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THE GENETIC STRUCTURE OF A CYCLICAL PARTHENOGEN

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Cyclical parthenogenesis is a mode of reproduction largely restricted to parasitic trematodes of the order Digenea, rotifers, cladocerans, aphids, gall wasps (Cynipidae), and some gall midges (Cecidomyiidae). In these organisms, unisexual propagation via eggs is periodically interrupted by a bisexual phase. In most cases parthenogenesis is not truly cyclical in that the length of the unisexual phase is a function of environmental circumstances and not of an inherent predetermined program. Nonetheless, such organisms should be considered distinct from facultative parthenogens. The cytogenetic events promoting unisexual and bisexual propagation in cyclical parthenogens are fundamentally different; males are parthenogenetically produced prior to the bisexual phase, and the mode of reproduction is not simply a function of the availability of males.

Much has been made of the evolutionary value of heterogonic life cycles (White, 1973; Williams, 1975; Maynard Smith, 1978). By altering unisexual and bisexual modes of reproduction individuals are thought to combine the short-term advantage of a rapid replication of their genome with the long-term advantage of recombination. Theoretically, parthenogenesis should allow rapid and efficient selection for highly fit linkage groups, and a periodic phase of recombination should facilitate the creation of coadapted gene complexes. This does not mean that populations of cyclical parthenogens will rapidly approach genetic homogeneity. Both segregation of heterotic linkage groups and shifting selection pressures in temporally and spatially variable environments will result in the maintenance

of genetic variability, as will the periodic release of hidden genetic variance that must accumulate in a clonal population subject to mutation and selection (Lynch and Gabriel, 1983). Nonetheless, the above arguments imply that the cyclically parthenogenetic mode of reproduction should provide an exceptionally effective mechanism for coping with environmental uncertainty.

Why then is cyclical parthenogenesis so rare? White's (1973) suggestion that the evolution of a reliable mechanism to support two radically different reproductive modes would require a cytogenetic tour-de-force certainly warrants consideration, particularly when one considers how common obligate parthenogenesis is relative to the cyclical mode. However, before resorting to an explanation based on cytological limitations, it is worth considering whether the supposed ecological and genetic advantages of cyclical parthenogenesis are indeed fulfilled in natural populations.

Ecological genetic analyses of cyclical parthenogens are rare. Although valuable genetic observations have been obtained with rotifers (King, 1977, 1980), aphids (Wool et al., 1978; Suomalainen et al., 1980; Tomiuk and Wöhrmann, 1980) and cladocerans (Hebert, 1978, 1980; Young, 1979*a*, 1979*b*), for the most part these studies are limited in the amount of ecological information that they convey and/or are short-term in duration. Validation of the assumptions in my second paragraph must involve long-term investigations of the genetic dynamics of populations and their association with the selective process. Here I report on the first five years of a continuous genetic

analysis of a population of the cyclically parthenogenetic cladoceran, *Daphnia pulex*.

Background

Both electrophoretic and biometric surveys have revealed that at least three reproductively isolated groups of clones morphologically referable to *Daphnia pulex* (in the broad sense, Dodson, 1981) coexist in Busey Pond, Illinois (Lynch, 1983). The pond is an ephemeral, woodland oxbow that has been permanently separated from its parent stream for more than 50 years. Of the three clonal groups, one (Group A) is entirely composed of obligate parthenogens almost all of which are identical at the electrophoretic level. Groups B and C are comprised of legitimate cyclical parthenogens, the former being much rarer than the latter. (A fourth and exceedingly rare group, D, previously reported in Lynch [1983], is now believed to be an exceptionally large *Ceriodaphnia* species that was accidentally loaded onto our gels.) In all cases, parthenogenesis is functionally equivalent to apomixis; i.e., there is no evidence of recombination. In the first three years of this study, 1978–1980, a consistent succession of clonal groups was observed in this pond, Group A individuals dominating the population in the spring and Group C going to near fixation in the summer (Lynch, 1983). This sequence of events was altered in 1981 and 1982 when Group C consistently comprised more than 90% of the *Daphnia* population. The analyses in this paper are applied solely to this clonal group.

Several electrophoretic criteria are used to routinely distinguish Group C individuals from the remaining clonal groups in this pond (Lynch, 1983). For example, obligately parthenogenetic Group A is fixed homozygous for the *M* allele at the *Alk-2* locus and fixed heterozygous *FM* at the *Got* locus, while Group C is fixed for the *F* and *M* alleles at these same two loci. The clonal groups also differ with respect to body size and reproductive

characteristics and the timing of resting egg formation (Lynch, 1983). Moreover, of the three clonal groups in Busey Pond, only Group C lacks the ability to make a rapid transition to a synthetic laboratory medium (Lynch, pers. observ.). Based on both female and male morphology, Group C individuals consistently meet the narrow criteria for *Daphnia pulex* suggested by Hrbáček (1959) and Brandlova et al. (1972).

At 1–3 week intervals, up to 300 random individuals from this population have been assayed at multiple structural gene loci by horizontal starch gel electrophoresis. In addition, partial life table analyses have been frequently determined for clones randomly sampled from the pond in order to identify potential selective differences between electrophoretic phenotypes. Since the details of the methods have been previously described (Lynch, 1983), I will proceed directly to the results.

RESULTS

Population Structure and Dynamics

The Group C population is oscillatory in nature with the only common trend between years being a tendency towards reduced numbers in June and/or July (Fig. 1). Part of this irregularity results from the unpredictability of the timing of drying of the pond, but seasonal changes in food availability and the density of two important predators (*Ambystoma* and *Chaoborus*) are important as well (Lynch, unpubl.). The population is always dominated by juvenile and parthenogenetically reproducing females. Males are rarer but are present on most dates, their highest densities tending to coincide with periods of high total population size. Ephippial (sexual) females are generally very rare, even compared to males, and also tend to be concentrated during periods of high total population size. There appears, however, to be no set period of sexual reproduction in this population, and it seems likely that at least a small

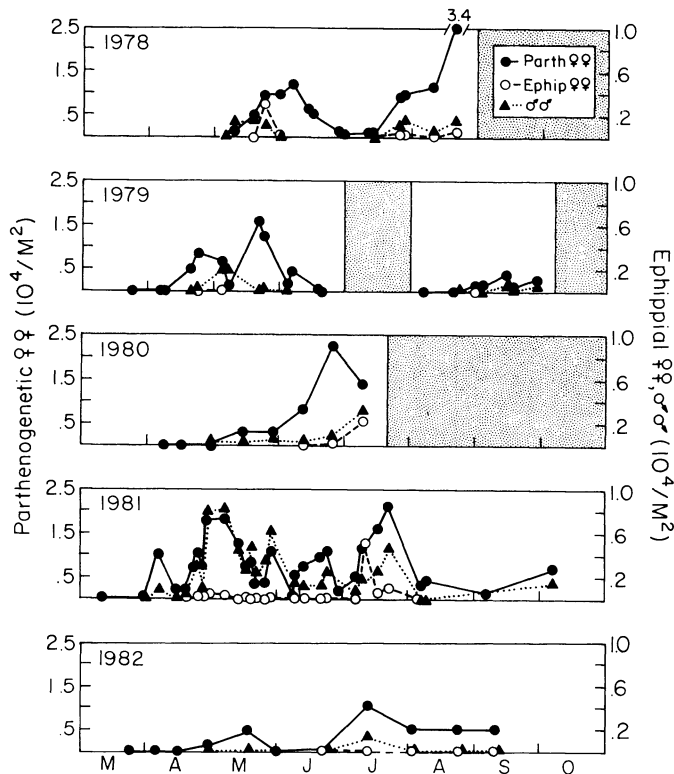


FIG. 1. Seasonal variation in the densities of parthenogenetic, male and ephippial Group C *Daphnia pulex* from Busey Pond. Stippled areas denote periods when the pond was dry.

amount of mating may occur during most periods.

