Carcinization as an Underlying Synapomorphy for the Decapod Crustacean Taxon Meiura

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Abstract - Scholtz and Richter (1995) have presented complex morphological synapomorphies uniting the reptant decapod taxa Anomura and Brachyura as sister taxa (constituting the taxon Meiura). It is proposed that the evolution of a crab-like morphology (carcinization), which has occurred independently in several anomuran clades and which is characteristic of the Brachyura, be regarded as an underlying synapomorphy for the Meiura. An evolutionary model of three aspects of carcinization -- dorsoventral compression of the carapace, broadening of the cephalothorax, and the reduction-folding of the abdomen -- is presented based upon hypothetical meiuran-specific alterations in homeotic gene regulatory elements. Carcinization in the Anomura and grades of carcinization in the Brachyura are hypothesized to result from the differential derepression of homeotic gene regulatory elements specifying the carcinized phenotype.

Introduction

The anomuran and brachyuran decapod crustaceans represent two of the most morphologically and ecologically diverse crustacean taxa known. Scholtz and Richter (1995), in a phylogenetic systematic analysis of the reptant decapods, have elucidated complex synapomorphies which unite these two infraorders as sister taxa (designated the Meiura). In their analysis the tendency to adopt a crab-like form or carcinization was not explicitly discussed, i.e., carcinization as a possible indicator of phylogenetic relationships.

The adoption of a crab-like habitus is a prime characteristic of the brachyurans in that every member of the infraorder is carcinized to some extent. Carcinization is manifested as the extremely dorsoventrally compressed carapace, a cephalothorax that is usually wider than long or (e.g., in the Raninidae) longer than wide, and with a greatly reduced pleon which is ventrally flexed under the thoracic sternites (Borradaile, 1916; Richter and Scholtz, 1994). The chief study of the morphological aspects of carcinization was by Borradaile (1916); Stevcic (1971) also discussed the morphological consequences of carcinization. The acquisition of the carcinized *Unterbauplan* in the urbrachyuran led to a number of anatomical readjustments which have enabled members of this infraorder to expand into a number environments (e.g., arboreality in some Gecarcinucidae; Cumberlidge and Sachs, 1991) which are inaccessible to most other decapod taxa. Furthermore, even the most derived brachyurans such as gall crabs (family Hapalocarcinidae) exhibit a carcinized *Unterbauplan*. Thus it can be concluded that the first brachyuran possessed at least a semicarcinized *Unterbauplan* (Stevcic, 1971).

Carcinization is less a defining characteristic of the Anomura (Richter and Scholtz, 1994). This is the most morphologically diverse decapod taxon including the mole crabs (hippids), squat lobsters (galatheids), aeglids, albuneids, king crabs (lithodids), and symmetrical abdomen (pylochelids) and asymmetrical abdomen hermit crabs (pagurids, diogenids, and coenobitids), etc.. All the anomuran clades, however, reveal a tendency for carcinization with some lineages, e.g., the Lomisidae, Lithodidae, and Porcellanidae, being partially or fully carcinized. Molecular evidence also suggests that some taxa typically considered primitive brachyurans, particularly the dromiids, are in fact highly carcinized anomurans (Spears *et al.*, 1992; for an opposing interpretation see Scholtz and Richter, 1995). That carcinization in the Anomura represents a 'rampant' process of parallelism (Sluys, 1989) can be determined from the coconut crab *Birgus latro* which is a carcinized coenobitid hermit crab (McLaughlin, 1983) and the Lithodidae which arose from pagurid hermit crabs (Cunningham *et al.*, 1992) not to mention intermediately carcinized taxa such as *Probeebei* (Gould, 1992). And even anomurans which are only slightly carcinized, e.g., the aeglids, nevertheless reveal the tendency to flex the pleon under the thoracic sternites (Martin and Felgenhauer, 1986).

