instances, solutions, into classical problems of skull design and evolution. The final two chapters address mechanisms of cranial evolution. Chapter 9 (Emerson and Bramble) emphasizes the importance of absolute size and size change for the evolution of cranial design and function, whereas chapter 10 (Liem) is primarily concerned with the interplay of ecology and morphology in cranial diversification. As in earlier volumes, chapters in this volume generally are intended to be synthetic overviews of a given topic, not exhaustive reviews. However, in chapter 2, on suspension feeding, and chapter 5, on locomotion, a more comprehensive treatment proved to be the only effective way to synthesize a widely scattered literature.

As an integrated whole, this volume provides an overview of a number of important and diverse functions of the vertebrate skull and relates these functions to patterns of cranial development and growth, as well as to ecological and evolutionary constraints, processes, and opportunities. In doing so, it offers a functional context for the treatments of skull development and diversity provided in the preceding two volumes. It is undeniably the most eclectic of the three volumes in this series. We would argue, however, that this is indicative of the field of functional and evolutionary morphology itself, where there is a wide range of research questions, analytical paradigms, and methodological approaches and techniques (Liem and Wake 1985). The volume does not cover all of these paradigms or approaches, or even all cranial functions, among other reasons because to do so would take several volumes in itself. More important, function and evolutionary mechanisms are arguably the most poorly understood of the three main areas of cranial biology covered in this series, and a truly comprehensive treatment is not possible at this time, however desirable. Rather, these chapters are intended to convey a sense of the range of topics, paradigms, and approaches that are being considered at present; in other words, what can or even should be done, and how. In this regard, many of the chapters focus on the authors' own work, as examples of the analysis of major problems. Consequently, the topics considered give an accurate assessment and representation of current interest and knowledge of skull function.

As in earlier volumes, we are pleased to thank the authors of the present volume for their excellent contributions.

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1

Mechanisms of Skull Diversity and Evolution

James Hanken and Brian K. Hall

INTRODUCTION

This volume attests to the breadth of current interest and inquiry into the function and evolution of the vertebrate skull. One cannot come away from reading the following chapters without a great appreciation of the adaptive diversity of cranial form and function. They also reveal the need to consider the function and evolution of the skull in their proper context, whether this be the suite of nonskeletal cranial tissues with which the skull is functionally, anatomically, and developmentally integrated, or related aspects of behavior, physiology, and ecology.

We begin this broad, interdisciplinary approach with a brief consideration of the mechanisms for the evolution of cranial diversity. In particular, we concern ourselves with two aspects that we consider to be particularly important: the role of development, and particularly the embryonic neural crest from which much of the skull is derived; and the nature of intraspecific variability, and its role in morphological diversification. Both aspects are relatively poorly known in the context of cranial evolution, but we believe that a comprehensive understanding of the mechanisms of morphological diversification of the skull—indeed, of any structure—must incorporate them. In focusing on them here, we also hope to underscore the need for more work in each area.

SKULL DEVELOPMENT

The diversity of form displayed by the vertebrate skull is, on the one hand, great (compare the skull of the elephant with that of a shrew, a snake with that of a bird) and, on the other hand, limited—all vertebrate skulls are built upon the same basic plan. The fundamental structural similarity is a reflection of conservative developmental processes within and among vertebrate taxa. In this section, we deal with those fundamental developmental processes—the neural crest origin of much of the vertebrate skull,

the epigenetic evocation of skull differentiation through tissue (epithelial-mesenchymal) and functional interactions, and the mechanisms for cranial patterning. It is these processes and their stability/modification during vertebrate phylogeny that provide the ontogenetic basis for both phylogenetic stability and for diversity of the vertebrate skull.

The Contribution of the Neural Crest to the Skull

The neural crest was first described by Wilhelm His in 1868 when he reported the existence of a specialized zone of cells between the neural and epidermal ectoderms at the boundary of the future neural tube (i.e., in the crests of the neural folds) in neurula-stage chick embryos. In the years since its discovery, our view of the neural crest and of neural crest cells has progressed from surprise, through mistrust, ridicule, heresy, ignorance, indifference, reawakening, reevaluation, reinvestigation, and orthodoxy, to the current enthronement of the neural crest as a quintessential vertebrate character (Hall 1988b; Gans 1993). As these authors indicate: "this embryonic tissue [the neural crest] (and the ectodermal neurogenic placodes) represents the common denominator for vertebrate synapomorphies" (Gans 1993 2:17); "The neural crest as part and parcel of a dorsal nerve cord and notochord is a quintessential vertebrate characteristic, or according to some *the* quintessential vertebrate characteristic" (Hall 1988b, 19–20).

The first challenge to orthodoxy, especially with regard to the origin and development of the skull, came from the studies of Kastschenko (1888) and Goronowitsch (1892, 1893a, b) who argued that some cranial mesenchyme in shark, fish, and bird embryos arose not from mesoderm, but from cells derived from the neural crest (de Beer 1947). Although unexpected, these studies did not occasion the controversy that followed Platt's (1893, 1897) assertion that the visceral arch cartilages of *Necturus* also arose from ectoderm, either neural crest or placodal head ectoderm. This heretical notion of an ectodermal origin of a skeletal (mesodermal) tissue, and its challenge to the firm grasp that the germ layer theory held on late-nineteenth-century biology and biologists, has been discussed elsewhere (Oppenheimer 1940; de Beer 1947; Hörstadius 1950; Hall 1988b).

Fortunately, prevailing orthodoxy did not discourage persistent and enquiring experimental embryologists from pursuing the problem of the origin of the head skeleton. Platt, on the basis of differences in yolk between ecto- and mesodermal cells, assigned the origin of the visceral arch cartilages to placodal ectoderm. A more comprehensive analysis of *Ambystoma jeffersonianum* by Landacre (1921) assigned the neural crest as the chondrogenic source; provided the first mapping of individual skeletal elements (the anterior aspects of the cranial base [trabeculae cranii] and visceral arch cartilages, except the second basibranchial, derived from the

neural crest; the balance of the skull from mesoderm); and gave the first indication that the viscerocranial skeleton had a dual origin, arising in part from neural crest–derived and in part from mesodermally derived mesenchyme.

Descriptive studies being equivocal, Stone (1922, 1926, 1929), Raven (1931, 1936) and Harrison (1935a–c, 1938) extirpated neural crest from, and exchanged neural crests between, urodele and frog embryos. Using these techniques they mapped the migratory pathways of neural crest cells and confirmed the neural crest origins of viscerocranial cartilages. They further demonstrated that trunk neural crest cells could not substitute for extirpated cranial cells to prevent skeletal deficiencies—the now well-known dichotomy between the skeletogenic and odontogenic cranial neural crest and nonskeletogenic and nonodontogenic trunk neural crest (but see Smith and Hall [1990] for a reevaluation of this dichotomy).

Subsequent detailed analysis by Hörstadius and Sellman (1941, 1946), Chibon (1964, 1966, 1967, 1974) and Sadaghiani and Thiébaud (1987) have mapped the neural crest origin of the larval skulls and visceral arch skeletons of two urodeles (*Ambystoma mexicanum, Pleurodeles waltl*) and one anuran (*Xenopus laevis*). This neural crest contribution to amphibian skull development has now been extended to representatives of all classes of vertebrates (see Hall 1987b, 1988b for summaries). The major (in some cases the only) studies for each taxon are as follows:

Cyclostomes (lampreys): Damas (1944, 1951); Johnels (1948); Newth (1950, 1951, 1956); Langille and Hall (1986, 1988b, 1989b).
Teleosts: Matsumoto et al. (1983); Langille and Hall (1987, 1988a)
Reptiles: Toerien (1965a, b) (For the neural crest origin of cranial mesenchyme in turtles and alligators see Meier and Packard [1984] and Ferguson [1984, 1985].)
Birds: Johnston (1966), Le Lièvre (1971a, b, 1974, 1976, 1978); Le Lièvre and Le Douarin 1974, 1975); Noden (1978a, 1984); Johnston et al. (1979)
Mammals: Johnston and Hazelton (1972); Johnston et al. (1981); Tan and Morriss-Kay (1986); Morriss-Kay and Tan (1987); Smits-van Prooije et al. (1987, 1988) (For references on the neural crest origin of cranial mesenchyme in mammals see Hall [1988b, 62–72].)