Genetic Structure

Of the five structural gene loci that are routinely analyzed in the Busey Pond population (*Pgm*, *Pgi*, *Alk-2*, *Got*, and *Pept-2*), only *Pgm* and *Pept-2* (not surveyed until 1980) are polymorphic in Group C. Although both polymorphisms have been maintained throughout this study (Fig. 2, Table 1), significant temporal changes in both allele and genotype frequencies have occurred. A comparison of pooled samples from the first and second halves of each year by the *G*-test (Sokal and Rohlf, 1981) reveals significant within-year changes in genotype frequencies in five of eight cases, and completely pooled yearly samples differ significantly from each other in 10 of 13 cases (Table 1). Compared to the changes

within a growing season, the differences between the end of one year and the first half of the next tend to be less significant or even contrary to the within-year trends. The picture one gets at the *Pgm* locus is that of a loose ratchet—the *F* allele consistently advancing each year during the wet season, but then sliding back slightly at the onset of the next year. The situation is more complex at the *Pept-2* locus; the *S* allele dramatically increased within 1980, but declined in 1981 and 1982.

A statistical comparison of genotype frequencies of individual dates with Hardy-Weinberg expectations was frequently thwarted by a combination of small sample size and extreme gene frequencies resulting in expected frequencies of the rare homozygote < 5 . However, of the 23 *G*-tests that could be performed on the *Pgm* locus, 10 revealed significant deviations from Hardy-Wein-

TABLE 1. *Pgm* and *Pept-2* genotype and gene frequencies for pooled data from first (E) and second (L) halves of the cumulative time that *Daphnia* were present in the pond and for the entirety of each year (T). SE is the standard error of allele frequency, $(pq/2N)^{.5}$. *G* (distributed approximately as χ^2 , Sokal and Rohlf, 1981) is presented for tests of the homogeneity of E and L samples within years, of L with the E sample in the following year, and of T samples between years. * and ** denote differences significant at the .05 and .01 levels.

		Genotype frequency			Allele frequency			<i>G</i>			
		<i>MM</i>	<i>FM</i>	<i>FF</i>	<i>M</i>	<i>F</i>	SE	<i>N</i>	E vs. L	L vs. E, next yr.	Between yrs.
<i>Pgm</i>											
1978	E	.773	.142	.085	.844	.156	.019	176	5.993*	1.042	79-2.209
	L	.713	.230	.057	.828	.172	.014	352			80-8.428*
	T	.733	.201	.066	.834	.166	.011	528			81-31.327**
											82-93.860**
1979	E	.765	.190	.045	.860	.140	.018	179	1.525	.027	80-16.402**
	L	.782	.151	.067	.858	.142	.015	284			81-50.869**
	T	.775	.166	.059	.858	.142	.011	463			82-118.072**
1980	E	.791	.209	.000	.896	.104	.026	67	8.287*	.421	81-2.916
	L	.606	.314	.080	.763	.237	.020	226			82-34.539**
	T	.648	.290	.062	.793	.207	.017	293			
1981	E	.621	.294	.085	.768	.232	.008	1,314	3.217	12.632**	82-57.427**
	L	.584	.329	.087	.748	.252	.011	844			
	T	.606	.308	.086	.760	.240	.007	2,158			
1982	E	.487	.374	.139	.674	.326	.017	374	2.310		
	L	.481	.345	.174	.654	.346	.013	638			
	T	.483	.356	.161	.661	.339	.011	1,012			
		<i>MM</i>	<i>MS</i>	<i>SS</i>	<i>M</i>	<i>S</i>	SE				
<i>Pept-2</i>											
1980	E	.985	.015	.000	.992	.008	.008	66	26.284**	6.188*	81-14.337**
	L	.741	.236	.023	.859	.141	.017	220			82-5.407
	T	.797	.185	.018	.890	.110	.013	286			
1981	E	.660	.301	.039	.810	.190	.008	1,314	16.312**	3.796	82-113.712**
	L	.741	.233	.026	.858	.142	.009	842			
	T	.692	.274	.034	.829	.171	.006	2,156			
1982	E	.754	.236	.010	.872	.128	.012	394	51.294**		
	L	.917	.081	.002	.957	.043	.006	638			
	T	.855	.140	.005	.925	.075	.006	1,032			

berg frequencies, all due to heterozygote deficiency (Table 2). The positive values for F_{IT} (Wright, 1965) on 35 of 44 dates (Table 2) indicate that heterozygotes were generally deficient at this locus.

Few *G*-tests could be performed on the *Pept-2* locus because of the extreme rarity of the *S* allele. No significant differences from Hardy-Weinberg expectations were detected, but there is some hint of a heterozygote deficiency at this locus as well. While only 11 of the 27 *F*-statistics for *Pept-2* were positive, one of them was significant, and six were more than a standard error from zero; none of

the negative values were significant and only four were more than a standard error from zero.

Assortative mating hardly seems responsible for the slight heterozygote deficiencies at these loci considering the frequent between-year increases in heterozygosity observed in this study (Table 1). A more likely explanation is that a Wahlund effect is involved—several bouts of mating within a year (Fig. 1), but at slightly different gene frequencies (Fig. 2), followed by the simultaneous hatching of most ephippia at the onset of the next season would generate

TABLE 2. Frequencies for *Pgm/Pept-2* composite genotypes for Group C parthenogenetic females. *Pept-2* was not assayed in 1978 and 1979. *G*-tests for agreement with Hardy-Weinberg expectations at single loci were employed only when expected frequencies for each of the genotypes ≥ 5 ; * and ** denote differences significant at the .05 and .01 levels. $F_{IT} = 1 - (\text{obs. hets./exp. hets.}) \pm 2 \text{ SE}$ is also given for the individual loci, the standard error being estimated following Rasmussen (1964). In *A* (as defined in the text) and its associated *G* statistic are given as measures of interlocus association. Genotypes grouped under "others" were not used in the statistical analyses. Underlined dates represent the last samples included in the pooled E samples (Table 1) for their respective years.

