

the trailing edge (C) of the polysome distribution shows even greater modification. Here there are fewer ribosomes in the polysomes and they are often widely separated. In this trailing edge, we would expect great heterogeneity, since it might contain viral RNA strands which do not have the full number of ribosomes. Our interest in this regard is the fact that we do not simply observe smaller clusters of ribosomes but rather frequently observe ribosomes which are widely separated from each other. An example is shown in Fig. 7d where five ribosomes can be seen attached presumably to an RNA strand, but they are separated by nearly 1000 Å. Several examples of this configuration have been seen in the trailing edge as well as other small clusters of ribosomes which may represent breakdown products associated with sample manipulation.

Because of the great difficulty of preparing samples and because artifacts may arise so easily, clear-cut conclusions cannot be drawn from this type of electron micrograph study. However, this information seems compatible with the type of scheme illustrated in Fig. 6a but not entirely compatible with the alternative interpretation in which the ribosome moves along the entire length of the polio RNA strand and repeatedly makes a copy of each protein for which the viral RNA has information. It is clear that the type of mechanism which we are suggesting for polio RNA is capable of a certain amount of control on the polysome level. Thus, for example, it may be possible to produce a small number of copies of the RNA polymerase protein while at the same time producing a large number of copies of the polio-coat protein. There are some indications that a control mechanism exists for polio virus production since approximately half the C¹⁴ amino acids in infected, actinomycin-D treated HeLa cells are found in virus-coat protein (9). Other systems of polycistronic reading mechanisms have also been discussed recently (13).

Our observations and interpretations may be regarded as tentative; they do not establish a mechanism for the polio virus polycistronic messenger RNA, but they may stimulate further experimentation (14).

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Nitrogen and Potassium Effect on the Color of Red Roses

Abstract. *Variable coloration was displayed by Rosa hybrida, var. Better Times, grown in solutions of various concentrations of nitrogen and potassium. As revealed by measuring the color in Hunter values, growth in high potassium and low nitrogen concentrations resulted in less reddish and more bluish roses, while cultivation in a low nitrogen concentration, irrespective of the N/K ratio, resulted in roses which were more lightly colored than those grown in high concentrations of nitrogen and potassium. The results were statistically significant at the 1 percent probability level.*

The chief pigment of red roses is the anthocyanin, cyanidin 3,5-diglucoside (1). The color of plant tissues pigmented with anthocyanins is affected by a number of factors, such as the concentration and association of these pigments with other substances, the pH, the ash content and colloidal state of the cell sap, the nature and concentration of other pigments, and so forth (2).

The nutritional state of the plant has also been connected with the color of anthocyanin-pigmented tissues (3). Red roses tend to turn paler owing to inadequate carbohydrate supplies (4), and an excess of potassium in relation to nitrogen in the growth medium imparts a bluish hue to the petals (5).

The object of this study was to assess the effect of certain levels of potassium and nitrogen available to the plant on the color of red roses, and to use instrumental methods of color evaluation rather than visual observation.

Nonrooted rose cuttings of *Rosa hybrida*, var. Better Times, were placed in moist sand and after rooting they were transferred to glazed crocks filled with No. 7 AGSAE Flint quartz sand. Growth was initiated in a standard Hoagland solution. The sand was then washed with distilled water and nutrient solutions representing four combinations of two concentrations of N and K were applied. The two concentrations of these elements differed by a factor of three (Table 1). The nutrient solutions were applied twice a day to the surface of the sand and the excess solution was collected in containers kept below the crocks. The nutrient solutions were made fresh weekly.

The roses were cut 4 days after opening and six petals were detached from the two outermost rows of each rose. The color of each petal was measured individually with the Gardner color difference meter. The petal was placed between the two plates of a petri dish (the smaller plate was blackened and inverted) with the centers of the light spot and the petal made to coincide. Evaluation of the color was made in terms of the Hunter values L, a_L, and b_L. The instrument was calibrated with standard tile B44, L 24.8, a_L +20.4, b_L = +6.0 (6).

This method of measuring the color of roses was considerably more sensitive than either the comparison of the absorption spectra of extracts and expressed sap of the petals or the comparison of the reflectance spectra of the petals by means of a Beckman DU spectrophotometer.

Table 1. Composition of nutrient solutions (grams/liter). In addition, the solutions contained magnesium sulfate, the iron salt of EDTA, and trace elements.

1N-1K	1N-3K	3N-1K	3N-3K
0.4723	Ca(NO ₃) ₂ · 4H ₂ O 0.4723	0.4723	0.4723
0.1726	NH ₄ H ₂ PO ₄ 0.1726	0.1726	0.1726
0.0160	NH ₄ NO ₃ 0.0160	1.0086	0.5436
0.6571	KNO ₃ 0.6571	0.6571	1.8196
0.1361	KH ₂ PO ₄ 0.1361	0.1361	0.1361
0.07455	KCl 1.3419	0.07455	0.4848

Table 2. Effect of nitrogen and potassium on the color values of rose petals. The two means connected by a curved line are not different at the 5-percent level of significance, while the third mean is different from the other two at the 1-percent level of significance. The three Hunter values have been converted mathematically to the Munsell system and the values are given in the characteristic notation (RP, red purple).