Rather than provide an exhaustive list of elements of the skull and visceral skeleton derived from the neural crest (for which the papers and reviews listed above and Langille and Hall's chapter in volume 1 may be consulted), we utilize the scheme shown in figure 1.1, which depicts the regionalization (rostro-caudal extent) of the neural crest that forms the chondrocranial and visceral arch skeletons in a variety of vertebrates. It is very clear that regionalization of the skeletogenic neural crest is a highly

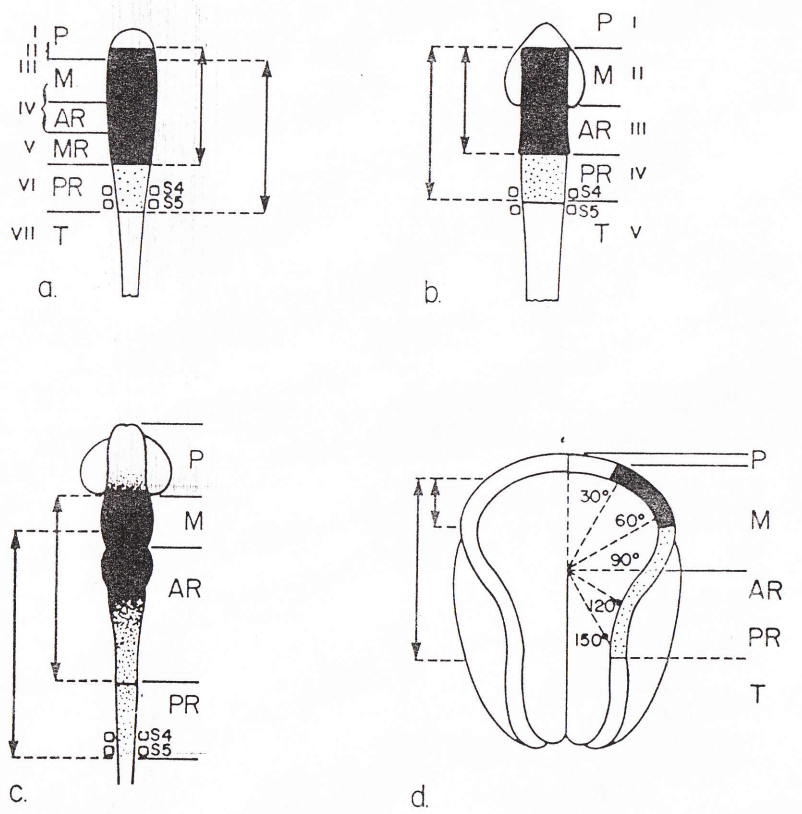

Fig. 1.1. Constancy of regionalization of the skeletogenic neural crest in (a) *Petromyzon marinus* (a lamprey), (b) *Oryzias latipes* (the Japanese medaka, a teleost fish), (c) *Gallus domesticus* (the common fowl), and (d) *Ambystoma mexicanum* and *Pleurodeles waltl* (two urodele amphibians). Chondrocranial neural crest is shown in black, viscerocranial neural crest is stippled; both are demarked by double-headed arrows. The skeletogenic cranial neural crest extends from the anterior mesencephalon (mid-prosencephalon in *P. marinus*) caudad to the level of somites 4 or 5 (S5, S5). Other abbreviations: AR, anterior rhombencephalon; M, mesencephalon; MR, mid-rhombencephalon; P, prosencephalon; PR, posterior rhombencephalon; T, trunk neural crest. Roman numerals I–VII in (a) and I–V in (b) refer to boundaries of regions excised to generate fate maps. The angles from the midline in (d) represent sectors of neural crest excised from *P. waltl* by Chibon (1966, 1967), projected onto the fate map for *A. mexicanum*. Reproduced from Hall (1988b) with permission of the publisher.

TABLE 1.1 Regionalization of the skeletogenic neural crest in the lamprey, *Petromyzon marinus*, in relation to the rostro-caudal regions I to VII shown in figure 1.

	Regions						
	I	II	III	IV	V	VI	VII
Trabeculae	✓	✓	✓	✓	✓		
Branchial arches							
1–3			✓✓	✓✓	✓✓	✓	
4 & 5			✓	✓✓	✓✓	✓✓	
6 & 7				✓	✓✓	✓✓	

Note: Single checks indicate only a minor contribution from this region.

conserved vertebrate feature; skeletogenic neural crest extends in all taxa from the anterior mesencephalon caudad to the level of somite 5. Although chondrocranial neural crest lies rostral to viscerocranial crest, the two regions overlap substantially. Regionalization is also evident *within* the chondrocranial and viscerocranial neural crest; more anterior elements arise from rostral, and more posterior elements from caudal, neural crest (table 1.1). (We have, however, minimal knowledge of how the neural crest itself is specified—see Hall [1988b] and Thomson [1988] for some discussion).

We take this rostro-caudal regionalization of the neural crest, the source of much of the cranial skeleton, to be a fundamental, ancient feature (synapomorphy) of the vertebrates (Langille and Hall 1989a). This dictates how we currently evaluate developmental mechanisms underlying diversity and evolution of the vertebrate skull.

Tissue Interactions in Skull Development

The skull arises epigenetically, largely through cell and tissue (epithelial-mesenchymal) interactions and through the action of adjacent nonskeletal tissues. It was in the pioneering study by Hörstadius and Sellman (1946) mapping the skeletogenic neural crest of *Ambystoma mexicanum* that evidence for induction of cranial cartilage by epithelia was obtained, viz. neural crest–derived visceral arch cartilage by pharyngeal endoderm and mesodermally derived otic capsule cartilage by otic vesicle epithelium. Such interactions have chiefly been studied in anuran amphibians (Holtfreter 1968; Cusimano-Carollo 1963, 1969, 1972; Cusimano et al. 1962), in urodeles [Wagner 1949; Okada 1955; Rollhaüser-ter-Horst 1977; Cassin and Capuron 1979; Corsin 1975; Drews et al. 1972; Epperlein and Lehmann 1975; Minuth and Grunz 1980; Graveson and Armstrong 1987), and in birds (Schowing 1968; Tyler and Hall 1977; Hall and Tremaine 1979; Bee and Thorogood 1980; Thorogood 1981; Thorogood and Smith 1984) (see Hall 1987b, 1988a for reviews). As known in greatest detail for the embryonic chick, every cartilage and bone in the developing skull

TABLE 1.2 Epithelia involved in the epithelial-mesenchymal interactions required for differentiation of the components of the viscerocranial and chondrocranial skeletons of the embryonic chick

Skeletal component	Epithelium
angular	mandibular arch epithelium
basisphenoid	rhombencephalon, notochord
dentary	mandibular arch epithelium
frontal	prosencephalon, mesencephalon, cranial ectoderm
maxilla	maxillary arch epithelium
Meckel's cartilage	dorsal cranial ectoderm
occipital	rhombencephalon
otic capsular cartilage	otic vesicle epithelium
palatine	palatal epithelium
parasphenoid	notochord
parietal	mesencephalon, rhombencephalon
pterygoid	palatal epithelium
scleral cartilage	pigmented retinal epithelium
scleral ossicles	scleral epithelial papillae
squamosal	mesencephalon
surangular	mandibular arch epithelium

Note: See Hall (1987b) for information on the timing of these interactions and for the primary literature.

depends for its differentiation on one or more epithelial-mesenchymal interactions (table 1.2). Discussion of the mechanism of these interactions, which is outside the scope of this paper, may be found in Hall (1987a, 1988a, 1989).

The timing of these epithelial-mesenchymal interactions is not constant throughout the vertebrates for, on the one hand, neural crest cells at different stages in their migration, and on the other hand, different epithelia, are involved in the interactions. That this is so for different skeletal elements in the same embryo is not surprising (table 1.2). For example, the dentary and the frontal arise from different neural crest cell populations which are differentially localized along the neural axis, migrate along different paths, and settle at different sites. Inevitably, these populations encounter different epithelial environments—cranial ectoderm, pharyngeal endoderm, and mandibular arch epithelium encountered by dentary mesenchyme; cranial ectoderm and mesencephalic neural ectoderm encountered by frontal mesenchyme.