		Pgm/Pept-2 genotype frequencies-parthenogenetic females										Pgm		Pept-2		ln <i>A</i>	<i>G</i>				
		FF/ MM	FF/ MS	FF/ SS	FM/ MM	FM/ MS	FM/ SS	MM/ MM	MM/ MS	MM/ SS	Other	<i>N</i>	<i>G</i>	F_{IT}	<i>G</i>			F_{IT}			
1978	18 MY										15	15	—	—							
	31 MY	4									21	25	—	—							
	13 JN	3			10						21	34	—	-.088 ± .372							
	29 JN	8			15						79	102	—	.429 ± .246*							
	13 JL	7			12						75	94	—	.463 ± .263*							
	27 JL	6			36						65	107	.115	.032 ± .198							
	10 AU	7			28						64	99	—	.154 ± .222							
	21 AU				5						47	52	—	-.053 ± .379							
1979	6 AP	1			4						23	28	—	.252 ± .536							
	20 AP	5			24						71	100	—	.150 ± .232							
	4 MY	1			3						13	17	—	.296 ± .617							
	21 MY	1			3						30	34	—	.356 ± .583							
	3 JN	2			10						75	87	—	.219 ± .333							
	18 JN	7			12						90	109	—	.474 ± .262*							
	14 SP	1			7						7	15	—	-.111 ± .531							
	28 SP	9			14						50	73	—	.439 ± .337*							
1980	30 AP				4						9	13	—	-.180 ± .695	—	—	—				
	14 MY				3						19	1	1	-.096 ± .539	—	—	—				
	28 MY				6						24	30	—	-.111 ± .469	—	—	—				
	12 JN	9	1		17	7					45	3	4	86	2.951	.191 ± .226	—	-.073 ± .294	1.821	—	
	25 JN	3	1		15	10	1				36	14	2	82	—	.034 ± .228	—	.006 ± .222	.539	1.072	
	9 JL	2	2		12	3	1				24	11	1	2	58	—	.134 ± .286	—	.028 ± .277	-.606	—
1981	31 MR	17	6	2	54	26	3	125	42	8				283	8.934**	.184 ± .129*	2.667	.102 ± .132	.360	1.454	
	14 AP	7	4		15	18		59	26	3				132	7.041**	.241 ± .197*	1.830	-.116 ± .190	1.002	5.687*	
	28 AP	9	1	1	21	12	1	59	21	4				129	5.905*	.224 ± .196*	—	.099 ± .195	.473	1.157	
	7 MY	1			10	1	3	23	10	1				49	—	-.047 ± .306	—	.291 ± .330	-1.470	—	
	12 MY	6	4		25	13	3	52	22	3				128	1.680	.117 ± .188	.251	.044 ± .185	.206	.221	
	27 MY	6	5		24	7		46	30	2				120	6.758**	.250 ± .203*	—	-.128 ± .203	-.805	2.854	
	30 MY	5	6		27	5		38	16	1				98	2.916	.183 ± .212	—	-.093 ± .231	-.821	2.197	

TABLE 2. Continued.

	Pgm/Pept-2 genotype frequencies-parthenogenetic females												Pgm			Pept-2		
	FF/		FF/		FM/		FM/		MM/		MM/		G	F _r	G	F _r	ln A	G
	MS	SS	MM	MS	SS	MM	MS	SS	MM	MS	SS	Other						
8 JUN	2	4	1	19	9	2	50	31	1	119	—	.164 ± .209	.864	—	-.085 ± .195	-.269	.351	
20 JUN	9	5	2	27	13	3	46	20	3	128	4.368*	.189 ± .184*	1.362	—	-.108 ± .191	.102	.046	
23 JUN	6	3	29	15	1	50	21	3	3	128	.383	.052 ± .182	—	—	-.015 ± .180	.208	.243	
6 JUL	6	3	1	27	7	1	59	23	1	128	3.666	.178 ± .195	—	—	-.042 ± .190	-.408	.719	
21 JUL	5	1	1	33	11	1	37	7	2	98	.819	-.091 ± .207	—	—	.185 ± .253	.566	1.114	
22 JUL	8	3	21	10	6	31	8	1	88	88	.257	.056 ± .216	—	—	.251 ± .244*	.613	1.192	
6 AU	9	2	28	14	3	72	21	1	150	150	2.729	.134 ± .177	—	—	.033 ± .173	.538	1.672	
19 AU	2	14	8	1	66	21	66	21	112	112	—	.027 ± .202	—	—	-.088 ± .218	.586	1.214	
6 SP	9	6	19	3	58	16	44	4	1	116	21.065**	.450 ± .198*	—	—	.113 ± .219	-.558	—	
6 OC	14	3	62	8	44	21	44	21	152	152	.120	-.023 ± .163	—	—	-.120 ± .208	-1.308	8.781**	
1982	29	AP	15	7	2	51	15	76	26	2	20	6.058*	—	—	-.033 ± .144	-.151	.164	
17	MY	12	4	30	13	43	8	43	8	110	1.826	.130 ± .194	—	—	-.125 ± .240	.846	2.833	
21	JN	9	3	28	3	22	5	22	5	70	.356	.072 ± .240	—	—	-.079 ± .309	-.752	—	
12	JL	17	1	27	8	48	3	48	3	104	6.421*	.251 ± .197*	—	—	-.056 ± .258	1.556	—	
2	AU	21	5	59	9	66	5	66	5	165	1.787	.110 ± .157	—	—	-.054 ± .203	.700	1.545	
23	AU	34	4	63	5	102	5	102	5	214	17.674**	.288 ± .188*	—	—	-.082 ± .226	.482	—	
10	SP	28	1	46	3	74	3	74	3	155	13.792**	.300 ± .160*	—	—	-.004 ± .173	.475	—	

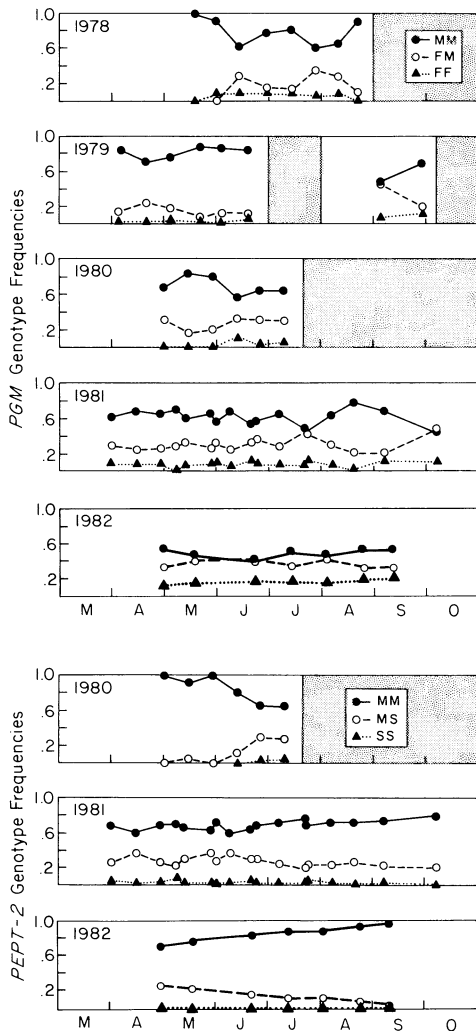


FIG. 2. Genotypic frequencies at the *Pgm* and *Pept-2* loci for Group C parthenogenetic females (plotted only for dates on which $N \geq 10$). *Pept-2* was not assayed in 1978 and 1979.

an excess of homozygotes. Any hatching of ephippia that had been deposited for more than one year would have a similar effect.

