# SOME CONSIDERATIONS ON SIBLING COMPETITION AND AVIAN GROWTH RATES

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ABSTRACT.—Werschkul and Jackson (1979) suggested that sibling competition is a dominant force in the evolution of avian growth rates. Two predictions of this hypothesis, (1) that species with broods of one (B1) should grow more slowly than those with larger broods (B2+), and (2) that growth rates should be more variable among B1 species than among B2+ species, are confirmed. A simple numerical calculation illustrates that sibling competition may exert very strong selection on growth rate compared to that of external mortality factors, provided that growth rate accurately predicts the outcome of competition between sibs. The results of such selection depend on the evolutionary flexibility of growth rate in response to factors causing mortality of nestlings.

Attempts to relate growth rate to total mortality rate of nestlings and to mortality within broods failed. Hence, I could find neither direct support for the sibling-competition growth-rate hypothesis nor evidence for flexibility of growth rate with respect to nestling mortality. Three alternatives for slow growth among B1 species are discussed: (1) limitation by availability of food, (2) limitation by the availability of essential nutrients in the diet, and (3) increased precocity of the chicks of B1 species. *Received 11 August 1980, accepted 19 May 1981.*

NATURAL selection adjusts the reproductive patterns of birds by means of the interaction between factors in the environment and the evolutionary potential of the phenotype. The characteristic genetically determined growth rate of the young of each species balances factors favoring slower growth and those favoring more rapid growth (e.g. Lack 1968). Among the first of these are the rates at which energy and nutrients are required by the chick. Among factors favoring more rapid growth are those that cause mortality of whole broods (e.g. predation and storms) and competition among siblings.

Recently, Werschkul and Jackson (1979) have suggested that ". . . the influence of sibling competition on nestling development is a dominant force determining the evolution of avian growth rates." This idea receives support from comparisons between species having broods of one, hence without sibling competition, and those rearing larger broods. The sibling-competition hypothesis allows two predictions concerning growth rates: (1) individuals of species with broods of one grow more slowly than those of species with larger broods, and (2) the characteristic growth rates of chicks vary more among species that rear broods of one than they do among species that rear larger broods. The second prediction is implied by the hypothesis, because, if growth rate were released from the intense selection resulting from sibling competition, it could ". . . respond more freely to selective pressures outside the nest (e.g. nest site, predation) and to the food gathering abilities of adults . . . ." Werschkul and Jackson tested the predictions of their hypothesis by comparing the growth rates of species with broods of one (hereafter B1) to those of species having larger broods (B2+). In all the species in their sample, young are fed by their parents until fully grown. Both predictions were confirmed by the analysis, but there remained unresolved difficulties with the hypothesis. First, Wershkul and Jackson's statistical comparisons did not satisfy the assumptions of an analysis of covariance and were not, therefore, strictly valid. Second, because all the B1 species in their sample were pelagic seabirds, support for the sibling-competition hypothesis may fortuitously reflect the influence of other ecological or demographic factors associated with these species. Third, the sibling-competition hypothesis contains implicit assumptions concerning the strength of selection and genetic variation in growth rate that as yet require evaluation. Fourth, the slow growth rates of B1 species suggest several alternative but not mutually exclusive hypotheses, namely that slow growth in B1 species is

related to ecological factors affecting food supply or food quality rather than to brood size and that growth rate is determined by precocity of development, which in turn is related to brood size.

Understanding the role that sibling competition has played in diversifying growth rates of birds requires a quantitative evaluation of many alternative hypotheses. At this point, so few relevant data have accumulated that we can hope to do little more than set forth basic ideas, expose some implicit but unstated assumptions, seek available data to test the predictions of each hypothesis, and devise lines of inquiry that bear directly upon factors that determine avian growth rates. This problem has assumed some importance in avian biology, and in evolutionary studies more generally, because it highlights the relative roles of ecological factors and internal constraints in evolutionary diversification (Case 1978, Ricklefs 1979a, Stearns 1980).

My purpose in this paper is to reanalyze the relationship between growth rate and brood size, explore some assumptions implicit in the sibling-competition hypothesis, and discuss alternative hypotheses for slow growth in B1 species. Although there is ample evidence for sibling competition (see Werschkul and Jackson 1979), its role as a selective agent depends on the evolutionary flexibility of growth rate in response to mortality, for which there is no direct evidence.