Treatment	Hunter system			Munsell system	
	L	a _L (mean)	b _L		
3N-3K	32.2	+ 41.9	- 2.7	6.5 RP	3.7/11.8
1N-1K	39.6	+ 40.0	- 3.1	6.0 RP	4.5/9.2
1N-3K	40.2	+ 32.3	- 6.5	3.5 RP	4.6/8.5

Only three of the four fertilizer treatments were used in the statistical evaluation of the color effect. No flowers were produced on the plants that were treated with the 3N-1K combination.

The means of the values from the Hunter color test on the petals were compared by Duncan's multiple range test (7) and are presented in Table 2, along with calculated (8) Munsell re-notations.

The lightness in color, of which L is a measure, seems to be affected more by the absolute quantity of N and K rather than the N/K ratio, since a large amount of N and K results in darker roses (that is, those having less white component). The positive a_L values indicate redness and since roses grown in 3N-3K and 1N-1K are significantly redder than those grown in 1N-3K, it can be inferred that an excess of K over N results in less red color. The negative b_L values indicate blueness and here again the significantly bluer roses resulting from growth in 1N-3K indicate that a large ratio of K over N favors the appearance of

blue color while it reduces redness. This result agrees with Twigg's observation that "blue" roses contained more potassium than red roses (5).

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1). In the present investigation, five inbred strains of mice (DBA/1J, C57BL/10J, A/J, C3H/HeJ, and BALB/cJ) and all 20 F₁ hybrids resulting from their intercrossing were used. The five inbred strains were chosen at random from those available in our colony. Twelve mice per genotype and sex were used (total $N = 12 \times 25 \times 2 = 600$). All animals were fed Purina mouse breeder chow, a diet containing 11 percent fat. When each mouse was sexually mature and about 4 months old, its blood was analyzed. Blood was drawn from the tail vein and centrifuged. Two microliters of serum were then pipetted and analyzed for cholesterol according to the fluorometric ultramicro-method developed by Webster (2).

Table 1 (a 5 × 5 diallel table) contains data for each of the 25 genotypes. Data for a given genotype are entered at the intersection of the maternal strain row and the paternal strain column. The leading diagonal of the table contains data for the five inbred strains. As the first step in determining the mode of inheritance of SCL, midparent values for each F₁ hybrid were computed and entered in Table 1. For example, the midparent value for F₁ (DBA ♀ × C57 ♂) was arrived at by averaging the SCL of DBA/1 ♀♀ and C57BL/10 ♂♂: $(91 + 133)/2 = 112$ mg percent. The mean absolute discrepancy between the thus computed midparent values and F₁ averages, $(♀ + ♂)/2$, was 5.7 mg percent (standard deviation, 4.01 mg percent), and the correlation between theoretical and observed values was 0.837 (18 degrees of freedom) and highly significant. If reciprocal crosses are pooled, midparent values and F₁ values become even more alike. For example, for F₁ (DBA ♀ × C57 ♂) and F₁ (C57 ♀ × DBA ♂) the pooled midparent value becomes 111.25 mg percent and the F₁ value becomes 112.75 mg percent. The second step was to compute the average for all parental scores and that for all F₁ hybrid scores. These two averages were 129.10 mg percent and 132.05 mg percent. The difference of 2.95 mg percent was clearly not significant. These preliminary analyses indicate intermediate rather than dominant or recessive inheritance of SCL. The F₁ hybrids resemble neither the parent with the higher nor the parent with the lower SCL, but fall midway between them; the parental average and the F₁ average do not differ. Such intermediate inheritance often is called "additive"

Additive Inheritance of Serum Cholesterol Level in Mice

Abstract. Serum cholesterol level (SCL) was measured in 600 mice belonging to five inbred strains of mice (DBA/1J, C57BL/10J, A/J, C3H/HeJ, and BALB/cJ) and all 20 F₁ hybrids resulting from the systematic crossing of the inbreds. Diet was kept constant and its effect on SCL was not evaluated. The data show that in mice, the mode of inheritance of SCL is neither dominant nor recessive, but is intermediate. A simple additive model accounts for the results: the SCL is a linear function of the SCL of the mother, the SCL of the sire, and the sex of the subject; the three factors do not interact.

In 1962 Bruell, Daroczy, and Hellerstein (1) reported that inbred strains of mice differ in their serum cholesterol level (SCL), and that the SCL is higher in males than in females. Here I present data which show that the mode of inheritance of SCL in mice is neither dominant nor recessive, but intermediate and additive. The SCL of F₁ hybrids can be predicted accurately by

adding half the SCL of one inbred parent to half the SCL of the other; and the correlation between these midparent values and observed F₁ values is highly significant.

A genetic diallel design was used. In this approach, data are obtained for N inbred strains and all $N(N-1)$ possible F₁ hybrids resulting from the systematic intercrossing of the inbreds (see Table