The different temporal dynamics of these two polymorphic loci provides some indication that they are not mutually constrained by linkage. However, formal estimates of linkage disequilibrium based on zygote frequencies require that mating is random and genotypes at

both loci are in Hardy-Weinberg equilibrium (Hill, 1974; Hedrick et al., 1978), assumptions that appear to be violated in this population. It is only possible to measure the association between genotypes at different loci. Such association between two loci will arise in the absence of gametic phase disequilibrium and continuously develop over the growing season of a clonal population when selection acts consistently on the different composite genotypes (Annest and Templeton, 1978). Because of the rarity of several of the nine *Pgm/Pept-2* genotypes in Table 2, I chose an index of association similar to that introduced by Young (1979a) which only depends on the frequencies of the four most common genotypes,

$$\ln A = \ln \left[\frac{p(MM/MM) \cdot p(FM/MS)}{p(FM/MM) \cdot p(MM/MS)} \right].$$

The expected value of $\ln A$ is 0 when the zygotic classes at the two loci are randomly associated, and the significance of any deviation from 0 can be determined by a *G*-test of independence (Sokal and Rohlf, 1981). For the Busey Group C population there was no consistent temporal pattern for $\ln A$, the sign frequently changing from date to date, but on two occasions in 1981 it was significantly different from zero, both times in a different direction (Table 2). Thus, although it appears unlikely that any single *Pgm/Pept-2* composite genotype is continuously favored in this population, differential expansion of composite genotypes may occur.

Based on these two polymorphic loci, it is clear that at least nine distinct genotypes of Group C *Daphnia* coexist in Busey Pond. However, this estimate is hardly indicative of the amount of genetic diversity that resides in this population. On October 6, 1981 along with the routine electrophoretic survey of this population, leucine aminopeptidase was assayed. Two loci were scorable, one of which (*Lap-2*) was polymorphic for two alleles. Not only were the *Lap-2* genotype

TABLE 3. Frequencies for *Pgm/Pept-2* composite genotypes for Group C males and ephippial females. *Pept-2* was not analyzed in 1978, nor were males. *G*-tests were used to compare male ($\delta\delta$) and ephippial female ($e\varphi\varphi$) genotype distributions and to compare each of these groups with the parthenogenetic female ($p\varphi\varphi$) distributions in Table 2 only on dates when observed and expected frequencies of two or more composite genotypes were ≥ 5 ; * and ** denote significance at the .05 and .01 levels.

		<i>Pgm/Pept-2</i> genotype frequencies-sexual individuals										<i>G, d.f.</i>		
		<i>FF/</i> <i>MM</i>	<i>FF/</i> <i>MS</i>	<i>FF/</i> <i>SS</i>	<i>FM/</i> <i>MM</i>	<i>FM/</i> <i>MS</i>	<i>FM/</i> <i>SS</i>	<i>MM/</i> <i>MM</i>	<i>MM/</i> <i>MS</i>	<i>MM/</i> <i>SS</i>	Other	<i>N</i>	<i>e\varphi\varphi</i> vs. $\delta\delta$	<i>p\varphi\varphi</i>
1978	27 JL	$\varphi\varphi$	3					9				16	28	—
	21 AU	$\varphi\varphi$						12				36	48	—
1980	12 JN	$\delta\delta$	2					3				10	15	—
	25 JN	$\delta\delta$	2	3				3	4			12	28	—
	9 JL	$\varphi\varphi$		2				14	3	2		28	8	3
		$\delta\delta$	1					11	3	2		8	3	2
1981	14 AP	$\delta\delta$										13	13	—
	28 AP	$\varphi\varphi$										18	4	2
		$\delta\delta$						2		1		24	8	1
	12 MY	$\delta\delta$	2	1				9	4			20	14	1
	27 MY	$\varphi\varphi$						6				8	5	1
		$\delta\delta$						5				18	11	2
	8 JN	$\delta\delta$							1			17	1	1
	23 JN	$\varphi\varphi$	2					5	1			4	4	
		$\delta\delta$	1					4	4			18	6	
	6 JL	$\varphi\varphi$	5	1				9	1			9	6	
		$\delta\delta$	2					5				7	2	
	21 JL	$\varphi\varphi$						6	1			6	3	
		$\delta\delta$	1					4	2			7	4	
	6 OC	$\varphi\varphi$	5	2				13	4			13	2	
		$\delta\delta$	2	2				14	2			15	2	
1982	17 MY	$\varphi\varphi$	1					13	3	1		12	7	
	12 JL	$\varphi\varphi$	6					9	6			16	2	
		$\delta\delta$						3	3			13		
	2 AU	$\delta\delta$	1					5				19		
	23 AU	$\varphi\varphi$	3					2				4		
		$\delta\delta$	2					4				4		

frequencies consistent with Hardy-Weinberg expectations ($G = 2.815$, $d.f. = 1$, NS), but the genotypes were approximately independent of those at the *Pgm* ($G = 2.471$, $d.f. = 2$, NS) and *Pept-2* ($G = 1.604$, $d.f. = 2$, NS) loci. A more thorough isozyme survey of 31 Group C clones collected during 1981 yielded one additional monomorphic locus (*Pept-1*) and seven more polymorphic loci: *Alk-1*, *Cat*, and *Sdh*—two alleles each, *Est*, *Mdh*, and *Me*—three alleles each, and *Aldox*—four alleles. Each of the 31 clones was electrophoretically unique.

There is little doubt that this population contains at least a few thousand clones at any one time. The minimum number of hatching Group C ephippial eggs can be estimated from spring population densities (Fig. 1; multiplied by the pond's surface area) recorded before any progeny could have been released by the first parthenogenetic generation. Since each Group C resting egg is a product of recombination, each hatching egg represents a distinct genetic entity. In 1979, 1980 and 1981, at least 70,000, 7,000 and 235,000 clones attempted to colonize the pond. Depending on how many ephippia hatch during other seasons, these numbers may be gross underestimates.

Since copulations are rarely observed in natural cladoceran populations, it is necessary to rely on inference to define the mating system in these animals. The general rarity of sexual individuals and consequent small electrophoretic sample sizes for them in this population further hinder such an analysis. However, distributions of sexual individuals were different from those of parthenogenetic females on at least three occasions (Table 3), in each case because the *MM/MM* genotype was in excess in the sexual forms. The total G value (Sokal and Rohlf, 1981) over all sexual vs. parthenogenetic comparisons ($G_T = 51.568$, $d.f. = 32$, $P < .05$) further indicates that some clones disproportionately contribute to resting egg production. Nevertheless, among the individuals equipped for mating at any one point in time, it is

likely that mating will be random. The rarity of ephippial females relative to males in this population (Fig. 1), the anticipated low encounter rates between the sexes (Gerritsen, 1980), the elevated mortality rate of highly visible ephippial females in the presence of visually-orienting predators such as *Ambystoma* (Mellors, 1975), and the absolute necessity of producing resting eggs in a temporary pond hardly seem conducive to the evolution of a selective breeding system. In no case were significant differences in the composite genotype distributions of males and ephippial females detectable (Table 3).