Carcinization thus represents a problem which is the bane of phylogenetic analysis: homoplasic transformations of characters. Phylogenetic analysis *sensu* cladistics is the search for synapomorphic character states which enable the demarcation of monophyletic groups. Parallelisms can only obscure this

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search for monophyletic taxa. In the case of most carcinized Anomura, apomorphic character states (e.g., asymmetrical abdomens in female lithodids) reveal the convergent nature of other character states such as the well calcified dorsal region of the carapace or the reduced, ventrally flexed abdomen (Richter and Scholtz, 1994). Establishing robust synapomorphies aside, parallelisms can nevertheless tell us much about not only the evolutionary process in general (Salthe, 1993) but also about the genetic potentiality of clade. This genetic potentiality refers to genomic information which is silenced or only expressed during some ontogenetic stages but which can be reexpressed and retained into the adult stages. Atavisms would be one such example of a genetic potentiality (Hall, 1995). It is thus possible that carcinization reflects a meiuran-specific genetic potentiality which is not an atavism but rather a genetic network that is universally expressed in one clade although to different degrees (grades of brachyuran complexity; Stevcic, 1971) and is reexpressed in parallel in a number of anomuran lineages also to different degrees.

It has long been recognized that transformations of the *Bauplan* need not involve large scale alterations of structural genes but probably involve subtle changes in the circuits formed by key regulatory loci. Zuckerkandl's (1976) assertion that it would be possible to make any crustacean out of the genome of a higher crustacean has received support from the analysis of homeotic genes involved in *Bauplan* determination from clades as diverse as arthropods and chordates (Akam, 1995; Carrol, 1995). The molecular genetic evidence is mounting that evolutionary transitions in morphology involve the rewiring of "essentially constituted genomes" with parallelisms the result of gene regulatory circuits adopting the same wiring topologies (Zuckerkandl, 1976, 1994). The key to carcinization may therefore be found in such regulatory circuits as entailed by *Hox* loci and downstream regulated homeotic genes. These *Hox* and homeotic gene encoded regulatory circuits constitute a 'genetic potentiality' with circuit topologies (stable ontogenetic patterns of gene regulation) representing different instantiations of this potentiality.

With the above in mind, carcinization as a defining characteristic of the Brachyura and a rampant parallelism (Sluys, 1989) in the Anomura, can be phylogenetically interpreted in two ways. First, for the Brachyura, the carcinized *Unterbauplan* can be interpreted as an autapomorphy of the clade; the carcinization events which have occured in the Anomura thus represent true homoplasies, i.e., parallelisms genetically and ontogenetically uncoupled from each other and with the unique carcinization event characterizing the brachyurans. Alternatively, carcinization of the Brachyura and the parallel carcinization of many anomuran taxa can be regarded as the complete expression, partial expression, and repression of *Hox* and homeotic gene regulatory circuitry specific for the Meiura. The fact that carcinization is limited to these two sister taxa and does not constitute an evolutionary potentiality of the decapod *Bauplan* as such leads to serious consideration of the second phylogenetic alternative. A model of homeotic gene regulatory evolution leading to grades of carcinization is presented and the role of regulatory circuits as underlying synapomorphies is discussed. Only three morphological aspects of carcinization are considered: dorsoventral compression of the carapace, broadening of the cephalothorax, and the reduction-folding of the abdomen. This model is only partly speculative in that it is testable and based on known mechanisms of homeotic gene regulation. Furthermore, I submit that the ideas presented here, although perhaps unorthodox, are compatible with the concepts developed by Slack *et al.* (1993) and Minelli and Schram (1994).

Hox Codes, Homeotic Gene Regulation, and Carcinization

The broadening of the cephalothorax and the reduction-ventral flexing of the abdomen that occurs during carcinization is a transformation of tagma morphology. The carapace, here assumed to be an extension of the cephalic shield and possibly a maxillary derivative (Hessler *et al.*, 1982), is thus a head structure that coevolved with the cephalothoracic segments. Carcinization involved, at least for the three aspects considered here, a transformation of the structures of three tagmata: cephalon, thorax, and the abdomen. Thus if the potential for carcinization constitutes an underlying synapomorphy for the Meiura, the genetic initiating conditions for this process most likely resides with loci governing segment identity and tagmosis.

