ratio. Differences in the time required to produce the color change were observed. Ten samples of the same variety (Dresden) showed color variations of essentially the same order of magnitude as those obtained with the different varieties. The only exception was a recently named strawberry, Fairland, which showed violet color only after 27 days' storage. Common varieties like Marshall, Blakemore, Dresden, Howard 17 and Culver all turned violet in one to five days. The minimum amount of glucose required to produce the violet color in the strawberries was found to depend largely on the presence of undissolved glucose. Violet color formation was observed only in those samples which contained glucose in a solid form. It is significant to note that all the violet material was always found associated with the undissolved glucose and that in those places where there was no solid glucose the strawberry preparation stayed red. Whether the glucose crystallized during storage or whether it was originally present as a solid did not seem to affect the color change. Another observation was that the time of separation of the glucose often does not coincide with the first appearance of the violet color.

As was expected, the anthocyanin pigment of strawberries plays a dominant role in the violet color development. When the anthocyanin chloride (*1*) (believed to be mainly pelargonidin 3-monoglucoside) is dissolved in Sorensen's sodium citrate-hydrochloric acid buffer of pH 3.4, or distilled water, and the solution is saturated with chemically pure D-glucose, and frozen at −23.3° C, a violet color is obtained. If similar solutions are allowed to evaporate at room temperature, the anthocyanin also turns violet. This residue is stable at room temperatures. The anthocyanin reverts to the red form when the frozen samples are thawed or when distilled water is added to the dry material.

These tests led to the conclusions that (1) the violet color is not due to possible impurities of the glucose used since c.p. D-glucose gives identical results; (2) low temperatures are not necessary for the formation of the violet color; and (3) the color change is reversible. The rate of color formation is influenced by the pigment concentration. Solutions containing 25% glucose showed violet spots after 10 days' storage at −23.3° C only if the anthocyanin concentration was 12 mg% or higher. The role of pH seems to be of minor importance. When the pH of frozen samples is lowered to pH 3.0, 2.5 and 1.8 a retarding affect with increasing acidity is noted. The air-dried, violet anthocyanin-glucose mixture turns red on heating. The transition temperature, which is not very sharp, is between 65° and 70° C for the pulverized material. Whether or not a loss of water occurred during the heating period was not determined.

Although a number of sugars were tested, only glucose gave a characteristic color change with the anthocyanin. Tests with sucrose, galactose, rhamnose, fructose, maltose, and α methyl glucoside were negative. Several of these sugars did not separate from solution under the conditions of the experiment and this may have caused the negative results. Inconclusive results were obtained with invert sugar. Arabinose seems to have a very slight effect.

Should it be found that the described reaction really is specific for pelargonidin 3-monoglucoside (or other glycosides of pelargonidin) it could well serve as a qualitative test for these compounds.

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The Effect of Pteroylglutamic Acid on the Aromatic Amino Acid Metabolism of Premature Infants[1]

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Scorbutic guinea pigs fed tyrosine (*4*) and prematurely born infants fed cow's milk mixtures of relatively high protein content (*3*) excrete large amounts of hydroxyphenyl derivatives in the urine. These can be decreased markedly by the administration of ascorbic acid (*3*, *4*). Woodruff and Darby (*5*) studied the effect of administration of PGA on the excretion of hydroxyphenyl derivatives by scorbutic guinea pigs and found that PGA repaired the defect produced by lack of ascorbic acid. Johnson and Dana (*2*) have reported that ascorbic acid produced significant increases in weight gain and leucocyte and normoblast count in PGA-deficient rats.

TABLE 1

EFFECT OF ORAL PGA ON "TYROSYL" EXCRETION

Infant B. L.—weight at birth: 2.15 kg			
Age in days	Weight in kg	Urine "tyrosyl"	
		mg/cc	mg/kg/24 hr
14	2.04	1.5	40
16	2.05	1.1	34
21	2.47	.4	18
29	3.98	5.2	289
30	2.95	5.4	357
31	PGA—5 mg started daily by gavage		
34	2.95	4.8	368
37	3.09	4.3	279
41	3.10	3.4	234
42	PGA—10 mg started daily by gavage		
44	3.26	3.1	267
46	3.40	1.4	112
50	3.52	0.2	17

In the present study, PGA was given to 10 premature infants fed cow's milk mixtures containing 6 g of protein and 120 cal per kg of body weight per day, but no ascorbic acid. Urine was collected for timed periods

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of approximately 24 hr and hydroxyphenyl derivatives were determined (*1*).

