Table 1. Distribution of *Glaucopsyche* eggs on the two types of inflorescences shown in Fig. 1.

Number of eggs	Number of inflorescences	
	No open flowers	Open flowers
0	43	120
1	53	8
2	14	2
3	9	0
4	3	0
5	1	0
6	1	0
7	0	0
8	1	0

(presumably last instar). Most larvae, however, were intermediate in size. Clearly, further damage would have been done if the inflorescences had been permitted to progress to seed set. We feel, however, that such damage would have been relatively minor since the ovaries were all well developed and were subject to little attack.

Eight inflorescences were destroyed in the course of the study, leaving 41 controls and 51 exposed to attack. A total of 111 eggs were laid on the exposed inflorescences, 2.18 per inflorescence. The 41 control inflorescences had a potential production of 967 mature flowers. This group actually produced 693, or 71.66 percent of potential. The experimental group had a potential production of 1433 mature flowers and actually produced 533, or 37.19 percent of potential. Of these mature flowers 138 were so badly damaged that they would have abscissed without setting seed, so that a more realistic estimate of realized potential in the experimental group is 395/1433, or 27.56 percent. Both experimental groups (with and without damage) are, of course, highly significantly different from the controls ($P \ll .01$).

A sample of 100 large inflorescences was taken on 15 July from the Crested Butte population of *L. amplus* on which *G. lygdamus* is rarely seen. This population of lupines is essentially continuous with that at Gothic, some 5 miles (8 km) away. Of a potential of 4169 flowers, 3149 (75.53 percent) were realized, and 3091 (74.14 percent) were judged sufficiently undamaged to set seed. Only 11 egg shells or larvae were found on these plants. In contrast, an additional sample of 100 large inflorescences from the Gothic population, where *G. lygdamus* was abundant, was censused on 16 July. Of a potential of 4277 flowers, 2434 (57.31 percent) matured. Of these 2152 (50.67 percent) matured and were judged sufficiently undamaged to set seed. On these inflorescences 126 *G. lygdamus* egg shells or larvae were found. The differences between the two areas are highly significant ($P \ll .01$).

The damage done to the Gothic population of *L. amplus* by this small butterfly is stunning. In 1968 nearly 50 percent of the potential seed production was destroyed by *G. lygdamus*, which has been abundant at Gothic in every season since 1960 except 1964–65 (when no observations were made). There is no reason to believe that the 1968 density was unusual. Presumably the lupines have been subject to a long-term attrition of their seed production. This has a drastic selective effect on the plant population. Lupines are dependent on having an abundance of seeds widely distributed in the soil since they germinate only upon disturbance and scarification.

We can guess at one selective response of the plant to *Glaucopsyche* attack—advancement of flowering time. The Gothic population of *L. amplus* seems to have been pushed to its earliest limit, as many examples of frost-killed and damaged inflorescences were observed this year. The butterflies oviposit strictly on the immature inflorescences (Table 1), indicating that plants on which flowers mature before the adult butterflies emerge, or early in the flight season, would be least subject to damage. There is no other obvious reason for the early flowering, as seed production is completed with more than a month of growing season remaining. At this time, other explanations cannot be excluded.

DENNIS E. BREEDLOVE
Department of Botany, Botanical Gardens, University of California, Berkeley 94720

PAUL R. EHRLICH
Department of Biological Sciences, Stanford University, Stanford, California 94305

References and Notes

1. For example D. Janzen, *Evolution* **20**, 249 (1966).
2. P. R. Ehrlich and P. H. Raven, *ibid.* **18**, 586 (1965); P. R. Ehrlich, *Oreg. State Univ. Biol. Colloq.*, in press.
3. For example, C. H. Muller, *Bull. Torrey Bot. Club* **93**, 332, 351 (1966); *Z. Pflanzenkr. Pflanzenpathol., Pflanzenschutz* **74**, 333 (1967); and personal communication: Professor Muller considers the toxic compounds of plants to be "primarily metabolic wastes."
4. W B. Kemp, *J. Heredity* **28**, 328 (1937).
5. We thank J. A. Hendrickson, R. W. Holm, and P. H. Raven, for criticizing our manuscript. Supported by NSF grant GB-5645 and by a Ford Foundation grant.