*Brood size and growth rate.*—As an index to growth rate, Werschkul and Jackson used the rate constants ($K$) of Gompertz equations fitted to the averaged growth curve of each species (see Ricklefs 1967, 1968). Most of the data were from Ricklefs (1973). Individuals of species having large size as adults grow more slowly than those of species having small size as adults. To take into account the relationship between growth rate and body size, Werschkul and Jackson plotted the inverse of growth rate ($1/K$) as a function of the logarithm of the asymptote or final weight plateau of the growth curve and then applied a statistical analysis to the regressions of growth rate upon size for B1 species and B2+ species. The analysis revealed that B1 species grew more slowly than B2+ species and that, when the effect of adult body size was factored out, growth rates of B1 species were more variable than those of B2+ species.

Statistical inference about the difference between two regression lines or the difference between the variances of residuals is properly made only when the residuals about the lines are normally distributed. In Werschkul and Jackson's analysis, this condition was not met. The relationships for B1 species were not linear, and the residuals for both B1 and B2+ species increased as the value of the asymptote increased (Werschkul and Jackson 1979: Fig. 1).

Properly analyzed, growth rates are converted to logarithms, by which transformation the residuals are normalized (Fig. 1). The present analysis includes growth curves obtained in 43 studies of B2+ species (solid symbols in Fig. 1) and 29 studies of B1 species (open symbols). The relationships of growth rate to adult body size and brood size were examined by an analysis of covariance in which the dependent variable ($Y$) is the logarithm (base 10) of growth rate ($K$), and the independent variable or covariate ($X$) is the logarithm of the asymptote ($A$) of the growth curve. Brood size ($B$) is a dichotomous dummy variable separating B1 and B2+ species. I used the General Linear Models procedure (GLM) of SAS. First, a multiple regression revealed that the relation of $Y$ upon $X$ was essentially linear; variation in $Y$ was partitioned with respect to the variables as follows: $B$ ($F = 111.5$, $P < 0.0001$), $X$ ($F = 122.2$, $P < 0.0001$), $X^2$ ($F = 4.0$, $P = 0.05$), and $X^3$ ($F < 0.01$, $P = 0.96$). I then determined that the slopes of the regressions of $Y$ upon $X$ did not differ between the two brood-size categories by testing the significance of the $BX$ interaction ($F = 0.05$, $P = 0.82$). Assuming a common slope, I then calculated the regressions of $Y$ upon $X$ for each brood-size class. The slope of the regression was $-0.321 \pm 0.029$ SE ($F = 58.6$, $P < 0.0001$). The intercepts for B2+ species ($-0.036 \pm 0.086$ SE) and B1 species ($-0.288 \pm 0.030$) differed significantly ($F = 107.0$, $P < 0.0001$). Hence, Werschkul and Jackson's first prediction is confirmed. In addition, the standard deviation of residuals about the regression line for B1 species (0.150) exceeded that for B2+ species (0.103) ($F = 2.12$, $P < 0.025$), thus confirming Werschkul and Jackson's second prediction, although not so strongly as in their analysis.

*Mortality and the strength of selection due to sibling competition.*—The idea that sibling competition exerts a strong influence on the evolutionary optimization of growth rate implies

genetic variation in chick survival as it is influenced by sibling competition. In principle, one ought to be able to measure mortality caused by sibling competition and relate growth rate to it directly. It is frequently argued that the selection of a particular adaptation reduces the effect of the selective agent and obscures the relationship between adaptation and environment. But this argument cannot be applied to sibling competition and growth rate. As an allele for rapid growth spreads through a population under selection, it causes growth rates within a brood to become uniform and thereby intensifies sibling competition. In this case, it appears that the strength of the selective agent keeps pace with the adaptive response of the population. Hence, if sibling competition were responsible for selecting different growth rates in two populations, we would expect to see these differences expressed in mortality.

The relative importance of sibling competition and factors causing the death of entire broods may be estimated from data on nesting success. For example, in Wiens' (1965) study of the Red-winged Blackbird (*Agelaius phoeniceus*), 72% of nests in which eggs hatched fledged at least one offspring. Of the nestlings that hatched, 61% survived to leave the nest. Total nestling success ($S$) is the product of the survival probability of entire broods ($S_b$) and the survival probability within broods ($S_{wb}$). If we assume that all mortality within broods ($M_{wb}$) is caused by sibling competition ($M_{sc}$), then $S_{wb} = 1 - M_{sc}$ and $M_{sc} = 1 - (S/S_b)$ or $1 - (0.61/0.82) = 0.14$ in the case of the Red-winged Blackbird.