However, divergence in time, space, and components involved in epithelial-mesenchymal interactions is also seen when development of the same skeletal element is compared across taxa, the best-documented examples being those interactions involved in the differentiation of Meckel's cartilage in urodele and anuran amphibians, birds, and mammals (Hall 1984, 1987b). Here we have, in representatives of each of these groups, a

homologous structure arising from neural crest cells from the same rostrocaudal region of the neural crest, which follow similar migration pathways and encounter similar epithelia, but where the epithelium required for chondroblast differentiation varies from group to group. The active epithelium is cranial ectoderm adjacent to the neural tube in the embryonic chick, pharyngeal endoderm in anuran and urodele amphibians, and mandibular arch epithelium in the fetal mouse (fig. 1.2). What has altered or shifted during the evolution of amphibians, birds, and mammals apparently is not the neural crest component of the epithelial-mesenchymal interaction, but rather the epithelium providing the signal to which the neural crest–derived cells respond. Changes in the epithelia that evoke chondrogenic differentiation must relate to aspects of skull development other than basic structure, for basic chondrocranial form, including that of Meckel's cartilage, is largely conserved throughout the vertebrates.

Why should development of Meckel's cartilage in avian embryos require that the chondroblasts be specified earlier than in mammals or amphibians? Does the answer lie (a) in the amount of *in ovo* growth of Meckel's cartilage required to produce an elongated beak capable of func-

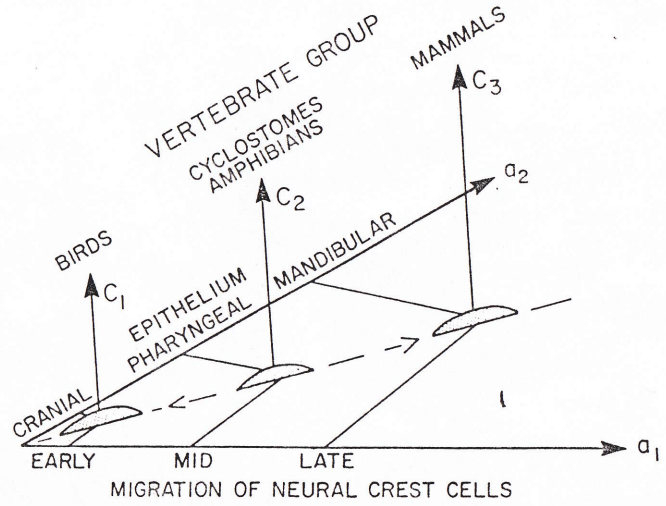

Fig. 1.2. Variation in the timing of the epithelial-mesenchymal interaction that initiates differentiation of Meckel's cartilage in amphibians, birds, and mammals, in terms of both the time during neural crest cell migration when the interaction occurs (a_1) and the epithelium involved in the interaction (a_2). If the amphibian condition is taken as primitive for extant tetrapods, then the interaction has been moved earlier in development during the evolution of birds (C_1) and later in development during the evolution of mammals (C_3). Modified from Hall (1984).

tioning immediately after hatching (Meckel's cartilage "controls" mandibular growth; Diewert 1982), extension of the time between epithelial-mesenchymal interactions and differentiation allowing more cells to be specified as chondroblasts, (b) in the fact that Meckel's cartilage persists unossified in many birds (except for the retroarticular process) but is transformed into ligaments or translated into ear ossicles in mammals, or (c) in some other aspect of avian mandibular development?

A parallel evolutionary change to that just described for Meckel's cartilage has been revealed by experiments transplanting facial ectoderm of anuran and urodelan embryos. The inability of anuran embryos to form a balancer and of urodele embryos to form an adhesive organ results from evolutionary changes in the epithelial rather than the mesenchymal component of the interactions. Thus, urodele epithelium can elicit development of a balancer from anuran mesenchyme and anuran epithelium can elicit development of an adhesive organ from urodelan mesenchyme (Spemann 1938, 350–366).

As epithelial-mesenchymal interactions determine the number of mesenchymal cells that will be able to differentiate into a particular cell type (chondroblasts) to make a particular skeletal element (Meckel's cartilage) at a particular site (the mandibular arch; Hall 1988a), the timing, duration, and/or strength of the interaction influences when the skeletal element arises in ontogeny and the extent of its subsequent growth. Variations in timing, duration, and strength of these interactions provide mechanisms for evolutionary change through heterochrony (Hall 1984; Smith and Hall 1990). Epithelial-mesenchymal interactions, however, are not isolated, single events. A cascade of inductive interactions has been demonstrated in the frog *Discoglossus pictus,* and in the urodele *Pleurodeles waltl,* such that, during jaw development, specification of chondroblasts is required before osteoblasts can arise, which in turn must be specified before teeth can form (see summary in Hall 1987b, 1988b). We do not know the cascades in other vertebrates (and indeed have only brief glimpses of the amphibian cascade) but it is clear that alteration in timing of the first step, specification of chondroblasts, could have a ripple effect on the subsequent development of other tissues. Alterations in the timing of even one epithelial-mesenchymal interaction involved in development of even a single skull element, must, on the one hand, be assessed in the context of such "epigenetic cascades," and on the other hand, provide a developmental mechanism for generating diversity during skull development (Hall 1988b; Smith and Hall 1990).

Patterning of the Skull

What are the consequences for patterning of the skull by epigenetic evocation of mesodermal and neural crest cells?

The knowledge that (a) key vertebrate characteristics are a consequence of the evolution of the neural crest and ectodermal placodes (because these characters either develop directly from, or under the influence of, neural crest cells in the epigenetic cascades noted earlier) and that (b) the cranial mesenchyme, much of the skull, and the viscerocranial skeleton are formed exclusively from neural crest cells, leads to the obvious corollary that (c) the vertebrate head is a new structure, a neomorph (Gans and Northcutt 1983; Northcutt and Gans 1983; Gans 1993). Thus, the classic notion, the origins of which lie in Goethe's famous 1790 inspiration while gazing at a sheep's skull in a Venetian cemetery, that the head and skull represent the anterior continuation of a segmented neuronal, muscular, and vertebral pattern seen in the trunk, is no longer tenable. In fact, the sequence is quite the reverse, for, as emphasized by Gans (1993) and Smith and Hall (1990), a postotic skull, and indeed, a trunk skeleton consisting of ossified vertebrae, arose only with the evolution of the jawed vertebrates. For discussions of our changing views on head segmentation see Huxley (1898), Russell (1916), de Beer (1937), Hall and Hanken (1985), and Gans (1993).

Cranial segmentation is confined to the neuromeres and myogenic somitomeres (Jacobson 1987). There is no evidence for segmentation of the cranial, nor indeed of the trunk neural crest, although as indicated in figure 1.1, cranial neural crest is regionalized on the basis of the arches to which mesenchyme migrates and of the neuro- and vicerocranial skeletal elements that arise from these regions: the neural crest is patterned but not segmented.

The recent mapping of homeobox genes in the mouse head indicates that *En-1* is distributed throughout the rostro-caudal extent of the neural tube, *En-2* is confined to mesencephalic and rostral metencephalic neural tube, and *Hox 1.5* and *2.1* do not extend more rostrally than the pre- and postotic myelencephalon respectively (Holland 1988; Holland and Hogan 1988). Homeobox gene distribution is regionalized and segmented, and this regionalization extends to the neural crest. No regionalization of the cranial mesoderm, however, has been found.

Does regionalization of the neural crest reflect a restricted morphogenetic capability with respect to ability to form specific components of the skull? Perhaps the situation is like that of the developing nervous system during neurulation, in which cells along the entire length of the neural axis undergo an equivalent cytodifferentiation into neurons, but where those neurons, because of inductions related to their position along the neural axis, organize themselves into distinct regions of the nervous system: fore-, mid-, hindbrain and spinal cord. If neural crest cells share this property with neuronal cells, then neural crest cells along the rostro-caudal extent of the cranial neural crest may possess a shared capability of cytodif-

ferentiation into chondroblasts (which they do) but a restricted capability for the chondrocranial elements that they could form. Alternatively, morphogenetic specificity may be resident within these cells before they leave the neural tube, as the evidence of Wagner (1949), Hörstadius (1950), and Noden (1978a, 1984) suggests (see Hörstadius [1950] and Thorogood [1993] for discussions of these alternatives).

