

Table 2. Molecular weights of immunoglobulins and polypeptide chains from *Mustelus canis*. The molecular weights were determined by the high-speed equilibrium method of Yphantis (10). Molecular weights and standard deviations were calculated from a weighted least squares analysis as suggested by Yphantis (10).

Immunoglobulin	Molecular weights		
	Intact molecule	Light chain	Heavy chain
17S	982,000 $\pm$ 25,500	20,100 $\pm$ 500	71,600 $\pm$ 2,800
7S	198,000 $\pm$ 6,000	20,500 $\pm$ 300	73,400 $\pm$ 4,000

from their amino acid and carbohydrate contents.

On the assumption that no carbohydrate was present, the $\bar{V}$ of light chains was 0.727. The value obtained for heavy chains, if all of the carbohydrate was present on this portion of the molecule, was 0.723. Both whole immunoglobulins had values of 0.725, close to the value of 0.722 (9) for human γ M-immunoglobulin. The molecular weights of the light chains are similar to those reported for light chains of the immunoglobulins in lemon shark (11) and mammals. Heavy chains on the dogfish immunoglobulins have molecular weights in the range reported (9) for

μ -chains on mammalian immunoglobulins (approximately 70,000); this value is considerably higher than that reported (12) for γ -chains (approximately 55,000). The molecular weight of the α -chains from γ A-immunoglobulin has not been reported and thus cannot be compared. A dogfish molecule composed of two light chains and two heavy chains would have a molecular weight of approximately 188,000. The observed value for the 7S immunoglobulin was 198,000 $\pm$ 6,000. Five units, each composed of two light and two heavy chains, would have a molecular weight of 940,000. The observed value for the 17S immunoglobulin was 982,000 $\pm$ 25,500. The amino compositions (Table 1) are consistent with the above assignment of chains within the dogfish immunoglobulin molecules.

Our data provide support for the conclusion that the 7S and 17S immunoglobulins of the smooth dogfish belong to the same class. This class resembles that of γ M-immunoglobulins of the higher vertebrates. A report (11) on immunoglobulins in the lemon shark suggests that similar relations obtain in other elasmobranchs. As proposed (1), the μ -chains may have been the first of the heavy chains to have evolved. This is of interest in the light of a suggestion (13) that the structural genes for heavy chains may have arisen from duplication and mutation of genes coding for light chains.

J. MARCHALONIS

G. M. EDELMAN

Rockefeller University, New York

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Initial and Resultant Population Densities in Chickens between Brooding and Sexual Maturity

Abstract. Seven flocks of chickens were raised in groups from weeks 5 to 9 after hatching. Initially the groups had the same number of chickens but they differed in population density. The number of birds that survived to week 9 was strongly related to the initial population density.

Thiessen and Rodgers (1) summarized the results of a number of studies on factors that limit population density in infrahuman groups. Thiessen (2) subsequently proposed a correlational model to take into account the types of variables related to mortality, but as yet he has not been able to assign relative weightings to these variables. One of his factors, "prior experience with grouping," may facilitate survival under high population density. In this report we seek a precise relationship between initial and resultant densities. Since most of the theory and data seem related to mammalian groups, domestic fowl were used to supplement them.

Although Thiessen (3) prefers to make an assumption about the role of interspecific aggression, both field and laboratory studies (4) have demonstrated social interactive processes that result in a reduction in density. Most of these processes, however, apply to sexually mature or infantile-dependent members of infrahuman groups. Since little is known about density reduction among juveniles, we examined chicks after brooding and before sexual maturity. Between weeks 5 and 9 after in-

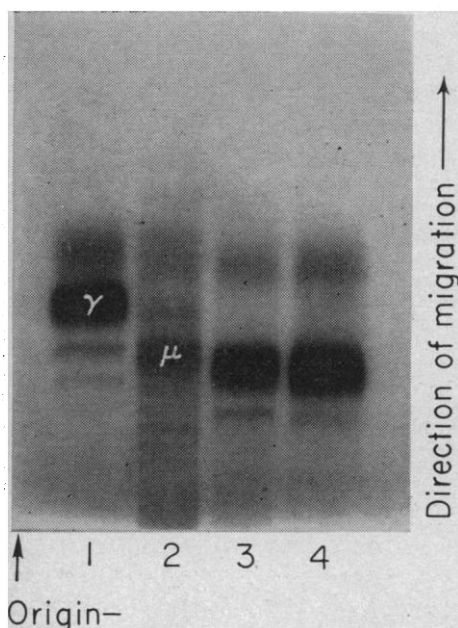


Fig. 1. Comparison of reduced alkylated immunoglobulins of dogfish and man by starch-gel electrophoresis in urea. Origin 1, human γ G-immunoglobulin; origin 2, human γ M-immunoglobulin; origin 3, dogfish 17S immunoglobulin; origin 4, dogfish 7S immunoglobulin. The letters γ and μ refer to the position of the heavy chains of human γ G- and γ M-immunoglobulins respectively. All samples were reduced and alkylated in the presence of 8M urea. Electrophoresis was performed in 8M urea-formate buffer according to the conditions of Edelman and Poulik (3).