Fitness Characters of Genotypes

A series of life table determinations made on random collections of clones drawn from the pond permits a comparison of fitness attributes of the electrophoretic genotypes. On five occasions throughout the spring and summer of 1981, 100–200 clones were initiated from gravid females; depending on the relative abundance of the C clones during a particular experiment, sample sizes for the individual composite genotypes ranged between 0 and 40. Isolated progeny of these females were monitored daily for growth and reproduction until the release of their second clutch. All individuals were raised in the laboratory under pond temperature and light conditions with fresh pond water being substituted every other day. The *Pgm/Pept-2* composite genotypes of clones were determined by electrophoresing sibs or offspring. After employing Levene's test to insure homogeneity of variances and applying a square root transformation to normalize clutch size measures, three-way ANOVAs with experiment, *Pgm* and *Pept-2* genotypes as factors were performed on individual body size and reproductive characters.

The results summarized in Tables 4 and 5 are in accord with an hypothesis of approximate neutrality at the *Pgm* and *Pept-2* loci. In no case were there significant phenotypic differences between *Pgm*

TABLE 4. Mean phenotypic values and their standard errors for fitness attributes of Group C composite genotypes during five periods in 1981. Starting dates for experiments: 1—1 April, 2—8 May, 3—31 May, 4—21 June, and 5—23 July. B_{01} , B_{02} = mean offspring size for the first and second clutch (mm), B_k = size at maturity (mm), k = age at first reproduction (days), and $\sqrt{C_1}$, $\sqrt{C_2}$ = square root transformations of the sizes of the first and second clutches.

Trait	Exp.	N	Mean phenotypic value and SE								
			FF/MM	FF/MS	FF/SS	FM/MM	FM/MS	FM/SS	MM/MM	MM/MS	MM/SS
B_{01}	1	92	.611 (.013)	.575 (.005)		.596 (.012)	.602 (.007)	.640	.600 (.005)	.617 (.003)	
	2	36	.600			.592 (.021)	.660	.600 (.020)	.591 (.009)	.581 (.016)	
	3	71	.607 (.009)	.598 (.013)		.608 (.008)	.583 (.015)		.598 (.007)	.613 (.016)	.630
	4	103	.598 (.010)	.598 (.025)	.595 (.005)	.593 (.006)	.598 (.010)	.570	.593 (.005)	.598 (.010)	.550
	5	71	.586 (.009)	.573 (.012)		.616 (.008)	.588 (.008)	.567 (.012)	.603 (.007)	.572 (.010)	
B_{02}	1	69	.609 (.009)	.610 (.010)		.637 (.011)	.623 (.007)	.620	.609 (.004)	.612 (.007)	
	2	27	.620			.598 (.006)	.630	.580	.596 (.007)	.615 (.021)	.610
	3	57	.590 (.010)	.578 (.013)		.582 (.005)	.560		.586 (.007)	.566 (.009)	
	4	104	.644 (.005)	.618 (.015)	.650 (.010)	.639 (.005)	.644 (.008)	.660	.641 (.003)	.654 (.008)	.630
	5	72	.597 (.010)	.607 (.020)		.606 (.006)	.595 (.009)	.613 (.013)	.612 (.004)	.608 (.004)	.610
B_k	1	100	1.639 (.040)	1.565 (.005)		1.619 (.026)	1.617 (.048)	1.685 (.115)	1.619 (.014)	1.614 (.032)	
	2	42	1.430			1.613 (.018)	1.640	1.600 (.038)	1.586 (.018)	1.610 (.036)	1.600
	3	83	1.535 (.034)	1.507 (.025)		1.549 (.015)	1.510 (.030)		1.541 (.014)	1.535 (.017)	1.580
	4	95	1.568 (.022)	1.634 (.059)	1.555 (.015)	1.563 (.014)	1.587 (.022)	1.560	1.577 (.014)	1.588 (.018)	1.520
	5	76	1.584 (.027)	1.527 (.012)		1.604 (.027)	1.534 (.021)	1.515 (.023)	1.543 (.013)	1.486 (.025)	1.650
k	1	98	17.00 (.94)	18.50 (1.50)		18.00 (.84)	17.00 (.68)	18.50 (3.50)	17.45 (.28)	17.25 (.63)	
	2	42	22.00			23.43 (1.49)	21.00	26.00 (1.16)	24.47 (1.00)	24.50 (2.12)	23.00
	3	83	13.50 (.50)	12.83 (.31)		13.41 (.25)	13.00 (.58)		13.39 (.22)	13.46 (.34)	13.00
	4	104	9.88 (.23)	10.20 (.37)	9.00	9.70 (.17)	9.67 (.22)	10.00	9.71 (.14)	9.72 (.20)	9.00
	5	76	9.50 (.63)	9.00 (.58)		9.19 (.26)	8.50 (.31)	8.50 (.29)	9.10 (.19)	8.60 (.24)	10.00
$\sqrt{C_1}$	1	100	2.28 (.24)	2.28 (.55)		2.06 (.13)	2.38 (.18)	1.50 (.50)	2.03 (.06)	1.84 (.13)	
	2	40	1.00			1.32 (.14)	1.41	1.57 (.16)	1.53 (.08)	1.43 (.15)	1.41
	3	83	1.91 (.18)	1.77 (.09)		1.70 (.08)	1.38 (.21)		1.65 (.07)	1.60 (.03)	1.73
	4	104	2.20 (.14)	2.42 (.20)	2.22 (.22)	2.30 (.07)	2.44 (.11)	2.45	2.30 (.07)	2.29 (.08)	1.73
	5	76	2.28 (.21)	2.31 (.07)		2.08 (.14)	2.09 (.07)	2.14 (.24)	2.05 (.07)	1.93 (.27)	2.00
$\sqrt{C_2}$	1	98	2.85 (.10)	2.91 (.09)		2.44 (.18)	2.86 (.18)	2.83 (.00)	2.66 (.07)	2.69 (.09)	
	2	40	1.00			1.24 (.36)	1.00	.58 (.58)	1.34 (.17)	.72 (.21)	1.41
	3	81	.93 (.54)	1.27 (.42)		1.83 (.18)	.74 (.74)		1.57 (.13)	1.73 (.22)	.00
	4	102	2.93 (.16)	2.64 (.31)	3.16 (.16)	2.69 (.14)	2.81 (.14)	2.83	2.71 (.10)	2.66 (.10)	2.65
	5	76	2.37 (.19)	2.43 (.20)		2.43 (.14)	2.46 (.20)	1.74 (.27)	2.53 (.09)	2.48 (.14)	2.65

TABLE 5. Three-way ANOVAs (with unequal sample size) for the data in Table 4.

Source	B ₀₁				B ₀₂				B _k			
	d.f.	SS	F	P	d.f.	SS	F	P	d.f.	SS	F	P
Model	28	.0288	.78	.79	27	.1599	9.21	<.01	28	.4345	2.21	<.01
Exp	4	.0055	1.04	.39	4	.1462	56.81	<.01	4	.2864	10.18	<.01
Pgm	2	.0005	.18	.83	2	.0008	.66	.52	2	.0057	.41	.67
Pept-2	2	.0008	.31	.73	2	.0003	.22	.81	2	.0034	.24	.79
Exp × Pgm	8	.0049	.46	.88	8	.0054	1.06	.39	8	.0603	1.07	.38
Exp × Pept-2	8	.0144	1.36	.21	7	.0063	1.41	.20	8	.0687	1.22	.29
Pgm × Pept-2	4	.0027	.51	.73	4	.0009	.33	.86	4	.0100	.35	.84
Error	337	.4459			285	.1834			369	2.5966		
Total	365	.4747			312	.3433			397	3.0311		

or *Pept-2* genotypes, nor were there any significant experiment × genotype or *Pgm* × *Pept-2* interactions. If there are important epistatic interactions between these loci, they are completely obviated at the population level. Only experiment date accounted for significant variation in the body size and reproductive traits analyzed. Given the consistently low *F* values for all terms involving genotypes and the use of 300–400 clones in all of the analyses, it seems likely that any general fitness differences between the *Pgm/Pept-2* genotypes must be exceedingly small and discernible only with the use of many more clones than I employed.