Segment identity in arthropods, chordates, and nematodes has a basis in the *Hox* genes (clusters of homeobox containing loci homologous to the insect and vertebrate homeotic gene clusters; Akam, 1995). Spatiotemporal patterns of *Hox* gene regulation undergird segment identity in the Arthropoda and these spatiotemporal patterns or *Hox* codes (Akam, 1995) are the result of different batteries of *Hox* regulatory elements being activated or silenced. Tagmata thus arise from groups of adjoined segments expressing the same *Hox* code and patterns of tagmosis have their substrate in different *Hox* codes being expressed along the arthropod trunk. This model of *Hox* specification of tagmosis proposes that transitions in the morphology of tagmata reside, at the micro-level, not in the emergence of new *Hox* genes but instead in

changed parameters of *Hox* codes (Akam *et al.*, 1994). Eucaridan tagmosis (and thereby decapod tagmosis) thus most likely has a molecular basis in the states of regulatory elements controlling the transcription of *Hox* loci.

One 'primitive' crustacean that has been examined for the expression of *Hox* loci during development is the branchiopod *Artemia* (Averof and Akam, 1993, 1995). *Artemia* shares with insects all the major *Hox* loci involved in trunk segment diversification and the *Hox* clusters that regulate crustacean tagmosis probably predate the radiation of the Mandibulata (Akam, 1995). Unless one postulates unique *Hox* duplications leading to the eumalacostracan, eucaridan, or decapod *Bauplan*, the expression of *Hox* loci in *Artemia* can serve as a plesiomorphic framework upon which to construct molecular hypotheses of carcinization.

Homologues of the *Drosophila Antennapedia* (*Antp*), *Ultrabithorax* (*Ubx*), and *abdominal A* (*abdA*) loci are expressed prior to and during thoracic development in *Artemia* in the segments posterior to the gnathal ones and anterior to the genitalia-abdomen (Averof and Akam, 1995). (These homologues are referred to here by their *Drosophila* designations.) Assuming this *Hox* expression pattern is the result of a plesiomorphic (mandibulate)*Hox* code, the decapod cephalothoracic segments posterior to the segments bearing the maxillipeds and anterior to the first pleon segment are postulated to be under identical regulation. These same investigators postulate that part of the malacostracan abdomen is homologous to the anostracan (*Artemia*) thorax which would place the anterior region of the decapod pleon under the influence of the *Antp*, *Ubx*, and *abdA* code. The finding that *Ubx* and *abdA* are coexpressed in the developing pleon (up to the fifth abdominal segment) in the opossum shrimp *Mysidopsis* (a eumalacostracan; Panganiban *et al.*, 1995) suggests that the decapod pleon might be under similar regulation. Although this does not rule out the involvement of *Antp* in the pleon *Hox* code, for the sake of simplicity *Ubx* and *abdA* are considered the key regulators of this tagma. Expression patterns of *Hox* loci governing head development (i.e., gnathal and anterior segments) have, to the author's knowledge, not been investigated in Crustacea. The *Antp* locus appears to be the *Hox* gene regulating head development in *Drosophila* (Morata, 1993); therefore, given the *Hox* code similarities between *Drosophila* and *Artemia* (Carrol, 1995), *Antp* or an *Antp* derivative is the most likely master switch in decapod cephalon development.

The *Hox* loci do not in and of themselves specify segment identity. Rather they serve to regulate in *trans* other homeotic and nonhomeotic loci that in turn regulate genes that provide the parameters of segment identity. Major morphological transitions in insects often involve mutations in the regulatory circuits encoded by *Hox* loci, downstream regulated loci, and other *trans* factors (DeSalle and Carew, 1992; Carrol, 1994). For example, hypercephaly and transformations of proboscipedial structures in the Drosophilidae involve mutations in *Hox*-regulated homeotic genes (DeSalle and Carew, 1992). The origin of insect wings and their evolution in various clades appears to have been similarly due to alterations in *Ubx* controlled loci (Carrol, 1994). Thus with all the genetic evidence pointing to *Hox* and downstream regulated loci acting as initial conditions in *Bauplan* formation, the following molecular framwork of carcinization can be formulated:

