was determined (Fig. 1). These flower bud stages were: 2 and 1 day prior to anthesis; the day of anthesis; and 1, 2, and 3 days after anthesis. Pollen at each of these stages of development was used to hand pollinate 5 flowers on each of 4 plants. Fruit-set and number of seeds per fruit were determined.

Pollen stored in a covered Petri dish at room temperature produced successful pollinations for 2 days but no longer (Table 1). Pollen remained viable up to 10 days when refrigerated at 2-6°C and relative humidity of 40-50%, and up to 50 days at 2-6°C over $CaCl_2$. Open pollinated fruits had more seeds per fruit than emasculated, hand pollinated fruits on the same plant, which might be a factor in the lower seed production.

No successful pollinations were obtained using pollen taken from flower buds 2 days prior to anthesis or pollen from flowers 3 days after anthesis. Pollen collected from flowers the day of anthesis produced maximum fruit-set; that collected 1 day prior to, or 1 day after anthesis resulted in a reduction of fruit-set and seed production. Hirose (4) reported a low level of seed production in pepper using pollen 4 days after anthesis. He stated that temperature had an influence not only on pollen germination but also on pollen development.

Fig. 1. Pepper flower buds: left, 2 days prior to anthesis; center, 1 day prior to anthesis; and right, the day of anthesis.

Table 1. Effect of pollen storage on fruit-set and seed production in pepper, 1965.

Storage treatment	Storage period days	Fruit-set %	Seeds per fruit	Seeds per fruit OPF[a]
22-26C	1	25	84	170
50-70% R.H.	2	10	42	214
	4	0	0	190
	6	0	0	179
2-6C	1	55	104	192
40-50% R.H.	2	45	121	205
	4	50	97	201
	6	30	113	220
	8	15	78	175
	10	5	90	182
	12	0	0	202
2-6C	1	50	125	169
over $CaCl_2$	2	45	116	218
	4	60	128	131
	8	25	89	137
	10	20	104	168
	12	10	75	147
	50	5	88	221

[a] OPF — open pollinated field fruits.

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The Burgundy Sport: Further Evidence of the Chimeral Nature of Pigmented Grapefruits

By E. O. Olson[1], J. W. Cameron[2], and R. K. Soost[2]

Recently, we proposed (1) that the pink grapefruit varieties, Thompson and Foster, are periclinal chimeras, carrying factors for color in certain histogenic layers. Lycopene and other carotenes are the pigments involved (4). In the Thompson, histogenic Layer I (L-I) should carry the factor and Layer II (L-II) should not, since color is present in the juice vesicles but not in the rind and since nucellar seedlings, evidently derived from Layer II, show no red pigment in their fruits. Thompson was derived as a sport from the white Marsh grapefruit (6); the two varieties seem essentially identical except for fruit color.

In 1954, a new pigmented grapefruit called Burgundy was described (5). Its characters were further studied in 1959 (2). The color characters of Burgundy indicate that it, too, is a chimera. Our data from fruiting seedlings (apparently nucellar) of Burgundy support this theory.

[1] Agricultural Research Service, U. S. Department of Agriculture, Weslaco, Texas.
[2] University of California Citrus Research Center, Riverside, California.

Table 1. Color expression and possible genotypes of fruits of parental clones and nucellar seedlings of Burgundy and other grapefruit varieties.

Variety	Color of fruit tissues in: Parental clones: Juice vesicles	Color of fruit tissues in: Parental clones: Rind and septa[a]	Color of fruit tissues in: Nucellar seedlings: Year seed planted	Color of fruit tissues in: Nucellar seedlings: No. of individuals tested	Color of fruit tissues in: Nucellar seedlings: Juice vesicles, rind and septa	Possible genotypes of parental clones[b]: Juice vesicles (Largely L-I)	Possible genotypes of parental clones[b]: Hypoderm and mesoderm of rind and septa (Largely L-II)	Possible genotypes of parental clones[b]: Hypoderm and mesoderm of rind and septa (Partly L-III?)
Marsh[c]	white	white	1916	many	white	Rr	Rr	Rr
Thompson[c]	pink	white	1952[d]	8	white	rr	Rr	Rr?
Redblush[c]	red	red	1952	many	red	rr	rr	Rr?
Burgundy	red	white	1958	3	white	A r'r	Rr	Rr?
						B rr	Rr	rr