Two sets of circumstances could be responsible for the maintenance of the high level of electrophoretic diversity in this population. Most clones in the pond might have equivalent fitnesses regardless of their *Pgm/Pept-2* composite genotypes, or real differences might exist between clones independent of their *Pgm/Pept-2* genotypes. To resolve this issue, the life history features for multiple offspring of several of the Group C clones were evaluated in three of the above experiments. In all three cases, all offspring were born on the same day and subjected to identical food, temperature and light conditions. The mean phenotypic values and standard deviations for the individual clones are summarized in Table 6.

Application of a two-level nested ANOVA to these data reveals several significant differences between clones nest-

ed within genotypes in individual experiments (Table 7). Although these analyses also suggest the existence of phenotypic differences between composite genotypes, contrary to the data just presented (Table 5), such differences may very well be a consequence of the low number of clones (1–3) representing each genotype. Both types of results are consistent with the hypothesis that, although many differences in fitness parameters exist between clones, at the population level selective differences at the one and two locus levels are obscured by their random association with genes encoding for life history features.

The Potential Significance of Random Genetic Drift

In the absence of very strong evidence for selection on the *Pgm* and *Pept-2* loci, it is essential to consider the possibility that genetic drift might be responsible for the long-term changes in gene frequency. The standard diffusion approximation,

$$V_{p,t} = pq[1 - \exp(-t/2N_e)],$$

can be used to estimate the probability of a given change in gene frequency in the absence of selection, mutation and migration (Nei, 1975). In this case N_e , the variance effective number, refers to the effective number of clones participating in ephippial production in a given year, not to the actual population size, and t is the duration in years.

TABLE 5. Extended.

k				$\sqrt{C_1}$				$\sqrt{C_2}$			
df.	SS	F	P	df.	SS	F	P	df.	SS	F	P
28	9,177.51	75.30	<.01	28	34.653	6.66	<.01	28	145.569	12.59	<.01
4	9,125.95	524.15	<.01	4	31.103	41.87	<.01	4	134.907	81.69	<.01
2	1.62	.19	.83	2	.819	2.20	.11	2	.027	.03	.97
2	4.46	.51	.60	2	.180	.48	.62	2	1.640	1.99	.14
8	19.95	.57	.80	8	.942	.63	.75	8	2.099	.64	.75
8	16.81	.48	.87	8	1.062	.71	.68	8	6.143	1.86	.07
4	8.72	.50	.74	4	.547	.74	.57	4	.753	.46	.77
367	1,597.46			367	68.150			363	149.875		
395	10,774.97			395	102.803			391	295.444		

TABLE 6. Mean phenotypic values and standard deviations for multiple offspring taken from second clutches of individual Group C clones. Clone number is simply an index of the stem mother isolated in a particular experiment. Characters and experimental starting dates are given in the legend of Table 4.

Exp.	Clone #	Genotype	B ₀₁		B ₀₂		B _k		k		$\sqrt{C_1}$		$\sqrt{C_2}$		N
			\bar{x}	SD	\bar{x}	SD	\bar{x}	SD	\bar{x}	SD	\bar{x}	SD	\bar{x}	SD	
1	102	FF/MM	.58	.02	.58	.01	1.58	.13	26.7	11.0	1.47	.50	1.15	1.00	3
	75	MM/MM	.59	.02	.64	.02	1.54	.07	18.4	2.1	1.37	.41	2.09	.22	6
	100	MM/MM	.59	.03	.58	.06	1.54	.05	22.1	1.1	2.05	.33	1.51	.81	7
	101	MM/MM	.63	.02	—	—	1.59	.06	24.2	1.5	1.57	.18	—	—	5
	85	MM/MS	.61	.04	.64	.02	1.56	.06	20.7	2.1	1.65	.39	2.12	.13	8
	118	MM/MS	.65	.01	.63	.03	1.58	.09	23.0	3.6	1.68	.47	—	—	5
4	115	FF/MM	.59	.02	.57	.05	1.54	.03	8.0	.0	1.81	.13	.74	.73	8
	125	FF/SS	.59	.02	.58	.01	1.49	.04	10.1	.6	1.85	.21	2.42	.12	9
	123	FM/MM	.58	.02	.61	.02	1.55	.10	10.8	1.5	2.01	.52	2.28	.61	4
	124	FM/MM	.58	.03	.55	.03	1.48	.03	9.0	.0	1.90	.19	1.53	.77	8
	126	FM/MM	.59	.05	.55	.04	1.51	.05	8.7	.8	1.69	.22	1.30	.82	10
	19	FM/MS	.58	.01	.57	.02	1.44	.03	7.1	.3	1.81	.29	1.85	.14	11
	121	MM/MM	.55	.03	.56	.01	1.54	.10	9.2	1.7	1.85	.50	1.52	.95	9
	122	MM/MM	.60	.04	.59	.03	1.44	.06	10.0	1.3	1.38	.32	1.83	.27	10
	120	MM/MS	.57	.03	.58	.01	1.46	.08	9.2	1.0	1.62	.24	1.47	1.18	6
	5	45	FM/MS	.63	.01	.65	.02	1.61	.04	7.0	.0	2.06	.45	2.99	.27
48		FM/MS	.48	.03	.60	.02	1.57	.09	8.0	1.2	2.39	.58	3.34	.28	5
59		MM/MM	.58	.02	.61	.03	1.53	.07	7.4	.6	2.57	.52	2.79	.08	5
30		MM/MS	.63	.01	.64	.01	1.70	.05	7.8	.5	2.78	.24	2.66	.63	5
73		MM/MS	.64	.02	.67	.02	1.71	.09	7.3	.5	2.47	.21	2.57	.61	7
20		MM/SS	.60	.01	.62	.01	1.55	.05	6.0	.0	2.06	.12	3.04	.16	4

If drift is to account for the annual changes in gene frequency of the magnitude observed, the effective number of clones must be very low. Taking $.17 + 2 SE = .19$ and $.34 - 2 SE = .32$ to be conservative limits to the change in frequency of the *Pgm-F* allele from 1978 to 1982 (Table 1), then there is less than a 5% chance that drift alone could account for such a change if $N_e \geq 69$; at $N_e = 120$ and 197, the probabilities become .01 and .001. Similarly, taking conservative estimates of .14, .16, and .09 to represent

the frequency of the *Pept-2-S* allele in 1980, 1981, and 1982, $N_e \leq 579$ and 52 are required for the probability of drift alone causing changes of the magnitude observed between 1980 and 1981 and between 1981 and 1982 to exceed 5%.

Are such effective clone numbers compatible with what we know about the structure of this population? The appropriate formula for the variance effective number of a diploid monoecious population (as a population of cyclically parthenogenetic clones can be considered to

TABLE 7. Nested ANOVAs (with unequal sample size) for the data in Table 6.

