PROXIMATE DETERMINANTS OF SEXUAL DIFFERENCES IN ADULT BODY SIZE

In many animal species, males and females attain different adult body sizes. The processes underlying such sexual differences are undoubtedly complex and may involve an interplay between adaptive, exaptive, and nonadaptive genetic influences on the one hand and environmental variables on the other. For example, large body size may be shown by males because genes for that character confer a selective advantage in that sex (see, e.g., Darwin 1871; Trivers 1972), because such genes have been inherited from an ancestral form (see, e.g., Cheverud et al. 1985), or because local environmental conditions happen to be particularly favorable for the survival and growth of males (see, e.g., Howard 1981; Woolbright 1983, 1989; Halliday and Verrell 1986). The relative importance of such alternative factors is difficult to assess but may be clarified by considering the proximate determinants of sexual differences in body size (hereafter, "sexual size dimorphism," or SSD).

Alternative hypotheses about the causation of SSD make different assumptions and predictions about the stage in ontogeny at which sexual dimorphism in mean adult body size is determined. There are two main possibilities for ontogenetic determination of sexual differences in mean adult body size. (1) This dimorphism is determined mainly by the direction and degree of SSD present at the attainment of sexual maturity. Under this hypothesis, sexual differences in mean adult size are determined by processes operating on juveniles: sex differences either in growth rate or in the age at maturation. (2) Mean adult SSD is determined mainly by sex differences in growth or survival rates during adult life. Under this hypothesis, differences in adult size between the sexes result because adults of one sex grow faster or survive better than adults of the other sex.

The first of these alternatives—SSD at maturation is the prime determinant of dimorphism in mean adult body size—is consistent with a wide range of hypotheses that explain SSD as an adaptation or as a result of phylogenetic conservatism. Even if mean adult SSD is the actual target of selection, this may well be achieved evolutionarily by a modification of the genes that control SSD at maturation. Local environmental conditions as well as genotypic factors may influence the SSD at maturity (Gibbons et al. 1981; Stearns and Koella 1986).

The hypothesis that processes operating during adult life (i.e., after maturation) are the primary determinants of mean adult SSD is also consistent with a wide variety of evolutionary and ecological ideas. For example, such differences in growth or survival rates between the sexes could be influenced by sex-specific adaptations of physiology, morphology, or behavior, in which case the resulting
SSD could be interpreted in terms of adaptation. However, these adaptationist interpretations do not rely on the determination of SSD by processes operating during adult life; they are equally compatible with the determination of SSD earlier in ontogeny, as suggested above. The most interesting case concerns hypotheses that are inconsistent with the determination of mean adult SSD at maturity and that rely instead on processes during adult life as the prime determinants of mean adult SSD. For example, if the reason that adult males are smaller than females is that adult males allocate energy to reproduction rather than to growth (Woolbright 1983, 1989), sexual differences in mean adult SSD must be due primarily to sex differences in growth rate during adult life. Similarly, if the relative mean adult body sizes of males and females are strongly influenced by differential survival rates (as shown by Howard [1981] for *Rana catesbeiana*, and suggested to be of general importance by Halliday and Verrell [1986]), sexual differences in mean adult body sizes must be due mainly to processes operating on adult animals.

Clearly, this hypothesis is not applicable to animal species in which little growth occurs after maturation. Presumably for this reason, the hypothesis that processes acting during adult life determine SSD seems not to have been invoked in regard to birds and mammals, which typically show determinate growth. Nonetheless, growth continues after maturity in many mammals and may differ between the sexes (see, e.g., Jarman 1983; Georgiadis 1985), suggesting that processes operating during adult life might affect SSD in this group as well. Growth also continues after maturity in many invertebrates, although usually not in insects. The relevance of the hypothesis to ectothermic vertebrates is clear, since growth typically continues after maturity in these animals (in reptiles, maturity occurs at about 80% of maximum body length; Andrews 1982). In such a group, sex differences in growth or mortality during adult life could greatly influence observed SSD. There is no doubt that sexual differences in adult growth rates and survival rates do occur (see, e.g., Tinkle 1967; Dunham 1981; Howard 1981) and, hence, that mean adult SSD is affected by these factors. The question is whether these processes acting during adult life are sufficiently widespread, and of sufficient magnitude, to modify significantly any general patterns of SSD established earlier in ontogeny.

This question may be answered by examining the extent of correlation evident between the direction and degree of SSD measured at these two stages of the life history: at maturation and at mean adult body size. If SSD at mean adult body size is highly correlated with SSD at maturity, then nothing that happens after maturity (including sex differences in growth or survival rates during adult life) can have a major influence on broad patterns in mean adult SSD. Alternatively, if proximate effects during adult life are important determinants of mean adult SSD, then little correlation would be expected between SSD at maturity and SSD at mean adult body size. Comparative analyses of this type are best made among related taxa, rather than combining higher taxonomic categories (see, e.g., Clutton-Brock and Harvey 1984; Dunham and Miles 1985). Species can be used as independent units in the analysis because phylogenetic conservatism is irrelevant to the hypothesis. I examined eight data sets: five from reptiles, two from amphibians, and one from fishes (see table 1). Maturity was defined according to morphological rather than
behavioral criteria in each case. All eight lineages included some species in which females grow larger than males and others in which the reverse is true. In 89% of all species within these lineages ($n = 266$), the sex that matured at a larger size also attained a larger average adult size. The relationship between the direction of SSD at maturity and mean adult SSD was much stronger than would be expected by chance ($\chi^2, df = 1, P < 0.01$) in seven of the eight comparisons, with the exception being the small data set for mugilid fishes (see the table).