The mortality attributable to sibling competition was on the order of 14% of young in successful nests, assuming that sibling competition did not cause the loss of entire broods and its effects were expressed prior to fledging. If sibling competition caused the death of one chick per nest and brood size was four at hatching, mortality due to sibling competition would have occurred in 56% of nests. The nestling period of the Red-winged Blackbird is about 10 days. If the probability of death from factors causing the loss of whole broods were distributed evenly over the nestling period (cf. Ricklefs 1969a), the survival of nests would be a negative exponential function of time, declining at the rate of 3.3% of broods remaining each day. If growth rate were to increase by
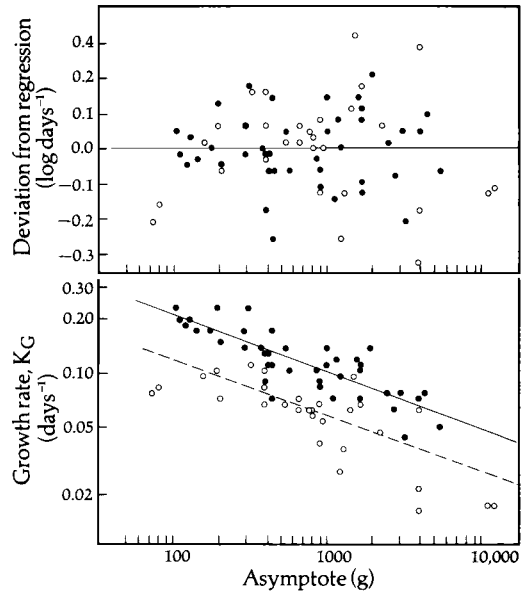


Fig. 1. Bottom: Relationship between growth rate ($K$, the rate constant of the Gompertz equation) and asymptote of the growth curve among species of birds that rear broods of one (open circles) and those that rear larger broods (filled circles). Data are from Ricklefs (1973). Top: Residuals of the data about the regression line.

10%, thereby reducing the nestling period by 1 day, the probability of survival as affected by external factors would increase by about 3.3%. The selective force of sibling competition depends upon the degree to which body size at a particular age determines the probability of losing contests among siblings. If all the young in a brood hatched at the same time, a 10% difference in growth rate constant ($K$) would result in a difference in body size on the order of 10% at most. If such an increase in growth rate guaranteed that a particular nestling would not die, then one out of three of its sibs would die within 56% of all broods, a mortality rate of 19% for each. Hence, the rapidly growing sib would gain a selective advantage of 19% in this simple example. The calculation illustrates the potential selective strength of sibling competition compared to mortality caused by external factors.

Competition among siblings may be reduced by parental manipulation of the spread of hatching. By virtue of hatching one day apart, nestling blackbirds having identical growth rates would differ in size by as much as 30–

40% during the first few days of the postnatal period and by about 10% when the chicks are half grown. Therefore, if the incubation pattern of the parents caused the last one or two eggs in the clutch to hatch later than the others and if the effects of sibling competition fell disproportionately upon the last hatched in the brood, small changes in growth rate might not reverse the last-hatched chick's size disadvantage and would not improve the success of first-hatched chicks in sibling competition. Although the ability of parents to manipulate sibling competition by hatching their eggs asynchronously (Lack 1954, Ricklefs 1965, O'Connor 1978) could obscure the effects of variation in growth rate on survival probability, sibling competition holds the potential for exerting a powerful selective force on rate of development.

*Selective mortality and growth rate.*—If variation in probability of survival were responsible for genetic differences in growth rate among species, one would expect to find considerable evolutionary flexibility in the length of the development period and a strong correlation between growth rate and selective mortality. I have argued that growth rate is bounded with an upper limit determined by tissue-level constraints relating embryonic activity to level of differentiation for mature function (Ricklefs 1973, 1979a, b). According to this hypothesis, both strong and weak selective mortality are sufficient to push growth rate close to the physiological maximum (Ricklefs 1969a); variation among species reflects patterns of differentiation and anatomical proportions rather than variation in selective mortality.

To test the hypothesis that variation in growth rate reflects selective mortality, I plotted the relationship between daily mortality rate of nests and the growth rate constant of the logistic equation for temperate zone passerines (Ricklefs 1969a). The two were not significantly correlated, nor could I find any systematic relationship between growth rate and mortality in other groups. Case (1978) argued that, because mortality data are variable within species and because my growth and mortality data were drawn from different studies for many of the species, one might expect a relationship between the two to be obscured. But because the same studies reveal a strong neg-

ative correlation between nestling period and daily mortality rate (Ricklefs unpubl.), the test of the relationship between growth rate and mortality and the conclusion that growth rate is relatively inflexible appear to be valid.