Trait	Source	Exp. 1				Exp. 4				Exp. 5			
		df.	SS	F	P	df.	SS	F	P	df.	SS	F	P
B ₀₁	Genotype	2	.0059	4.05	.033	5	.0040	1.14	.348	3	.0113	6.01	.002
	Clone within genotype	3	.0059	2.69	.074	4	.0062	2.22	.076	2	.0088	7.04	.003
	Error	20	.0146			64	.0444			31	.0194		
B ₀₂	Genotype	2	.0058	2.47	.118	5	.0046	1.15	.344	3	.0080	5.09	.006
	Clone within genotype	3	.0091	2.60	.090	4	.0133	4.20	.005	2	.0067	6.47	.005
	Error	15	.0176			54	.0429			30	.0156		
B _k	Genotype	2	.0034	.34	.718	5	.0767	4.30	.001	3	.1109	5.24	.005
	Clone within genotype	3	.0130	.85	.480	4	.0770	5.39	.001	2	.0091	.64	.533
	Error	29	.1485			74	.2642			31	.2185		
k	Genotype	2	110.303	2.69	.086	5	58.499	11.72	<.001	3	2.765	1.11	.359
	Clone within genotype	3	90.443	1.47	.245	4	17.867	4.48	.003	2	2.804	1.69	.201
	Error	27	554.224			73	72.862			31	25.675		
√C ₁	Genotype	2	.097	.30	.744	5	.755	1.17	.332	3	.723	1.34	.278
	Clone within genotype	3	.925	1.90	.151	4	1.898	3.68	.009	2	.252	.70	.502
	Error	29	4.699			72	9.291			31	5.553		
√C ₂	Genotype	2	1.842	2.41	.112	5	10.950	4.81	.001	3	2.858	6.81	.001
	Clone within genotype	3	1.577	1.38	.275	4	3.062	1.68	.164	2	.444	1.59	.221
	Error	23	8.774			73	33.228			30	4.195		

be) has been derived by Kimura and Crow (1963):

$$N_e = \frac{2N_p}{1 - \alpha + (1 + \alpha)V_k/\bar{k}} \quad (1)$$

In this case, N_p = the total number of resting eggs surviving to hatch in the following year, \bar{k} = the mean number of viable resting eggs per parent clone through both male and female function = $2N_p/N_a$ where N_a is the number of parent clones, V_k = the variance in progeny number, and α is a measure of the breeding population's deviation from Hardy-Weinberg proportions (here $\alpha = 1 - (H/H_o) \approx .8$). In a variable environment the actual variance effective number is approximated by the harmonic mean of the yearly N_e and is much closer to that in years of low than of high N_e . If mating is approximately random in this population, then $V_k \approx \bar{k}$, $N_e \approx N_p$, and there is very little chance that drift could be influencing the gene frequencies since the minimum estimate of N_p is 7,000.

Suppose, however, that my assumptions about random mating are in error. A simple but extreme way to modify equation (1) to account for nonrandom mating is to assume that, because of mate discrimination, ephippial mortality, etc., only a subset of the clones in the population produces viable resting eggs and that the members of this subset have equal reproductive success. Under these conditions, the variance in progeny production becomes

$$V_k = \bar{k}^2 \left\{ \frac{N_a}{N'_a} - 1 \right\} \quad (2)$$

where N'_a is the number of clones producing resting eggs. We can now determine the types of population structure that would be required to give a variance effective number in the range that could cause enough drift to account for the gene frequency changes observed in this study. Substituting (2) in (1), the variance effective number of clones becomes

$$N_e = \frac{2N_p}{1 - \alpha + (1 + \alpha)\left(\frac{2N_p}{N_a}\right)\left(\frac{N_a}{N'_a} - 1\right)}$$

and it becomes clear that the Group C population structure would have to be rather different from what I have assumed for drift to take on significance. For instance, at the minimum progeny level observed in this study ($N_p = 7,000$) and with a very conservative estimate for the number of adult clones, $N_a = 1,000$, $N_e = 139$ when only 100 clones participate in breeding. Not until $N'_a = 50$ does $N_e = 66$. Thus, with the possible exception of the *Pept-2* locus between 1980 and 1981, it appears unlikely that random drift is responsible for the observed year-to-year changes in gene frequencies.

The Intensity of Selection

Having eliminated random drift as a major short-term evolutionary force operating on the *Pgm* and *Pept-2* loci, we can now proceed to examine the mode in which selection might be operating on this population. A measure of relative fitness can be determined by taking the ratio of genotype frequency in the second to the first half of a year. However, even with the large sample sizes in Table 1, the confidence limits for the resultant fitness estimates are quite large (Table 8) rendering it difficult to make any precise statements about selection.

At the *Pgm* locus the confidence limits are narrowest for the *MM* genotype, and it appears that there may have been a slight increase in its fitness in the last two years of the study. Nevertheless, in all five years either the *FF* or *FM* genotype had the superior fitness. Averaged over all five years the relative fitnesses of the *Pgm-FF*, *FM*, and *MM* genotypes are $.97 \pm .38$, $1.00 \pm .25$, and $.80 \pm .16$. At an average of ~ 10 parthenogenetic generations/year the fitnesses/generation necessary to bring about these annual differentials are .977, 1.000, and .997.

Although the 1980 data for the *Pept-2* locus are questionable because of the small sample sizes and absence of *SS* in-

TABLE 8. Relative fitness values of single locus genotypes estimated by dividing late (P_L) by early (P_E) genotype frequencies in Table 1 and normalizing so that the highest genotypic fitness = 1.00. Standard errors were determined by Taylor expansion of P_L/P_E which gives

$$\text{Var}(P_L/P_E) \approx \frac{1}{P_E^2} \left[\frac{P_L(1 - P_L)}{N_L} + \frac{P_L^2(1 - P_E)}{P_E N_E} \right]$$

where N_E and N_L refer to sample sizes in the first and second halves of the year.

Relative fitness ± 2 SE			
<i>Pgm</i>	<i>FF</i>	<i>FM</i>	<i>MM</i>
1978	.41 \pm .27	1.00 \pm .42	.57 \pm .06
1979	1.00 \pm .82	.53 \pm .22	.69 \pm .07
1980	—	1.00 \pm .52	.51 \pm .08
1981	.92 \pm .26	1.00 \pm .13	.84 \pm .06
1982	1.00 \pm .31	.74 \pm .13	.79 \pm .11
<i>Pept-2</i>	<i>MM</i>	<i>MS</i>	<i>SS</i>
1980	.05 \pm .01	1.00 \pm 2.01	—
1981	1.00 \pm .06	.69 \pm .10	.59 \pm .30
1982	1.00 \pm .06	.28 \pm .09	.16 \pm .34

dividuals in the first half of the year, a comparison of the 1981 and 1982 data again suggests that the relative fitnesses changed significantly in value but not rank. Again assuming 10 generations/year, the average fitness values of the *MM*, *MS*, and *SS* genotypes over these two years (1.00, .48, and .38) can be accounted for by per generation fitness differences of only 1.00, .93, and .91.