Analyses of these data in terms of the degree (rather than just the direction) of SSD yielded similar results. I calculated Fitch’s (1981) measure of dimorphism (the ratio of female body length to male body length) for each species in the above lineages. In each case, strong correlations were evident between SSD at maturity and SSD at mean adult size. The proportion of the interspecific variance of mean adult SSD that could be explained by SSD at sexual maturity ranged from 40% to 88% and was significantly greater than expected from the null hypothesis of no relationship between these variables ($P < 0.01$ in each case). The correlation between SSD’s at these two stages of the life cycle tended to be highest for comparisons among congeneric species and slightly lower for comparisons incorporating less closely related taxa (table 1). Stamps (1983) reported a similarly high correlation between SSD at maturity and SSD at mean adult size in 33 lizard species ($r = 0.87$). Using ratios in such analyses may sometimes introduce statistical artifacts (see, e.g., Packard and Boardman 1988), but an alternative analysis of these data using residuals from general regressions gave results essentially identical to those based on ratios ($r = 0.61$ to 0.95). Visual inspection of these regressions revealed no obvious departures from linearity. Intercepts of the regression equations were generally close to zero: the difference between SSD’s at these two stages of the life history averaged less than 3% (range of means, 0% to 8%). Slopes of the reduced major-axis regressions were not significantly different

### Table 1

The Relationship between Sexual Size Dimorphism (SSD) at the Time of Sexual Maturation and at Mean Adult Body Size

<table>
<thead>
<tr>
<th>Lineage</th>
<th>No. of Species</th>
<th>Proportion with SSD in Same Direction at Each Stage</th>
<th>Correlation Coefficient between SSD at Two Stages</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lizards</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anolis</td>
<td>65</td>
<td>0.95**</td>
<td>0.93**</td>
<td>Fitch 1981</td>
</tr>
<tr>
<td>Sceloporus</td>
<td>47</td>
<td>0.77**</td>
<td>0.82**</td>
<td>Fitch 1981</td>
</tr>
<tr>
<td>Gekkonidae</td>
<td>41</td>
<td>0.88**</td>
<td>0.63**</td>
<td>Fitch 1981</td>
</tr>
<tr>
<td>Snakes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Natricinae</td>
<td>36</td>
<td>0.89**</td>
<td>0.63**</td>
<td>Fitch 1981</td>
</tr>
<tr>
<td>Turtles</td>
<td>31</td>
<td>0.94**</td>
<td>0.90**</td>
<td>Fitch 1981</td>
</tr>
<tr>
<td>Frogs</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chinese spp.</td>
<td>17</td>
<td>0.88**</td>
<td>0.93**</td>
<td>Liu 1950</td>
</tr>
<tr>
<td>Neotropical hylids</td>
<td>19</td>
<td>0.95**</td>
<td>0.92**</td>
<td>Duellman 1970</td>
</tr>
<tr>
<td>Fishes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mugilids</td>
<td>10</td>
<td>0.60</td>
<td>0.83**</td>
<td>Grant et al. 1977</td>
</tr>
</tbody>
</table>

** $P < 0.01$. 
from 1.0 in six of these eight data sets, suggesting that dimorphism in body size tends to be similar at maturity and at mean adult size. Overall, it is clear that the direction of adult SSD in these animals is determined primarily by SSD at maturity. This result should also apply to endothermic vertebrates, which typically show relatively little growth after maturity. Correlations between the degree of SSD at these two stages of the life cycle are less than 1.0 (table), possibly because of error in the data or because of post-maturational sex differences in growth rates or survival rates affecting adult SSD.

The hypothesis that SSD is primarily determined by sex differences in growth rate or survival rate during adult life (in species in which growth continues after maturation) also makes at least one prediction that can be readily evaluated. Because fighting is risky and energetically expensive, sex differences in growth or survival rate during adult life in species with male combat should tend to reduce mean adult body size in males relative to that in females, compared with the SSD expected if combat did not occur. Hence, all else being equal, one might expect a decrease in the ratio of male size to female size in taxa with male combat compared with taxa without this behavior. This prediction relies on a lack of systematic shifts in other factors (such as maternal investment) concurrent with the evolution of male combat, and such potential confounding factors deserve further investigation. Nonetheless, it is interesting to note that the prediction derived from the hypothesis of post-maturational determination of adult SSD is directly contrary to that from Darwin’s (1871) adaptationist hypothesis that male-male combat favors the evolution of large body size in males. Darwin’s hypothesis does not specify at what point in ontogeny this SSD should be established; it simply predicts that males should be large in species with male combat. The success of the Darwinian prediction among both ectotherms and endotherms (see, e.g., Darwin 1871; Trivers 1972; Wiley 1974; Clutton-Brock et al. 1977; Shine 1978, 1979; Wells 1978; Berry and Shine 1980; Veuille and Rouault 1980; Crespi 1986) is consistent with the notion that broad patterns of SSD among animal species are set at maturity, with only minor modification by differential growth or survival rates of adult males and females.

Overall, the patterns documented in this study suggest that differences in prematurational growth, age at maturity, or both are the primary determinants of sexual dimorphism in adult body size. Post-maturational differences in growth or survival rates between the sexes are not totally irrelevant but probably influence the direction (as opposed to the degree) of adult SSD only rarely. Influences on SSD at maturity thus deserve further investigation. A clarification of the extent of phenotypic flexibility in SSD at maturity (as in Gibbons et al. 1981) would be particularly valuable: if proximate environmental factors are important determinants of SSD at maturity, interpretations of mean adult SSD in terms of adaptation may be in error (Ralls and Harvey 1985).

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