Thorogood et al. (1986) and Thorogood (1988, 1993) have demonstrated a spatial and temporal heterogeneity in the distribution of type II collagen along the neuroepithelium that corresponds to the sites of chondrocranial capsule formation and have argued that neural crest cells encountering this type II collagen during migration will receive the primary signal in specification of chondrocranial patterning, i.e., that irrespective of level along the cranial neural axis, neural cells that become trapped in particular sites should be able to produce a chondrocranial element appropriate to that site. As Thorogood puts it: "The (neuro) epithelium simply specifies, to a responsive mesenchyme where and when cartilage should form. Viewed in this way, differential lineage composition of 'homologous' skeletal elements, in terms of ectomesenchymal or mesodermal contributions, becomes largely irrelevant" (Thorogood 1988, 152).

This interpretation assumes no site-specificity in the epithelial-mesenchymal interactions that initiate or promote chondrogenesis, an issue that is currently unresolved (Hall 1986, 1988c, 1991). In fact, much of the facial ectoderm, at least in the avian embryo, arises from the most rostral region of the prosencephalic neural folds (Couly and Le Douarin 1987, 1988), a site consistent with the possibility that facial ectoderm may itself be regionalized during primary embryonic induction of the neural ectoderm.

Thus, there appears to be a hierarchy of epigenetic processes explaining both stability of basic skull form and diversity upon that basic theme. The localization of type II collagen provides a means of trapping cells that are migrating from the regionalized neural crest, thereby ensuring stability of basic chondrocranial form across the vertebrates. A diversity of subsequent tissue interactions, and their modification through heterochrony, permits variation on that basic structural scheme.

INTRASPECIFIC VARIATION

The Relevance of Intraspecific Variation

As emphasized in the previous section, knowledge of development offers valuable insights into the organismal mechanisms that may in some instances constrain, and in other instances facilitate, morphological diversification. Yet, an understanding of how cranial morphology evolves requires

more than simply identifying embryonic tissues and eludicating developmental processes, or even using this information to delineate the potential range of organic form. Developmental events must also be linked to interspecific differences. In other words, it is also necessary to resolve how structural variation at the level of developmental processes translates into morphological variation among taxa. We consider this to be one of the most important challenges facing future investigators of the vertebrate skull. Valuable information may come from studies of the nature, extent, and causes of intraspecific variation in natural populations.

Remarkably little, however, is known about naturally occurring intraspecific variation in the cranium. This is especially surprising in view of the enormous literature dealing with, on the one hand, mechanisms of skull development and growth (see volume 1, this series), and, on the other, patterns of cranial diversity among taxa (volume 2). Moreover, much of the data that exist have been amassed in the context of taxonomic investigations where the primary interest has been to define the limits of variability for the purposes of delimiting taxa; there have been few studies of variation per se, including its nature, range, developmental/genetic basis, or ecological/evolutionary causes.

Perhaps the variation that is of most interest and relevance in the context of mechanisms of cranial diversity involves so-called discontinuous variants (Falconer 1981); that is, variation not simply involving mensural variables but characterized by discrete and often large-scale differences between variants, and which is largely independent of sex, age, and adult body size. Several examples of discontinuous, intraspecific variation involving cranial characters are listed in table 1.3. This list is neither comprehensive nor necessarily representative of the range or nature of variation within particular groups or among vertebrates generally. It is simply intended to convey some idea of the potentially enormous variability that exists in nature. Most of the examples, documented only in the last 10–20 years, differ widely with respect to both the magnitude of morphological differences involved and their ecological and evolutionary significance, but they can be readily divided among three distinct classes of variation—gross malformations, epigenetic polymorphisms, and trophic polymorphisms.

Classes of Intraspecific Variation

Type 1: Gross Malformations. This class of variation includes teratologies, deformities, and other gross malformations that typically lie outside a species' developmental "norm of reaction" (Schmalhausen 1949) (table 1.3). An excellent example is cleft palate, a common congenital malformation in humans that is also found in other vertebrates, including many squamates (Bellairs and Boyd 1957; Bellairs 1965). Such variants are of considerable biomedical importance when occurring in humans, and they

TABLE 1.3 Intraspecific variation involving discontinuous characters in the skull

Variant type/taxon	Variable character	Reference
Type 1: Gross malformations		
Caretta caretta (R)[1]	partial cyclopia, monorhinia	Bellairs 1983
Natrix maura (R)	cyclopia	in den Bosch and Musters 1987
Eunectes murinus, Natrix natrix, Vipera berus, Lacerta spp. (R)	cleft palate	Bellairs 1965; Bellairs and Boyd 1957
Type 2: Epigenetic polymorphisms		
Amia calva (F)	parietal bones paired or fused; number of postorbital, infraorbital, and extrascapular bones; frequent asymmetry	Jain 1985
Osteolepis macrolepidatus (F)	numbers of parietal shields and intertemporal bones	Graham-Smith 1978
Salvelinus alpinus (F)	number of foramina; bone fusion	Medvedeva and Savvaitova 1981
Bolitoglossa occidentalis (A)	prefrontal bone present or absent	Alberch 1983
Notophthalmus viridescens (A)	branchial cartilages present or absent; atavisic epibranchials	Reilly 1987; Reilly and Lauder 1988
Rhyacotriton olympicus (A)	nasal bone present or absent	Wake 1980
Taricha granulosa (A)	branchial cartilages present or absent	Reilly 1987
Thorius spp. (A)	nasal, prefrontal, and septomaxilla bones present or absent; asymmetry	Hanken 1984
Geochelone radiata (R)	parietal foramen present or absent	Crumly 1982
Phrynosoma solare (R)	epipterygoid bone present or absent	Axtell 1986
Megapodius freycinet, Chamaepetes goudotii, Crax pauxi, C. rubra, Penelope purpurascens, Tympanuchus phasianellus, Callipepla californicus, Acryllium vulturinum, Guttera plumifera, G. pucherani, Numida meleagris, Opisthocomus hoazin, Corythaixoides leucogaster, Crinifer piscator, Tauraco erythrolophus, T. hartlaubi, T. schalowi, Guira guira, Centropus goliath (B)	number and pattern of scleral ossicles	de Queiroz and Good 1988
Erethizon dorsatum (M)	number of interparietal (wormian) and squamosal bones; squamosal foramen present or absent; masticatory and buccinator foramina separate or joined	Sutton 1972
Homo sapiens (M)	number of foramina, bone fusions, etc. (29 variants); number of interparietal bones	Berry and Berry 1967; Berry 1968; Pal 1987; Pal et al. 1984
Mus musculus (M)	number of foramina, bone fusions, etc. (24 variants)	Berry 1968; Berry and Searle 1963
Sciurus carolinensis, Cavia porcellus, Lemmus lemmus, Microtus agrestis, Peromyscus maniculatus, Micromys minutus, Rattus spp. (M)	number of foramina, bone fusions, etc. (10–22 variants per species)	Berry and Searle 1963
Type 3: Trophic polymorphisms		
Cichlasoma managuense (F)	skull dimorphism: obtusorostral vs. acutorostral[2]	Meyer 1987
Cichlasoma minckleyi, C. citrinellum, C. haitensis (F)	skull and tooth dimorphism: papilliform vs. molariform	Sage and Selander 1975; Liem and Kaufman 1984; Kornfield et al. 1982; Meyer 1989, 1990a, b, 1991
Ilyodon spp. (F)	skull dimorphism: tapered vs. blunt	Turner and Grosse 1980; Grudzien and Turner 1984a, b
Poeciliopsis sp. (F)	dentary polymorphism	Vrijenhoek 1978; Levinton 1988
Saccodon spp. (F)	jaw and tooth polymorphism: 3–4 morphs per species	Roberts 1974
Ambystoma tigrinum (A)	larval skull dimorphism: cannibal vs. typical	Collins and Cheek 1983; Powers 1907; Pierce et al. 1983; Rose and Armentrout 1976; Lannoo and Bachman 1984
Scaphiopus spp. (A)	skull dimorphism: cannibal vs. typical (includes keratinized mouth parts)	Orton 1954; Bragg 1965; Bragg and Bragg 1959; Pomeroy 1981
Chondrohierax uncinatus (B)	bill size dimorphism: large vs. small	Smith and Temple 1982
Pyrenestes spp. (B)	bill size dimorphism: large vs. small	Smith 1987, 1990a, b, c
Peromyscus maniculatus (M)	mandible dimorphism: woodland vs. grassland	Holbrook 1982

[1] Abbreviations denote major group, as follows: A, amphibian; B, bird; F, jawed fish; M, mammal; R, reptile.
[2] Variation between morphs is not discontinuous. Instead, morphs denote extreme endpoints of a morphological continuum.

often provide useful models for examining basic processes underlying skull development generally. However, the often considerable morphological changes involved typically confer a tremendous decrease in fitness and have no obvious present or even potential adaptive value. Thus, variants of this type are of doubtful prospective significance and are unlikely vehicles of subsequent evolutionary change.