5 August 1968

C'3 Synthesis in the Human Fetus and Lack of Transplacental Passage

Abstract. *Allotypic differences in the third component of complement between mothers and their newborns provided evidence for synthesis of this complement component by the fetus. There was no indication that this protein traversed the placenta. The known low level of C'3 in the neonate was confirmed, and the maternal concentration was found to be significantly elevated.*

The third component of complement, C'3 or β_{1c}-globulin, has been shown to be present in human fetal blood as early as 9 weeks of age (*1*), but it is not known whether the protein is synthesized by the mother or the fetus. The demonstration of differences in phenotype between the mother's and her newborn's plasma proteins has provided evidence for human fetal synthesis of haptoglobin (*2*), transferrin (*3*), and Gc-globulin (*4*). With the recognition of genetically controlled polymorphism in C'3 (*5*), it has become possible to examine in similar fashion the question of fetal synthesis and transplacental passage of C'3 in man.

The C'3 polymorphic system in man is composed of two common codominant alleles, designated F (fast) and S (slow). Approximately 97 percent of 200 people typed to date are either FF, FS, or SS. In the remaining 3 percent, certain rare alleles have been found. We have named the rare allotypes by their positions relative to the S allotype. The fastest and slowest rare allotypes thus far detected are F_1 and S_1, and we have found three other rare allotypes similarly named $F_{0.8}$, $F_{0.5}$, and $S_{0.6}$ (Fig. 1).

Typing of C'3 was performed on 25 maternal and cord paired serums. Samples were stored for a maximum of 24 hours at 4°C before analysis by prolonged agarose electrophoresis. Total

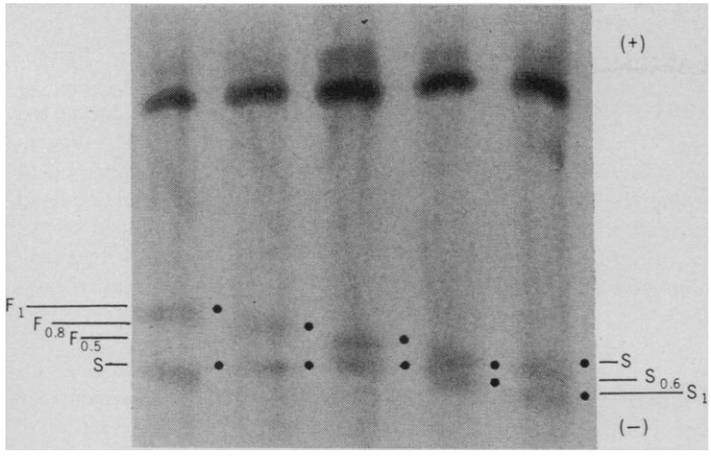
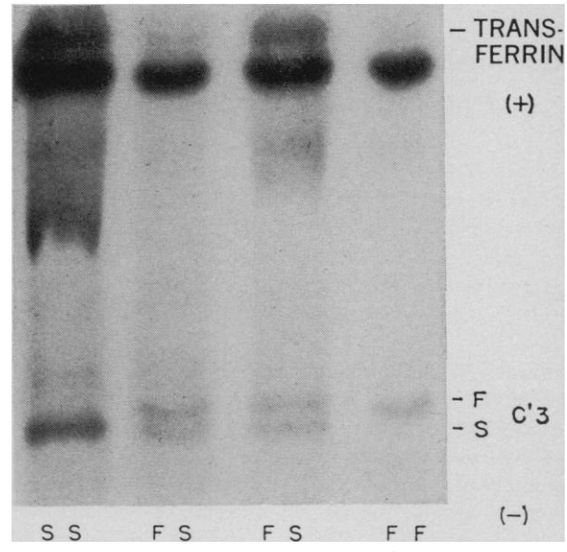


Fig. 1 (above). Prolonged agarose electrophoresis patterns of serums of individuals heterozygous for the *S* gene and rare alleles of C'3. From left to right, the types (bands are marked with solid circle) are F_1S, $F_{0.8}S$, $F_{0.5}S$, $SS_{0.6}$, and SS_1. Fig. 2 (right). Prolonged agarose electrophoresis of maternal and cord serums. Two pairs are pictured. From left to right, the serums are mother and child (pair 11) and mother and child (pair 23).



concentration of C'3 in all samples was determined by Laurell's electroimmunochemical method (*6*), or by a nephelometric method (*7*) with the use of monospecific rabbit or goat antiserums to preparations of human C'3 made in this laboratory by the method of Nilsson and Müller-Eberhard (*8*).