It is possible to test the relationship between growth rate and sibling competition directly if one assumes that mortality within broods is directly proportional to the strength of selection by sibling competition. The difference between rate of nestling mortality and brood mortality (the partial loss of Ricklefs 1969b) estimates the within-brood mortality. In 12 species of temperate-zone passerines, these values ranged between −0.12 and 1.19% per day. Growth rate constants of the same species ranged between 0.46 and 0.65 per day. The two measures were not significantly correlated ($r^2 = 0.09$, $P > 0.10$), and the sibling-competition growth-rate hypothesis is thus unsupported by these data.

According to Fig. 1, there is little overlap in the growth rates, corrected for body size, of B1 and B2+ species. Let us assume for the sake of argument that B1 and B2+ species have similar physiology and therefore that variation in growth rate reflects mortality. Because nearly all B2+ species grow more rapidly than nearly all B1 species, the sibling-competition hypothesis predicts that the combined selective forces of sibling competition and external mortality factors of any B2+ species are greater than the force of external mortality factors alone for virtually every B1 species. This implies that the selective force of sibling competition is uniformly greater than the variation among species in the selective force of external mortality factors. Although total mortality during the nestling period need not correspond closely to selective mortality (only time-dependent external mortality is relevant, and starvation is not relevant in B1 species), values obtained from B1 and B2+ species are not consistent with the sibling-competition hypothesis. Among seabirds, for example, species with large broods and rapid growth often have higher nesting success than those with broods of one and slow growth (Ricklefs 1969b). Among boobies (*Sula* spp.), three B1 species having slow growth had nestling survival rates of 20, 30, 40, 65, and 70% in five studies, while three rapidly-growing B2+ species had survival rates of 30, 60, 65, and 70% (Nelson 1978),

showing no particular pattern. The most rapidly growing of the Sulidae, the North Atlantic Gannet (*Morus bassanus*), is a B1 species with a nestling survival rate of 80%. We have seen, however, that sibling competition may exert a disproportionately strong influence on growth rate compared to that of externally caused mortality. Hence, comparisons of overall survival rates cannot provide a strong test of the sibling-competition hypothesis, especially when the result is negative.

Although I have found no direct support for the sibling-competition growth-rate hypothesis, available tests of the hypothesis are weak, and the confirmed prediction of slow growth in B1 species would be compelling enough to keep the idea alive, if the observation could not be accounted for by other causes.

*Alternative hypotheses.*—Except for three species of auks (Alcidae), all the B1 species portrayed in Fig. 1 are pelagic seabirds of the orders Procellariiformes, Pelecaniformes, and Charadriiformes. Most of the B2+ species included in the analysis in Fig. 1 belong to the orders Pelecaniformes (but mostly to different families than the B1 species), Ciconiiformes, Charadriiformes, Falconiformes, Strigiformes, and Passeriformes. None of the B2+ species is a pelagic seabird. In the only families with both types of species for which data on growth are available (Sulidae, Laridae, and Alcidae), the B1 species tend to feed on the open seas, while the B2+ species feed closer to shore (e.g. Lack 1968, Nelson 1977). Hence, in comparisons of growth rates between B1 and B2+ species, it is difficult to separate selection by ecological factors associated with feeding from that exerted by sibling competition.

Besides sibling competition, three alternative hypotheses have been proposed to account for the slow growth of pelagic seabirds. First, the single-chick brood, frequent starvation of young, and long intervals before laying replacement clutches suggest that the amount of food that parents can provide may limit the growth rate of the chick in B1 species (Lack 1968, Harris 1977, Nelson 1977). Second, slow growth may reduce the demand for essential nutrients that are in short supply in the diet. Third, differences in growth rates of precocial and altricial birds related to the maturity of their tissues suggests that the chicks of some pelagic seabirds may develop more precocially

than those of inshore feeders (Ricklefs 1979a, b).

Twinning experiments involving two B1 species—the North Atlantic Gannet (Nelson 1964) and the Swallow-tailed Gull (*Creagrus furcatus*) (Harris 1970)—indicated that parents could nourish two offspring adequately, whereas twinning experiments with Procellariiformes and Alcidae (Palmer 1962, Rice and Kenyon 1962, Harris 1966, Nettleship 1972, Lloyd 1977, Summers and Drent 1979) have failed. All species of gulls thus far tested have been able to rear artificially increased numbers of offspring regardless of normal brood size (Harris and Plumb 1965, Harris 1970, M. Coulter unpubl.). Because the North Atlantic Gannet grows as rapidly as most B2+ species, it contradicts the general prediction of the sibling-competition hypothesis to the same degree that it supports arguments against food limitation.