Type 2: Epigenetic Polymorphisms. The second class of discontinuous variation involves typically subtle variability of one or a few characters per individual in which the range of character states lies within, and in fact may define, the norm of reaction for the species or higher group involved (table 1.3). This variation is the result of a complex interplay between genome and environment, both of which exert a strong influence in determining the structure(s) involved. Instances of such variation have been termed "epigenetic polymorphisms" (Berry and Searle 1963), in recognition of the predominant role of developmental processes in mediating the interaction between genome and environment and in establishing the discontinuous nature of the variation. Although the mode of inheritance has been documented in relatively few instances, discontinuous variation that is characteristic of epigenetic polymorphisms is generally believed to reflect a developmental threshold acting on a continuously variable genetic liability (Berry 1968; Falconer 1981; fig. 1.3). Examples include the variable presence or absence of particular bones (e.g., nasal in the Olympic salamander, *Rhyacotriton olympicus;* Wake 1980; fig. 1.4), occasional fusion of typically paired elements (e.g., parietals in the bowfin, *Amia calva;* Jain 1985; fig. 1.5), and variation in the absolute number of bones or foramina (e.g., the accessory maxillary foramen in the house mouse, *Mus musculus;* Berry and Searle 1963).

Variability of this kind is important in several respects. First is the very obvious fact that it demonstrates the capacity, and possibly even the propensity, for natural populations to sustain sometimes substantial intraspecific variation in discontinuous characters. In certain instances, alternate character states are as different as those that distinguish species or even higher taxa. This variation represents a pool of discrete, alternate phenotypes that may provide the basis for subsequent evolutionary change and morphological diversification. Moreover, because the initial appearance of variant phenotypes may typically be regarded as incidental to subtle changes in either the genome or the environment and unrelated to current adaptation (equals "exaptation" of Gould and Vrba [1982]), the problem of explaining the initial evolution of novel morphologies, especially in the context of natural selection, is minimized. At the same time, should a novel variant prove to be selectively advantageous, all the ingredients are on hand for a transition, via genetic assimilation, from the chance occurrence

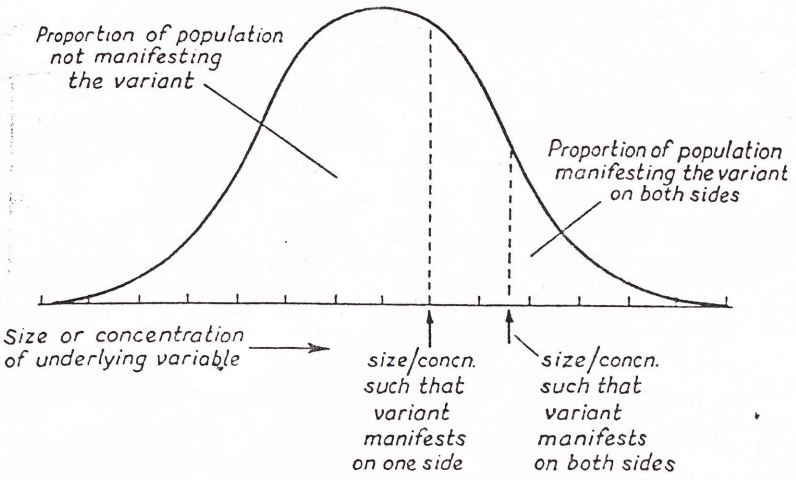

Fig. 1.3. Model for the developmental-genetic basis of epigenetic polymorphisms involving paired characters. The normal curve depicts the frequency distribution of a continuously varying genetic liability which, combined with two developmental thresholds, yields three variant classes in the population—absent, present on one side (i.e., asymmetry), present on both sides. (Reproduced with permission from Berry [1968].)

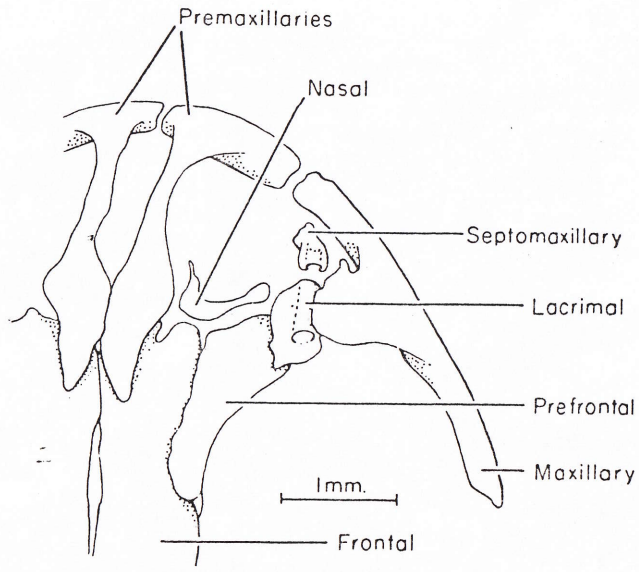

Figure 1.4. Nasal bone in the snout of the Olympic salamander, *Rhyacotriton olympicus* (dorsal view, right side). Of 16 specimens sampled from a single population, 8 had the bone on both sides, 7 had it on one side only, and 1 lacked it entirely. (Reproduced with permission from Wake [1980].)

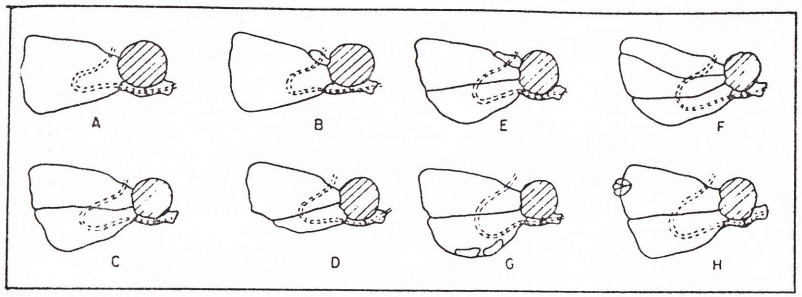

Figure 1.5. Variation in the number, size, and shape of postorbital bones in the skull of the bowfin, *Amia calva*. The modal number of postorbitals per side is 2 (n = 77 specimens), with a range from 1 to 3 or more and frequent asymmetry. Hatched circle denotes the orbit, dashed lines indicate the course of the lateral line sensory canal; all configurations are drawn as right sides. (Reproduced with permission from Jain [1985].)

of the variant in one or a few individuals to its incorporation as a characteristic feature of the entire population (Berry and Searle 1963).

Second, variants of this type may provide insight into the developmental mechanisms of evolutionary change in particular groups. For example, variable presence/absence of individual bones is frequently observed in paedomorphic taxa, i.e., those that fail to complete the ancestral cranial ontogenetic sequence during their own ontogeny. In such instances, the variable element typically is one that forms late in the ancestral ontogeny; its variable occurrence in descendant species reflects variability among individuals or even larger segments of the population (e.g., geographic regions; Wake 1980) in the degree to which they complete the ancestral ontogeny. In cases where a variant occurs at extremely low frequency, it is often interpreted as the atavistic "reappearance" of an ancestral feature (e.g., Alberch 1983). In most instances, however, there is insufficient information with which to eliminate the alternative interpretation that the variant has been continuously maintained in the population from the time of the ancestor, albeit at low frequency. Another example of the important role of epigenetic processes in evolution may be Bock's (1959, 1960) study of the evolution of secondary jaw articulations in birds, an important structural innovation which has evolved repeatedly in living taxa. Initial evolutionary steps likely involve the epigenetic development of an articulation between the mandible and the skull base, which are brought into incidental contact as a result of modifications that primarily affect other musculoskeletal components of the feeding apparatus. However, while the morphology of secondary articulations varies widely among taxa, polymorphisms involving variable development of these articulations within a species have not been reported.