All ten infants excreted significant amounts of "tyrosyl" before administration of PGA. For one infant, A.L., the gavage feeding of 5 mg of PGA daily for seven

TABLE 2

EFFECT OF INTRAMUSCULAR PGA ON "TYROSOL" EXCRETION

Infant P—weight at birth: 0.97 kg			
Age in days	Weight in kg	Urine "tyrosyl"	
		mg/cc	mg/kg/24 hr
48	2.01	3.4	224
52	2.24	3.2	187
53	PGA—30 mg started daily by gavage		
57	2.27	4.4	227
61	2.35	3.2	273
61	PGA discontinued		
65	2.50	4.8	281
69	2.55	5.1	286
74	2.78	4.1	310
76	PGA—30 mg started daily by intramuscular injection		
78	2.92	0.2	10.0

TABLE 3

LACK OF EFFECT OF PGA ON "TYROSOL" EXCRETION

Infant B. P.—weight at birth: 2.19 kg			
Age in days	Weight in kg	Urine "tyrosyl"	
		mg/cc	mg/kg/24 hr
15	2.13	4.6	240
17	2.27	4.5	337
18	PGA—10 mg started daily by gavage		
20	2.30	3.0	291
23	2.50	4.4	290
27	2.64	4.8	324
31	2.75	5.1	380
32	PGA—20 mg started daily by gavage		
37	2.95	6.1	212
39	3.03	3.9	278
43	3.15	4.6	282
43	PGA—15 mg started daily intramuscularly		
48	3.32	4.4	242
48	PGA discontinued—ascorbic acid 100 mg intramuscularly		
52	3.43	0.4	33

days produced a striking decrease in "tyrosyl" excretion, which persisted as long as 13 days following cessation of PGA therapy. For his twin brother, B.L., gavage administration of 5 mg daily for 10 days was without effect, but 10 mg was effective after four days, (see Table 1). For infant P., the gavage feeding of 30 mg of PGA for eight days was ineffective, but the intramuscular injection of the same dose for two days led to a prompt drop in hydroxyphenyl excretion (see Table 2). For infant R., the administration of folic acid, 10 mg orally for 16 days, had a partial effect, but the administration of 30 mg intramuscularly for three days produced a striking effect. For the remaining six infants, PGA was ineffective whether given orally or by gavage in daily doses of 10–30 mg for periods of 8–15 days, or intramuscularly in daily doses of 15–30 mg for periods of 5–8 days. In five of the six infants for whom PGA was ineffective and who were given ascorbic acid, there was a prompt decrease in excretion of hydroxyphenyl derivatives. The findings for one infant in this group are given in Table 3.

Definition of the conditions under which pteroylglutamic acid is or is not effective in premature infants remains the subject for future studies. It is of interest that a difference dependent on the route of administration was the deciding factor in two of eight infants tested. The variability in response recalls a similar experience with liver extract noted in a previous study (*3*).

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A Method for Self-Control of Population Growth among Mammals Living in the Wild[1]

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It is a common observation that the numbers of most organisms in a reproducing population reach an equilibrium with the conditions of their environment. In approaching this upper limit of the logistic of the population, Pearl (*2*) has pointed out that "the rate of reproduction or fertility is negatively correlated with density of population." This is merely a statement of fact which prompts the study of the mode by which the reduction in contribution of progeny to the population occurs. Whatever the means of reduction, the result is that there are less adults to compete for the existing supply of food and harborage.

A recent note in *Science* by Mirone, *et al.* (*1*) suggests one possible way in which the increment to a population may be effectively reduced. They observed in mice that newborn young of mothers on a poor diet frequently fail to survive past the fourth day. From the fact that these same mothers may successfully rear adopted litters born to mothers on an adequate diet, they conclude that failure of the litters to survive is due to inadequate nutrition during gestation. This conclusion suggests that any environmental conditions which affect the maternal

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