On balance, the evidence suggests that most B1 species do not gather sufficient food for larger broods. But this is not to say that they could not support more rapid growth of their single chicks. Measurements of energy budgets of chicks have revealed that the energy requirement for growth does not exceed 36% of the maximum total energy requirement in the Sooty Tern (*Sterna fuscata*) (Ricklefs and White 1981) and 8% in Leach's Storm-Petrel (*Oceanodroma leucorhoa*) (Ricklefs et al. 1980a). Therefore, more rapid development in these slow-growing species could be achieved with much less energy than that needed to add a second chick to the brood.

The hypothesis that nutrient content of the diet limits growth rate of pelagic seabirds cannot be tested, because diets and requirements have not been analyzed in sufficient detail. Diets of pelagic seabirds are, however, so varied that it is unlikely that nutrition could provide a general explanation for their slow growth.

Among terrestrial species, growth rate is inversely related to the precocity of the chick. This relationship between growth rate and the degree of mobility and homeothermy achieved is mediated by the balance between embryonic and differentiated function in the chick's tissues, particularly skeletal muscle. Seabirds having precocious development, mainly Charadriiformes (Nice 1962), apparently are con-

sistent with this pattern (Ricklefs 1979a). With regard to capacity for homeothermy, neonates of storm-petrels (Hydrobatidae) are as well developed as the neonates of gallinaceous species and waterfowl (Ricklefs et al. 1980b). In this respect, many procellariiform birds would be more properly classified as semi-precocial than they are as semi-altricial (Nice 1962). Howell (1963) has argued similarly for tropicbirds (Phaethontidae), and Ricklefs and White (1981) have found that the Sooty Tern exhibits more precocious development of its skeletal muscles than the closely related Common Tern (S. hirundo), a high-latitude, inshore, rapidly growing, B2+ species.

Species not portrayed in Fig. 1, but that rear broods of one, are found among frugivorous birds of tropical zones (e.g. Snow 1970). The slow growth of their chicks may be related to diet rather than to lack of sibling competition, particularly in view of the fact that one slowly growing, tropical, frugivorous species, the Oilbird (Steatornis caripensis), commonly rears broods of three (Snow 1961, 1962). Furthermore, the slowly growing, B1, frugivorous Bearded Bellbird (Procnias averano) may be much more precocious than other passerines with regard to the development of homeothermy (Snow 1970). The neonate has a thick down, and nestlings are rarely brooded during the day beyond the first day after hatching. In contrast, the B1, insectivorous Cicadabird (Coracina tenuirostris), which grows very slowly, is brooded extensively by the female parent (Marchant 1979). Other members of the genus are B2+, however, and also have prolonged development periods.

*Resolving the relationship between brood size and rate of growth.*—Sibling competition unquestionably may select rapid growth, but estimating selective mortality is liable to be very difficult except in thorough studies with large samples. Experimental synchronization of hatching times might reveal the potential force of sibling competition in the absence of parental manipulation. The results of such selection depend upon the heritability of growth rate and the degree to which variation in growth rate can alter the probability of survival of the chick. These factors may be resolved in part through separate studies on the genetic inheritance of growth rate and on survival in broods whose hatching times are manipulated to mimic variation in growth rate among siblings.

At present, the most testable prediction of the hypothesis that sibling competition is responsible for the differences in growth rate between B1 and B2+ species may be that, among B1 species, rates of growth and selective mortality should be positively correlated. This prediction follows from the required flexibility of growth rate and sensitivity to selective mortality needed to produce the differences between B1 and B2+ species. By looking only at B1 species, one can avoid problems of estimating the strength of sibling competition. Unfortunately, much of the mortality of B1 species, including starvation caused by drastic failure of the food supply, may not be relevant to selection on growth rate. The few data that are available do not support the sibling-competition hypothesis (see above).

At present, there is no direct evidence that variation in rate of mortality, regardless of its cause, is responsible for the diversification of growth rate among species of birds. Yet the difference in growth rate between B1 and B2+ species is an important phenomenon that requires explanation before we can claim to understand the adaptive significance of growth rate. Ultimately, this understanding must include knowledge of the constraints that relate growth rate to other aspects of development, parental care, and the environment during the breeding season.

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