Third, these variants may be useful in establishing homologies between anatomical features in ancestors and descendants. An excellent example is provided by Reilly and Lauder (1988), who used the presence of "atavistic" epibranchial cartilages in red-spotted newts (*Notophthalmus viridescens*) to resolve a long-standing debate concerning the homologies of branchial arch segments in urodeles with respect to bony fishes.

Type 3: Trophic Polymorphisms. The third class of variation involves the coexistence within a given population of two or more discrete cranial phenotypes, or morphs, that are distinguished from one another by coordinated changes involving a large number of individual characters. A second, fundamental characteristic of these variants is that the alternate morphs either represent an adaptive response to some environmental variable, or in some other way enhance the immediate fitness of the individual and/or species. In this way, this type of variation is readily distinguished from the gross malformations (type 1; see above) which, while frequently comprising a large number of characters, are maladaptive except under the most exceptional circumstances. Most of the polymorphisms of this type that have been reported involve structures pertaining to feeding; hence, they are termed trophic polymorphisms (table 1.3). Examples include the papilliform/molariform morphs of the Mexican cichlid fish *Cichlasoma minckleyi* (Liem and Kaufman 1984; Sage and Selander 1975; see also the review of trophic polymorphisms in cichlids by Meyer [1991]) and the large- and small-billed morphs in several species of African finches in the genus *Pyrenestes* (Smith 1987, 1990b).

Because of the magnitude of the morphological difference between constituent morphs and their frequently obvious relation to adaptation, these polymorphisms provide among the strongest evidence of the evolutionary significance of intraspecific variation in cranial features. At the same time, a great deal of fundamental information concerning their basic biology is unavailable for any trophic polymorphism so far described—information that must be obtained before their full significance for the evolution of morphological diversity can be assessed. Two questions are especially important:

(1) Is intraspecific trophic polymorphism an incipient stage in the evolution of interspecific morphological diversity? Qualitative and quantitative differences between trophic morphs may be remarkably large (fig. 1.6, 1.7). Some morphs are as different from one another as are related species or even genera (e.g., *Pyrenestes;* Smith 1987, 1990b); indeed, many were

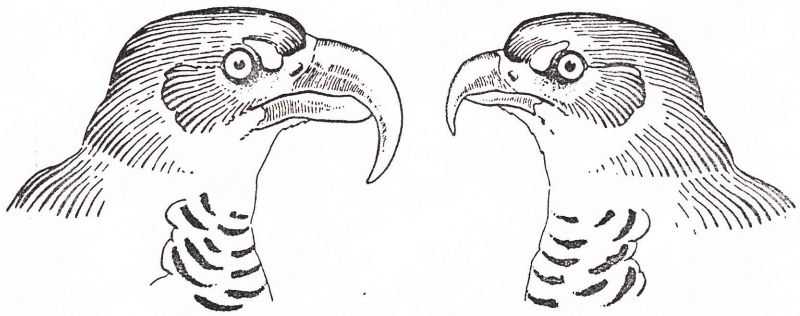

Fig. 1.6. Large- and small-billed morphs of the hook-billed kite (*Chondrohierax uncinatus*), a snail-eating raptor endemic to the New World tropics. Bill size is bimodally distributed in many parts of the species' range; variation is independent of sex and age. The distribution of bill sizes in regional populations is well correlated with the sizes of terrestrial snails, on which the birds feed almost exclusively. While these morphs likely are conspecific, previous studies have not been able to exclude the possibility that each morph represents a distinct species. (Reproduced with permission from Smith and Temple [1982].)

originally described as such (e.g., *Ilyodon:* Grudzien and Turner 1984a, b; Turner and Grosse, 1980; *Saccodon:* Roberts 1974; *Cichlasoma:* Sage and Selander 1975). It is therefore not surprising that trophic polymorphism has often been offered as a transitional stage, which need only be followed by segregation of the constituent morphs via speciation, in the evolution of interspecific morphological differentiation (e.g., Liem and Kaufman 1984; Meyer 1991; Orton 1954). This scenario is obviously tempting, for it circumvents many of the real problems that plague more traditional models for the evolution of large-scale, discontinuous differences among taxa, such as the difficulty in identifying the impetus for evolutionary transitions through putative intermediate stages that are seemingly maladaptive (Gould and Vrba 1982). It also avoids disputed features, such as random morphological change at speciation and species selection, that are central to certain hierarchical models of morphological evolution, such as punctuated equilibrium (Gould 1982; Gould and Eldredge 1977; Levinton 1988). Nevertheless, convincing, unequivocal evidence in favor of this model for the evolution of interspecific morphological diversity, which at the same time excludes alternative models, is lacking. Moreover, the seeming rarity of such discrete polymorphisms in many major taxa, including some, such as birds (Smith and Temple 1982), in which patterns of interspecific and intraspecific variation in the cranium are relatively well documented, suggests that trophic polymorphism is not a predominant "route" to interspecific diversification in cranial morphology in vertebrates generally.

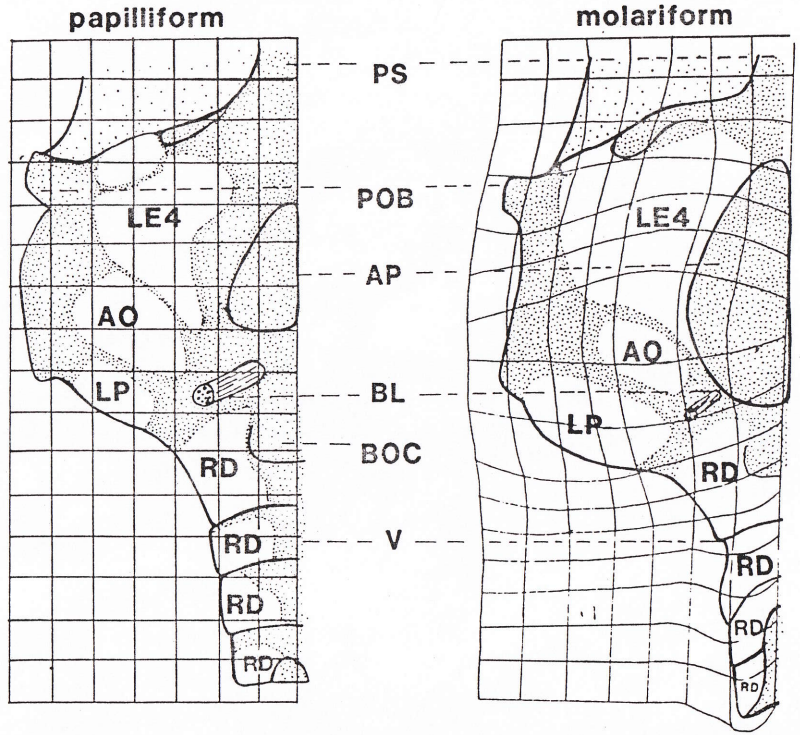

Fig. 1.7. Otic region of the neurocranium and anterior vertebrae of the two trophic morphs of the cichlid fish *Cichlasoma minckleyi*; ventral view, right side only. Unshaded areas depict origins of major muscles. Deformed Cartesian coordinates (standardized to the papilliform morph) graphically depict some of the morphological differences, and possible evolutionary transformation, between the morphs. Standard length for both specimens = 7.4 cm. Abbreviations: AO, adductor operculi; AP, pharyngeal apophysis of the parasphenoid; BL, Baudelot's ligament; BOC, basioccipital; LE4, fourth levator externus; LP, levator posterior; POB, postorbital process; PS, parasphenoid; RD, retractor dorsalis; V, vertebra. (Reproduced with permission from Liem and Kaufman [1984].).

