherited throughout the species. If 3-hydroxytyramine content becomes important in evaluation of leaf spot resistance, its inheritance in adapted germ plasm probably will be complex, and make selection and breeding for 3-hydroxytyramine content a difficult project.

Summary

A phenolic compound (3-hydroxytyramine) in sugarbeet is quantitatively related in some way to Cercospora leaf spot resistance (the exact relationship has not yet been established).

Anticipating that greater 3-hydroxytyramine content may be of interest in breeding for higher levels of leaf spot resistance, a limited study of its inheritance was conducted. From high and low 3-hydroxytyramine inbreds, the F_1, F_2, B_1P_1, and B_1P_2 generations were developed and their 3-hydroxytyramine content was measured. The partitioning method of genetic analysis was applied to the data. Genetic models involving different numbers of genes, varying additive gene effects, different degrees of dominance, epistasis, and linkage were tested in an attempt to explain the obtained F_2 and B_1 data. The quantity of 3-hydroxytyramine appeared to be conditioned by four or more genes. The 4-gene model which satisfactorily described the obtained means, genetic variances, and frequency distributions was isodirectional and included additive, partial dominance, and epistatic effects. Some of the interactions were complex and difficult to justify on the basis of commonly known types of genetic interaction. It was likely that 3-hydroxytyramine was conditioned by more than four genes with partial dominance, but with somewhat less complex interactions than were present in our 4-gene model. Population size (240) limited the analysis to 4-gene models. If the two inbreds in this study are typical of genotypes in the species, selection and breeding for higher 3-hydroxytyramine content will very likely be difficult because of multiple factors, dominance, and complex interactions.

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