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1. Comparison of fatty acid percentages of diploid and monosomic Zea mays embryos.*

Zea mays monosomics were employed to screen for genetic factors exhibiting dosage effects that alter maize embryo fatty acids. By comparing a given monosomic with its diploid siblings one compares simultaneously one versus two copies of every gene on that monosomic chromosome. We have successfully used this approach to detect genetic factors controlling total embryo lipids (Plewa and Weber, 1973a). The purpose of this study was to compare fatty acid profiles from specific monosomic types with those in their diploid siblings to determine if an alteration of gene dosage has an effect on fatty acid biosynthesis.

The maize monosomics were generated by the r-X1 deficiency, an X-ray-induced deficiency including the R locus in chromosome 10. The deficiency induces a high rate of chromosomal nondisjunction during the megagametophyte divisions. The r-X1 deficiency in inbred W22 was generously provided by Kante Satyanarayana.

R/r-X1 plants used as females were crossed by inbred Mangelsdorf's multiple chromosome tester that was also r/r. Scutellum samples were taken from each kernel of the r/r-X1 population and stored separately. The sampled kernels were planted and presumptive monosomics were detected by genetic markers. All presumptive monosomic plants were confirmed by chromosome counts. Since the r/r-X1 population was an F_1 produced by crossing two highly inbred lines, the r/r-X1 population was highly isogenic except for aneuploidy. In this study we compared monosomics 2, 7, 8, and 10 with diploid sibling controls.

The lipids were solvent extracted from scutellum samples and the fatty acids were esterified and analyzed by gas-liquid chromatography according to the methods of Plewa and Weber (1973b).

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The results are presented in Tables 1 and 2. The data from Table 1 indicate that embryo fatty acid profiles of monosomic 7 or 10 subpopulations do not greatly differ from the diploid control. There is a lower stearic acid percentage in monosomic 8 embryos than in the diploid control. However, the values for palmitic acid, oleic acid, and linoleic acid do not significantly differ from the control values. The monosomic 2 subpopulation has significantly increased oleic acid and decreased linoleic acid percentages as compared to the control subpopulation. There was also an increase in the palmitic acid percentage in the monosomic 2 subpopulation.

The control subpopulation and monosomic 7, 8, and 10 subpopulations are relatively homogeneous for the percentages for each fatty acid. However, the monosomic 2 subpopulation was surprisingly heterogeneous for the percentages of each fatty acid (Table 2). The only embryos analyzed exhibiting increased percentages of oleic acid and decreased percentages of linoleic acid were monosomic 2 embryos. As indicated in Table 2, certain fatty acid profiles from monosomic 2 embryos were similar to the control profiles.

The fact that monosomic 7 and 10 subpopulations were not significantly different from the control subpopulation indicates that monosomy *per se* does not significantly alter the relative proportions of the various fatty acids in maize embryos. Since monosomic 8 embryos were consistently lower in stearic acid, genetic factors located in chromosome 8 may determine the stearic acid content in maize embryos. Monosomic 2 embryos were highly variable, and the fact that some embryos had fatty acid profiles similar to control profiles has not been resolved. However, most of the embryos had significantly increased oleic acid and decreased linoleic acid percentages. These data support the hypothesis that oleic acid is the precursor of linoleic acid in higher plants (Mazliak, 1973). It is interesting to note that monosomic 2 embryos with decreased linoleic acid percentages had higher palmitic acid percentages. This suggests that preventing the desaturation of oleic acid to linoleic acid may cause an increase in fatty acid precursors of oleic acid. Thus, genetic factors are located in chromosome 2 that are involved in the conversion of oleic acid to linoleic acid.

Table 1

Comparison of mean fatty acid percentages in diploid and monosomic embryos

Chromosome constitution	Number analyzed	Fatty acids:mean per cent*			
		Palmitic	Stearic	Oleic	Linoleic
Diploid control	42	16.12±0.44	2.80±0.01	34.33±0.46	46.76±0.53
Monosomic 2	11	23.14±1.99	3.41±0.48	40.48±2.15	32.93±3.37
Monosomic 7	9	20.12±1.91	2.31±0.43	32.31±1.10	45.33±1.82
Monosomic 8	22	18.84±3.55	1.04±0.22	33.69±0.64	44.00±0.75
Monosomic 10	7	16.83±1.83	3.23±0.18	35.75±1.20	44.06±2.02

* ±1 standard error of the mean

Table 2

Fatty acid profiles of monosomic 2 embryo lipids

Sample number	Fatty acid percentages			
	Palmitic	Stearic	Oleic	Linoleic
MP34-37	14.67	3.01	39.45	42.07
*MP34-43	39.68	7.43	44.04	8.96
MP77-08	16.38	4.85	58.00	20.78
MP77-35	27.53	2.64	37.66	32.18
**MP77-49	23.16	1.95	38.56	36.36
MP81-81	22.90	1.78	35.37	39.95
MP81-110	18.72	2.54	38.16	40.59
MP83-73	24.66	2.69	34.53	38.12
MP84-62	18.56	3.28	35.68	42.51
MP84-89	20.76	2.60	33.71	42.95
MP85-98	27.47	4.69	50.08	17.76
Mean	23.14	3.41	40.48	32.93

* Double monosomic, 2-8
** Double monosomic, 2-7

Considering the altered stearic acid percentages in monosomic 8 embryos and the great differences exhibited by the monosomic 2 fatty acid profiles, it is clear that altering the gene dosage in certain chromosomes has profound effects on fatty acid levels. Thus, monosomic analysis has proven to be a valuable system in locating genes that express dosage effects controlling a biochemical pathway.

References:

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2. A monosomic mapping method.*

Recently, massive numbers of chemically induced maize mutants have been isolated. For this reason, it is desirable that the most efficient possible method be utilized in mapping these mutants. To this end, I would like to propose a new method of mapping certain classes of mutants to given chromosomes utilizing monosomics of Zea mays generated by the r-Xl deficiency. This method requires only one tester strain and two crosses.

The r-Xl deficiency is an X-ray induced deficiency of the r locus which was originally obtained by L. J. Stadler. Satyanarayana (unpublished) noted that gametes carrying the r-Xl deficiency included large numbers of monosomics and trisomics. He generously provided the deficiency to our laboratory where it has been studied for several years. Some of the characteristics of the system are:

1. The deficiency is only female transmissible.
2. We have isolated three or more cytologically and genetically confirmed cases of monosomy for nine of the ten maize

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