Perhaps the most plausible example yet offered of trophic polymorphism as a vehicle for speciation is the Central American cichlid fish, *Cichlasoma citrinellum* (Meyer 1989, 1990a, b, 1991). In these fishes, two morphs (papilliform and molariform), which are distinguished by several features of cranial morphology and body form, are specialized for eating different prey types. Moreover, each morph is significantly correlated with a distinctive coloration, which is a primary cue for assortative mating. Thus, all the ingredients are at hand for morphological, ecological, and

genetic isolation of the two morphs, which might eventually culminate in speciation. Indeed, Meyer (1990a, b, 1991) has offered this scenario as a mechanism for the explosive adaptive radiation of cichlid fishes in freshwater lakes of Africa and Central America.

An obvious alternative to the above view is that trophic polymorphism represents an evolutionary stable end point that bears no necessary relation to subsequent divergence culminating in speciation. According to this view, each polymorphism represents an adaptive "strategy" that enhances survival of the species by increasing the efficiency of trophic resource utilization and/or broadening the resource base (Kornfield et al. 1982; Liem and Kaufman 1984; Smith 1990c; Vrijenhoek 1978). While this may satisfy some of the preconditions for and enhance the likelihood of speciation (Maynard Smith 1966; McKaye et al. 1982), this would not be an inevitable outcome. Indeed, it has been argued that phenotypic plasticity, which underlies the development of variant morphs in many instances of trophic polymorphism, "may be a form of inertia against speciation" (Meyer 1987, 1366).

Both of these models are plausible; moreover, they are not mutually exclusive. A fundamental challenge to future studies is to establish which one predominates in nature and why. Predominance of the view of trophic polymorphism as an incipient stage of interspecific divergence would inevitably lead to investigation of how frequently morphological diversification among taxa involves polymorphisms of this type, viz., an initial phase in which alternate, discrete phenotypes are maintained within the same population. Predominance of the view of trophic polymorphism as a relatively stable end point within species would not diminish its role as a mechanism of morphological diversification per se, although its importance as a vehicle for interspecific divergence obviously would be lessened. Finally, it remains to be established what intrinsic factors (e.g., anatomical, genetic, developmental) and extrinsic factors (e.g., predator-prey relations, community structure, and dynamics) promote the appearance of trophic polymorphisms, and why they may be more common in some groups than in others.

(2) What are the developmental and genetic bases of trophic polymorphisms? The question of the developmental and genetic bases of a trophic polymorphism almost invariably follows its initial description. In general, morphological traits are under polygenic control (Falconer 1981; Levinton 1988), and it is reasonable to expect that this generalization applies to the characteristics of trophic morphs; see, for example, Atchley's (1993) discussion of the genetics of mandibular variation in mammals. Yet, the actual genetic basis of intrapopulation differences in cranial morphology, and especially the way genome and environment interact to mediate cranial development, is poorly known for virtually every known instance of trophic polymorphism. Moreover, the answers that have been obtained to date are so variable that it is difficult to generalize. Nevertheless, two features are apparent. First, species that display trophic polymorphisms obviously possess genetic liability for the development of particular morphs; without this liability, the morphs could not form, regardless of the nature of the environmental stimuli. Second, many instances of trophic polymorphism involve a predominant role for the environment in making the developmental choice among alternate patterns of cranial ontogeny and, ultimately, cranial morphology (e.g., *Ilyodon;* Grudzien and Turner 1984a). Even such instances of environmentally controlled developmental polymorphisms, however, may be caused by different underlying genetic mechanisms; for example, a single shared genotype and a highly variable genotype could each be expressed as a series of discrete morphs given the right combination of environmental stimuli and developmental thresholds (Collins and Cheek 1983).

The environmental factor that perhaps most commonly promotes the development of alternate trophic morphs is food. Holbrook (1982), for example, implicated likely dietary differences between field mice (*Peromyscus*) inhabiting woodland or grassland habitats as the primary explanation for the distinctive mandibular morphotype characteristic of mice from each area. Interestingly, despite its predominantly environmental cause, the dimorphism is apparently quite stable, being present both in prehistoric and contemporary populations at three widely separated localities in the southwestern United States. Meyer (1987) documented that differences in food (and possibly feeding mode) underlie the considerable differences in jaw and snout structure between "acuto-rostral" and "obtuso-rostral" morphs in the cichlid fish, *Cichlasoma managuense*. These morphs represent the extremes of a continuum of morphological variation in this species, yet Meyer was able to evoke the differences by raising laboratory lines on different diets during the first eight months after the onset of feeding; moreover, the obtuso-rostral morph transformed to acuto-rostral following a change of diet (fig. 1.8). Finally, Pomeroy (1981) was able to produce tadpoles with hypertrophied jaw musculature characteristic of the naturally occurring carnivorous morph in the North American spadefoot toad, *Scaphiopus multiplicatus,* by varying diet in the laboratory. He also documented a "modest genetic effect" (p. 106) contributing to the appearance of the carnivorous morph in carefully controlled laboratory experiments.

Other environmental factors besides diet may also mediate trophic polymorphisms. In a controlled laboratory study of the tiger salamander (*Ambystoma tigrinum*), for example, high population density promoted the appearance of the distinctive larval cannibal morph independent of food

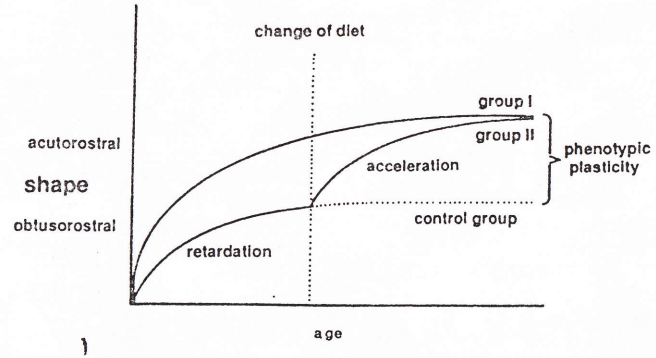

Fig. 1.8. Effects of diet and possibly feeding mode on cranial development in the cichlid fish *Cichlasoma managuense*. During ontogeny, the cranial morphology of group I, fed brine shrimp, changes from obtusorostral to acutorostral. Group II, fed flake food and worms, retains the obtusorostral morphology until 8.5 months of age (vertical dotted line), when a change of diet to shrimp promotes development of the acutorostral morph. (Reproduced with permission from Meyer [1987].)

level, which had no effect (Collins and Cheek 1983). Unlike diet or food processing, however, which likely mediate cranial development through changes in the mechanical environment of the musculoskeletal trophic apparatus, the specific way in which high density affects cranial morphogenesis is not known. Moreover, because the experimental design used in this study varied only food level, but not type, it is not known whether prey type or feeding mode may also promote the appearance of the cannibal morph.

Evidence suggesting that environmental stimuli may not be a primary cause of the development of all trophic polymorphisms, however, comes from another study of the cichlid fish *Cichlasoma minckleyi* (Sage and Selander 1975). When samples of broods were reared in aquaria on a soft diet, the fishes developed into both papilliform and molariform morphs. The latter is regarded as specialized to feed on a diet of hard food such as snails, but this food obviously is not required to evoke development of this morph. A predominantly genetic basis has also been claimed in other studies (e.g., Bragg 1965, Bragg and Bragg 1959; Smith 1987, 1990a), but the evidence is mostly preliminary, indirect, or circumstantial and does not exclude the possibility of a strong environmental component (see, however, Pomeroy 1981).

A final, intriguing aspect of the development basis of trophic polymorphisms that is only beginning to be explored is the possibility that variant morphs result from only slight perturbations to the normal developmental program characteristic of the species. Perhaps the best example

to date is Meyer's (1987) study of the cichlid fish *Cichlasoma managuense* discussed earlier in which the variant obtuso-rostral morph, which can be evoked and sustained indefinitely by a particular diet, is an early and transient ontogenetic stage of fishes that typically develop into the acuto-rostral morph (fig. 1.8). Moreover, transformation from the "paedomorphic" obtuso-rostral morph to the acuto-rostral morph is readily achieved by a simple change in diet. A similar example is seen in *C. citrinellum,* in which all fish begin life as papilliform morphs; molariform morphs develop only following subsequent allometric growth as well as qualitative morphological changes (Meyer 1990b).