[a] Excluding epidermis.
[b] Where r and r' are alleles leading to pink or red color. The column for L-II also represents the genotype of the nucellus.
[c] Data from Cameron et al. (1).
[d] An additional seedling of Thompson, planted in 1959, gave white fruit in 1964 and 1965.

According to the record (5), Burgundy was discovered in Florida as a limb sport of Thompson. The flesh color is deep red, rather than the pink shade characteristic of Thompson. The rind, however, as in Thompson, shows no red. In both varieties the pigment is insoluble in the juice.

In 1958, seeds from Burgundy were planted by the U. S. Department of Agriculture at Weslaco, Texas, and three seedlings were obtained. These trees have the growth habit and leaf type of the Thompson and Marsh varieties, and all three trees resemble one another. They do not show genetic differences that would indicate sexual recombination. Moreover, a high incidence of nucellar seedlings may be expected from Burgundy, since Thompson and Marsh are highly nucellar. Thus, we conclude that the seedlings of Burgundy are nucellar.

One Burgundy seedling fruited in 1965 and the others in 1964 and 1965. All fruits were white, and seemed indistinguishable from Marsh fruits. Budwood from one of the Burgundy seedlings was propagated at Indio, California, and its fruits in 1964 and 1965 were white. Thus the color factor in Burgundy like that in Thompson was not transmitted by the asexual embryos. Table 1 lists the color characteristics and possible genotypes of Burgundy and its nucellar seedlings, compared to those of some related types previously studied (1).

The Burgundy apparently represents a change in a color factor which did not become part of histogenic Layer II. If we postulate that a recessive gene is responsible for the lycopene, at least two alternate genotypes could explain the Burgundy color. According to genotype A (Table 1), Layer II would carry *Rr*, as do Thompson and Marsh. In L-I, an *r* gene of Thompson would have mutated to an allele *r'*, such that *r'r* produces red flesh instead of pink. L-III could be unchanged. Since L-II carries an *R* (colorless) gene, nucellar seedlings would produce white fruit.

If genotype B is correct, the *rr* shown in L-III could have arisen by substitution of *rr* cells from L-I, in the parent Thompson plant. No new mutation would be involved; the change in flesh color from pink Thompson to red Burgundy might be an indirect effect of one histogenic layer upon another, as suggested earlier (1) for Foster. The system of genotypes in Table 1, which utilizes genotype B for the Burgundy, suggests that when a single histogenic layer is *rr*, the flesh is pink; when two (or more?) layers are *rr*, the flesh is red. However, if juice vesicles are made up of both L-I and L-II, varying *proportions* of cells from the two layers, in different varieties, could contribute to vesicle color.

In the rind, a thin L-III tissue layer which is *rr* might not be visible within a white L-II layer, since there is much influence of environment on amount and distribution of red rind pigment. In the last column of Table 1 the question marks indicate that genotypes of L-III are less certain than those of other layers.

In Texas, an additional nucellar seedling of Thompson, planted in 1959, produced white fruits in 1964 and 1965. This substantiates our earlier results (1).

The color factor is not necessarily a gene, rather than some other chromosome change, nor must it be recessive. Furr et al. (3) have obtained a few hybrids between pink or red grapefruit and red-fleshed pummelos; all show some red color in the flesh, which might result from homozygosity of a recessive gene.

Dr. Mortimer Cohen of the University of Florida has obtained a limb sport of Burgundy with red-rinded fruit (personal communication). If this change is part of the system outlined here, histogenic Layer II should now be carrying a color factor, and nucellar seedlings of this sport should show color in the fruits. This hypothesis will be tested when possible.

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