Types and concentrations of C'3 in the 25 paired serums are presented in Table 1. Eight pairs were of different types and these are indicated by asterisks. Most of the discrepancies involved the common allotypes F and S, but two involved a rare allotype $S_{0.6}$. Another rare allotype, $F_{0.5}$, was found in both maternal and newborn serums in one pair. The high incidence of rare allotypes in this small number of serums may indicate that we have underestimated their incidence in the general population. The typing of larger numbers of random serums is currently in progress.

Table 1. Type and concentration of C'3 in maternal-cord paired serums. Pairs of different types are marked with asterisks.

Pair	Maternal		Cord	
	Type	C'3 (mg/100 ml)	Type	C'3 (mg/100 ml)
1	$F_{0.5}S$	191	$F_{0.5}S$	65
2	SS	200	SS	89
3*	$FS_{0.6}$	186	FS	121
4	SS	139	SS	82
5	SS	204	SS	118
6	SS	160	SS	71
7	SS	171	SS	71
8	SS	150	SS	82
9	SS	150	SS	100
10	SS	233	SS	114
11*	SS	171	FS	121
12*	FS	171	FF	64
13	SS	107	SS	60
14*	FS	191	FF	86
15*	SS	208	FS	82
16	SS	175	SS	132
17	SS	197	SS	71
18	FS	215	FS	96
19	SS	154	SS	93
20*	SS	150	$SS_{0.6}$	107
21*	FS	186	SS	68
22	FS	164	FS	56
23*	FS	160	FF	82
24	SS	225	SS	136
25	SS	200	SS	52
Mean (± S.E.)		178.3 ± 5.84		88.8 ± 4.86

Agarose electrophoretic analyses of two of the discrepant pairs are shown in Fig. 2. It will be noted that there is no evidence of the C'3 phenotype of the mother in her newborn's serum nor evidence of the C'3 phenotype of the fetus in its maternal serum.

These studies indicate that C'3 is synthesized by the human fetus and that there is no detectable transplacental passage of this protein. Thus, the heterozygous deficiency state of C'3, described elsewhere (*10*), and the postulated homozygous deficiency state should be detectable at birth, if the homozygous state is not lethal. In addition to confirming the previously demonstrated lower level of C'3 at birth as compared with later life, we have noted a significantly elevated concentration in maternal serum at the time of parturition.

RICHARD P. PROPP
CHESTER A. ALPER
Blood Grouping Laboratory and
Department of Medicine,
Children's Hospital Medical Center,
Boston, Massachusetts

References and Notes

1. M. Adinolfi and B. Gardner, *Acta Paed. Scand.* **56**, 450 (1967).
2. F. Galatius-Jensen, *Proc. 6th Eur. Congr. Haemat. Copenhagen* (Karger, Basel, 1957), p. 909; D. M. Kahlich-Koenner and G. Weippl, *Wien. Klin. Wochschr.* **72**, 674 (1960); A. R. Rausen, P. S. Gerald, L. K. Diamond, *Nature* **191**, 717 (1961).
3. A. R. Rausen, P. S. Gerald, L. K. Diamond, *Nature* **192**, 182 (1961).
4. J. Hirschfeld and N. O. Lunell, *ibid.* **196**, 1220 (1962).
5. C. A. Alper and R. P. Propp, *J. Clin. Invest.* **47**, 2181 (1968).
6. C.-B. Laurell, *Anal. Biochem.* **5**, 45 (1966).
7. A. A. Boyden, E. T. Bolton, D. G. Gemeroy, *J. Immunol.* **57**, 211 (1947).
8. U. R. Nilsson and H. J. Müller-Eberhard, *J. Exp. Med.* **122**, 277 (1965).
9. P. F. Kohler, *Fed. Proc.* **27**, 491 (1968); P. Fireman, D. A. Zuckowski, P. A. Taylor, *Program and Abstracts* [28th annual meeting, Soc. Pediat. Res., Atlantic City, N.J. (1968)], p. 136.
10. C. A. Alper, R. P. Propp, R. B. Johnston, Jr., F. S. Rosen, *J. Immunol.* **101**, 816 (1968); C. A. Alper, R. P. Propp, M. R. Klemperer, F. S. Rosen, in preparation.
11. Supported by PHS grant HD 02723. We thank Dr. Irving Umansky, Miss Sandra Carlin, and Mrs. Lorraine Gathman for providing the paired maternal-cord serums, Dr. Louis K. Diamond for critically reading the manuscript, and Miss Lillian Watson for expert technical help. R.P.P. is a special fellow, National Institute of Arthritis and Metabolic Diseases.

21 August 1968