A second example comes from the so-called cannibal morph in North American spadefoot toads, *Scaphiopus*. The cannibals, or carnivorous tadpoles, differ from typical, omnivorous larvae in several cranial and postcranial features, including hypertrophied jaw musculature, fewer labial teeth and oral papillae, shorter intestine, and decreased melanization (Bragg and Bragg 1959; Orton 1954; Pomeroy 1981). All of these features typically accompany both natural and thyroid-hormone-induced metamorphosis in anurans (Etkin 1968; Fox 1984; Hanken and Hall 1988; Hanken and Summers 1988), including *Scaphiopus* (Pomeroy 1981). Thus, the carnivorous tadpole may represent a case of accelerated development, in which the appearance of certain postmetamorphic features has been accelerated into the larval period. This model, however, cannot explain such cannibal traits as the enlarged horny beak; the moderate-sized beak of omnivorous tadpoles does not enlarge at metamorphosis, but instead is shed.

These examples are important in at least two respects. First, they reveal that the initial evolution of trophic polymorphisms may be a relatively simple matter, that is, it need not require any fundamental or large-scale repatterning of the ontogenetic program of cranial development or of the underlying genome (Liem and Kaufman 1984). Second, they illustrate how the "novel" cranial configurations frequently represented by variant morphs are nevertheless a function of and constrained by the typical pattern of cranial development in each species.

CONCLUSION

Developmental Rules for Skull Specification during Ontogeny

As a means of generalizing the available information on the development of the skull and of relating ontogenetic mechanisms to phylogenetic change, we have assembled the following set of development rules (sensu Oster et al. 1988) that appear, with current knowledge, either to apply to

skull development in all vertebrates, or to be capable of extrapolation to other vertebrate taxa from those for which data is available.

1. A regionalized, skeletogenic neural crest extends from the anterior mesencephalon/mid-prosencephalon caudad to somite 5 at the neurula stage of all vertebrates, providing a fundamental pattern of future skull-forming cells.
2. Transformation of epithelial neural crest cells (the state in the neural tube) to mesenchymal (mesectodermal, ectomesenchymal) cells occurs at the outset of migration.
3. Skeletogenic cells migrate from the neural tube as predictable streams, but not along predictable pathways, to "make" the head.
4. Extracellular matrix-mediated mechanisms trap regionalized subpopulations of neural crest–derived cells against neuroepithelia at sites of future cartilage capsule formation (so far demonstrated in avian and mammalian embryos), establishing the fundamental structural pattern of the vertebrate chondrocranium.
5. Initiation of chondrogenesis/osteogenesis requires interactions between neural crest–derived mesenchymal cells and epithelia. The timing of these interactions, the particular epithelia involved, and perhaps also the state of determination of the mesenchymal cells at the time of the interaction varies from group to group, and provides a mechanism, through heterochrony, of generating diversity on the basic structural plan of the skull.
6. Condensation (aggregation) of mesenchyme follows and is caused by the epithelial-mesenchymal interaction. Condensation size is important in determining the size of individual skeletal elements.
7. Cell-type specific molecules and macromolecules are synthesized and deposited into extracellular matrices as the mesenchymal cells differentiate into chondroblasts or osteoblasts. Variation in synthesis of extracellular matrix regulates skeletal form and growth.
8. Final determination of skull form involves integrated and coordinated growth of cartilaginous and bony elements, both among themselves and with respect to adjacent nervous, sensory, circulatory, muscular, and other connective tissues.

These eight rules provide the minimum number of developmental processes required to specify both the stability and the diversity of skull form among vertebrates. They are, however, derived from broad comparisons among mostly individual representatives of distantly related taxa. A fundamental challenge to future studies of skull morphology and evolution is to document the specific ways in which these (and possibly other) processes have been modified to achieve structural diversity in particular lineages, and how they relate to other mechanisms of adaptive and nonadaptive change.

Intraspecific Variation

Comprehensive understanding of the way that changes in developmental processes and patterns are translated into morphological differences among taxa requires consideration of the middle ground of intraspecific variation. As a beginning, we have considered three classes of cranial variants commonly observed in natural populations. *Gross malformations* offer an excellent opportunity to examine the basic mechanisms of cranial development and genetics. They also reveal the capacity for seemingly slight perturbations of developmental processes to effect large-scale changes in adult morphology. Nevertheless, because of the exceptional decrease in fitness typically conferred by these changes, we attribute little significance to them as vehicles of significant evolutionary change.

Epigenetic polymorphisms are routinely observed when adequate sample sizes are considered and may prove to be ubiquitous in natural populations. They provide a simple and rational mechanism for the initial appearance of novel, discontinuous variants and for subsequent incorporation of these initially rare variants into the population at large. Moreover, this mechanism is fully consistent with accepted principles of developmental genetics. Thus, epigenetic polymorphisms represent a likely source of the raw material necessary for directional change or diversification which may result from a variety of processes, such as natural selection, drift, and population fragmentation. For these reasons, we consider epigenetic polymorphisms to be of primary importance in morphological diversification among taxa.

Trophic polymorphisms are, except for sexual dimorphism, perhaps the most dramatic illustrations of intraspecific variation in cranial morphology in nature; they exemplify the fact that large-scale morphological change need not be contingent on speciation. They usually are of obvious selective value to the species involved, and pose a number of challenging and important questions that lie at the interface of ecology, morphology, development, genetics, and behavior. Yet, trophic polymorphisms in general would appear to be rare; while they may prove to be more abundant than now recognized in some groups, viz., teleost fishes, it is unlikely that they will prove to be a common phenomenon overall. They clearly represent a mechanism for morphological diversification that is distinct from more traditional models that entail speciation and subsequent divergence among taxa. At present, however, it remains unresolved how frequently trophic polymorphisms culminate in speciation and interspecific morphological differentiation, as opposed to remaining stable instances of intraspecific variation. Finally, the paucity of examples of adaptive cranial polymorphisms involving functional character complexes unrelated to feeding suggests that discontinuous variation of this type may be a viable

mechanism for morphological diversification only for trophic structures within the skull.

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2

Convergent and Alternative Designs for Vertebrate Suspension Feeding

S. Laurie Sanderson and Richard Wassersug

INTRODUCTION

SUSPENSION-FEEDING AQUATIC ANIMALS capture planktonic prey as water flows past the feeding apparatus. Vertebrate suspension feeders include species of fishes, tadpoles, whales, and birds and are of evolutionary, ecological, and economic importance. Ancestral vertebrates are thought to have been suspension feeders as larvae (Jollie 1982; Northcutt and Gans 1983) or as adults (Mallatt 1985). Suspension feeding appears to have evolved independently multiple times in teleost fishes and in elasmobranchs (Moss 1977, 1981; Cavender 1970). By consuming phytoplankton, zooplankton, and/or detritus, suspension feeders obtain their energy at a relatively low level in the trophic pyramid and may attain large standing stocks (e.g., herrings and sardines) or large body size (e.g., whales and whale sharks).

The morphology and physiology of a large number of invertebrate suspension-feeding species have been described (reviews in Wallace and Merritt 1980; Jørgensen 1966, 1975; Vanderploeg 1990; Wotton 1990). The physical mechanisms operating in trophic fluid transport systems and in biological filters have been examined through the application of theoretical fluid mechanics to invertebrate suspension feeding (Shimeta and Jumars 1991; LaBarbera 1990, 1984; Jørgensen 1983; Rubenstein and Koehl 1977). The hydrodynamics of feeding in aquatic vertebrates have only recently received attention (Sanderson et al. 1991; Lauder and Shaffer 1986; Muller and Osse 1984; Lauder 1980; Weihs 1980), and the mechanics of vertebrate suspension feeding remain an open field for research. Ecological information on vertebrate suspension feeding far exceeds our understanding of the functional morphology involved. For example, functional morphological studies are needed to establish the structure of the prey-capturing surfaces. But more important, the pattern and velocity of water flow within the oral and (in fishes) opercular cavities must be deter-

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