The above estimates probably exaggerate the true fitness differences between genotypes since some mating generally occurs early in the year before many of the clones of inferior genotypes will have been reduced in frequency. Thus, these results help clarify why the life table analyses failed to reveal significant fitness differences at these two loci.

DISCUSSION

These results suggest that the structure of the Group C population approximates that of an ordinary, randomly mating bisexual population except that the cost of producing males is avoided for a large portion of the life cycle. The lack of any strong evidence for temporal increases in linkage disequilibrium or associative

epistasis at electrophoretic loci indicates that this population is never dominated by a small number of clones, and is in striking contrast to the results of a clonal selection experiment performed on unisexual lines of *Drosophila mercatorum* (Annest and Templeton, 1978). Apparently many more than the annual average of ~ 10 parthenogenetic generations experienced by the Group C population would be required for the effects of clonal selection to reveal themselves at the marker level. The small number of clones employed in the *Drosophila* experiment (a maximum of 157 successfully making the reproductive transition to parthenogenesis) was conducive to the development of strong associations between marker loci via the chance hitch-hiking of alleles in superior clones. But the electrophoretic results and estimates of annual ephippial hatches suggest that the Group C population in Busey Pond may rarely ever consist of less than 10^4 – 10^5 clones. Thus, because of its large population size and annual episode of sex, the Group C population is able to attain the advantage of unisexuality (rapid proliferation) with little of the anticipated cost (reduced genetic variability).

Similar electrophoretic results have been obtained for two other cyclically parthenogenetic *Daphnia* living in temporary ponds that prevent the overwintering of clones: *D. magna* (Hebert, 1974a) and *D. carinata* (Hebert and Moran, 1980). In both studies the genotype frequencies of polymorphic loci were relatively stable within and between years as well as generally in close agreement with Hardy-Weinberg expectations. As illustrated by this study significant temporal changes in genotype frequencies do occur, but in no case are there any striking temporal discontinuities that would be expected if one or two clones suddenly came to dominance.

These results make White's (1973) suggestion that insurmountable cytogenetic barriers prevent the widespread evolution of cyclical parthenogenesis difficult to dismiss. Indeed, the transition to pure parthenogenesis alone has been repeat-

edly demonstrated to be exceedingly difficult (Stalker, 1954; Astaurov, 1967; Carson, 1967; Templeton, 1979b), only $\sim 10^{-6}$ eggs of several wild insects showing any parthenogenetic capacity prior to their experimentally imposed transition. The additional complications of cyclical parthenogenesis must make the requirements for a transition to it even more stringent.

Even if the cytogenetic barriers can be overcome, at least two additional problems face a newly arisen cyclical parthenogen. First, the fact that the cytogenetic requirements are so rigid means that cyclically parthenogenetic species are probably founded by single transitional events, and that the single founders will have radically different genetic constitutions than their bisexual parental species (Templeton et al., 1976). Even if the initial founder had a perfectly adapted reproductive system, the early ecological and evolutionary success of the species would critically depend on the genetic structure of the founder (Templeton, 1979a). Moreover, the obligatory inbreeding that would result from such a transition would very likely intensify the selective process even further.

Another problem facing even well-established cyclical parthenogens will be the perpetual possibility of displacement by secondarily derived obligate parthenogens. Once the transition to cyclical parthenogenesis has been made, it may be a relatively minor step to remove the sexual phase. This has occurred in *Daphnia* on many occasions (Hebert and Crease, 1980; Hebert, 1981; Lynch, 1983), and is known in rotifers (Ruttner-Kolisko, 1946) and aphids (Dixon, 1977) as well. Numerous examples exist for the displacement of bisexual species by their obligately parthenogenetic derivatives (and vice versa) via genetic disruption, sperm robbing and direct competition for resources (Lynch, unpubl.), and there is no reason that this should not apply to cyclical parthenogens. The fact that many *Daphnia pulex* populations consist exclusively of obligate parthenogens (Hebert and Crease, 1980) and that others

contain both cyclical and obligate types (Lynch, 1983) suggests that local extinctions of cyclical parthenogens do indeed occur.

The only other cyclical parthenogens on which any genetic work has been done are the rotifers (King, 1977, 1980) and aphids (May and Holbrook, 1978; Wool et al., 1978; Suomalainen et al., 1980; Tomiuk and Wöhrmann, 1980, 1981), and the data are much less extensive than those for *Daphnia*. However, those species that have been electrophoretically surveyed are <10% polymorphic, and several aphid populations surveyed at as many as 19 enzymatic loci have been found to be 100% monomorphic. Striking temporal discontinuities in genotype frequencies have been reported for both rotifers and aphids, and the gross deviations from Hardy-Weinberg expectations noted for rotifers have led to the suggestion that their populations are heavily dominated by a few clones. These attributes are much more similar to those of permanent than intermittent *Daphnia* populations.

In permanent environments the sexual production of resting eggs is not a necessity for *Daphnia* and although they retain the potential for resting egg production, many clones function as obligate parthenogens by overwintering in the water column. This prolongs the competitive process for any set of clones and presumably greatly reduces the number of clones coexisting in a population at any one time. As a consequence, permanent *Daphnia* populations exhibit strong interlocus associations and deviations from Hardy-Weinberg equilibrium (Hebert, 1974b; Lynch, 1983). Massive temporal changes in genotype frequencies are observed not because the intensity or direction of selection is necessarily different from that acting on intermittent populations but because any phenotype that is selected for is likely to be highly correlated with specific alleles when clonal diversity is low. Presumably the Busey Group C population would begin to behave in an analogous fashion should the pond fail to dry for one or more years.

More information on the breeding systems and overwintering mechanisms of rotifers and aphids will have to be gathered before their genetics can be interpreted in light of these generalizations based on *Daphnia*. Indeed, it is quite possible that the apparent discrepancies between intermittent *Daphnia* populations on the one hand and rotifers and aphids on the other may exist simply because the latter are not as completely reliant on cyclical parthenogenesis as the former are. Since the rotifers examined by King (1977, 1980) inhabited permanent environments, it is plausible that their populations were at least partially initiated each year by rare and undetected individuals overwintering in the water column. It is also feasible that some clones within electrophoretically surveyed populations of rotifers and aphids were obligate parthenogens. The existence of either condition would help explain the similarities with permanent *Daphnia* populations.

SUMMARY

As a consequence of annual mating and the resultant recombination, populations of cyclically parthenogenetic *Daphnia* consist of several thousands of genetically distinct clones. Major selective differences exist between individual clones, but differences between genotypes at the one and two locus levels are rendered nearly undetectable by their random association with important fitness polygenes. The absence of any pronounced evidence of linkage disequilibrium, associative epistasis, or exceptionally strong selection at the electrophoretic level is contrary to theoretical expectations and suggests that the structure of large cyclically parthenogenetic populations is very similar to that of bisexual species except that the cost of producing males is avoided for a large portion of the life cycle. While this would seem to make cyclical parthenogenesis an evolutionarily attractive strategy, several significant barriers may account for the rarity of organisms that have made successful transitions to

this mode of reproduction: the evolution of a reproductive system capable of both unisexual and bisexual propagation, the founding of populations by single transitional events that reduce genetic diversity and enforce inbreeding, and the vulnerability of established cyclical parthenogens to displacement by secondarily derived obligate parthenogens.

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