Table 1. Number of survivors in flocks of chickens between brooding and sexual maturity.

Group	Square meters per survivor	Number of survivors				
		Week 5	Week 6	Week 7	Week 8	Week 9
Series 1						
A1	4.2	12	12	12		
B1	2.8	12	11	10	10	
C1	1.4	12	11	8	6	
Series 2						
A2	4.2	12	12	11	11	11
B2	2.8	12	11	10	10	10
C2	1.4	12	10	8	7	7
D2	0.31	12	12	12	5	2

cubation, "survivorship" was examined in small flocks that were equal in initial size but occupied different areas of floor space. If some factor arising out of or associated with "experience with density" should facilitate survival, then the correlation between initial and resultant densities would be positive, and the relationship between these variables would monotonically increase. (Such a relationship, however, must have a lower and should have an upper limit.) On the other hand, if the numbers of survivors were unrelated to the chick's "experience with density," then the correlation between initial and resultant densities, under the present conditions, would be of zero order, that is, all treatment groups would reduce to about the same level.

In series 1, 36 incubated, New Hampshire-White Leghorn crossed chicks were taken from heated brooders ($N = 12$ per brooder) and randomly allocated to three flocks. The initial amount of space allotted each bird was 4.2 m² for group A, 2.8 m² for group B1, and 1.4 m² for group C1. Series 2 was a replicate with an additional flock ($N = 12$) that had an initial area of 0.31 m² per bird. In each flock, food (a medium energy commercial ration) and water were available as desired. Flocks were maintained indoors under artificial heat and light (14 hr/day).

Table 1 shows that series 1 and 2 are almost identical with respect to replicates and this was confirmed by statistical testing. The number of survivors between density levels was significantly different on the chi-square test ($p < .05$) except for those birds maintained in areas between 4.2 m² and 2.8 m² when the difference approached significance ($p = .15$).

In series 1 recordings were discontinued during week 9 since dominance

fighting was observed in all treatment conditions, a result consistent with earlier studies (5). The number of survivors during week 9 for series 2 is given, and, although dominance encounters (but no mortalities) were then observed in groups A2, B2, and C2, no such behavior was observed in group D2. Since sexual maturity and dominance fighting coincide, it seems likely that the former may have been delayed under very high density. Similar findings (6) have been reported with mice.

Records of wounds and scars indicated that physical assault as a cause of death was confined to one flock only, C1. In this group all carcasses

had been extensively cannibalized about the tail and saddle. This commenced in week 6 with members of C1 pecking each other's tail quills; should hemorrhage ensue the member was pecked until dead. When the resultant density was reached, however, no member died after such assaults although several hemorrhages subsequently occurred.

Within the limits of age, strain, and densities under study, a strong consistent relationship ($r = 0.999$, $p < .001$) existed between initial and resultant density. Also, within the limits specified, the function relating the variables is: Resultant density = 0.64 initial density + 1.77.

G. L. MANGAN

M. G. KING

Menninger Foundation, Topeka, Kansas, and Department of Psychology, University of Sydney, Sydney, Australia

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Respiratory Control: Loss in Mitochondria from Diseased Plants

Abstract. *Mitochondria from healthy oat seedlings oxidized succinate with good respiratory control and high ratios of adenosine diphosphate to oxygen. After treatment with victorin, the pathotoxin responsible for symptoms of Victoria blight of oats, susceptible seedlings yielded mitochondria with little respiratory control and lower ratios of adenosine diphosphate to oxygen. No such effects were obtained with victorin-treated resistant seedlings or when victorin was added directly to mitochondria from healthy susceptible or resistant plants. These data indicate that victorin-induced disease results in a reduction in efficiency of the energy-generating system of isolated mitochondria.*

Pathological increases in respiration are characteristic of diseased plant tissue (1). Attempts to determine the extent to which pathological respiration is coupled to synthesis of high-energy phosphate bonds have yielded conflicting results. In two investigations, phosphorylative-oxidative (P/O) ratios were lower in mitochondrial preparations from diseased plants than in those from healthy ones (2, 3); in another, no such change in P/O ratios was found during development of disease (4). The conflict in these results may be apparent rather than real because the pro-

cedures used, particularly the extraction of mitochondria in strong buffers, have resulted in loss of respiratory control with both animal and plant preparations (5). Respiratory control (RC) ratios (rate of substrate oxidation in the presence of a suitable phosphate acceptor divided by rate in the absence of such an acceptor) are considered to be better indicators of the intactness of mitochondria than P/O ratios (5). Mitochondria from potato tuber (*Solanum tuberosum* L.) slices inoculated with the fungus *Ceratocystis fimbriata* and incubated for 2 to 3 days had lower RC