> I. The *Hox* codes involved in the development of the decapod cephalothorax and abdomen entail the *Antp-Ubx-abdA* and *Ubx-abdA* loci, respectively. Segments bearing the maxillilae and antennae have their initiating conditions in the *Antp* locus.
>
> II. Shape transformations of tagmata in the Decapoda are not due to alterations in the plesiomorphic *Hox* codes but instead involve mutations in downstream regulated homeotic loci. As the carapace appears to arise from head structures, the putative homeotic locus involved in carapace shape would be regulated by the *Antp* code. The other homeotic loci involved in the shape of the cephalothorax and abdomen are likewise regulated by the *Hox* codes of the respective tagma. Putative genes involved in carcinization are hereafter referred to as 'c-loci'.

One other group of genes appear likely to be involved in carcinization. The *polycomb-group* (*Pc-group*) homeotic loci are involved in the long-range silencing or heterochromatinization of homeotic loci, or more specifically, their regulatory elements, in cells where they are not to be expressed (Kennison, 1995). After the products of gap genes silence the enhancers of specific homeotic loci in sets of primordia in the early embryo, complexes of *Pc-group* products take over the role of the gap gene products to epigenetically maintain transcriptional inactivation of the loci (Müller *et al.*, 1995). Alterations in *Pc-group* genes or their mode of regulation can result in morphological changes similar to that which occur due to mutations in *Hox* genes or other homeotic loci. This general repressor function of the *Pc-group* genes is

highly conserved in both *Drosophila* and mice (Müller *et al.*, 1995). Therefore, large transformations in segment structure could involve Hox and Hox regulated homeotic loci, their cell-specific silencing by gap and*Pc-group* genes, or a combination of these. Indeed, mutations affecting *Pc-group* products binding to homeotic gene regulatory elements are often the molecular basis for homeotic transformations of structure (Kennison, 1995). As the products of *Hox* loci act as positive and negative (Carrol, 1994) modulators of downstream regulated homeotic loci so the *Pc-group* genes behave as general repressors. Carcinization may therefore be as 'simple' as slight shifts in the regulatory interplay of *Hox*, c-loci, gap, and *Pc-group* genes.

The above does not pretend to be even a brief review of the action of *Hox* and homeotic regulatory loci. Rather it was presented to suggest that the carcinization process need not be the result of changes in many small-effect loci distributed throughout the anomuran and brachyuran genome but instead could result from alterations in the regulation of genes with large effects (DeSalle and Carew, 1992). Using the brief outline above, a testable and parsimonious -- if unorthodox -- model of carcinization can be presented.

A Model of C-Loci Regulatory Evolution in the Meiura

A homeotic gene model for the parallel emergence of the carcinized phenotype in the Anomura and grades of complexity (development) of carcinization in the Brachyura is based on three postulates: 1) independent loci specify dorsoventral compression of the carapace, broadening of the cephalothorax, and reduction-ventral flexing of the pleon; 2) c-loci as such are not unique to the Meiura -- novel regulatory elements allowing the carcinized phenotype were acquired by the c-loci in the ancestor to the Meiura; and 3) parallel acquisition of the carcinized *Unterbauplan* in the Anomura and the brachyuran grades differing in their degree of carcinization have a basis in the same molecular mechanism -- differential derepression of the meiuran-specific c-loci regulatory elements. Knowledge concerning key regulatory genes in the Decapoda, i.e., genes governing the decapod *Bauplan*, is lacking of course. For the purposes of the model the homeotic loci controlling the shape and size of the decapod carapace, and the overall conformation of the cephalothorax and pleon are designated *kpx*, *cpx*, and *ple*, respectively.

In this model each of the three loci independently acquired novel regulatory elements governing the spatiotemporal transcription of these genes. Homeotic genes are regulated by a limited number of *Hox* loci (Morata, 1993) so it is not unlikely that the meiuran-specific regulatory elements acquired by the *kpx*, *cpx*, and *ple* genes will show some sequence similarities. Figure 1 shows a schematic of proposed c-loci regulatory evolution in the Meiura. This model presents the acquisition of c-loci regulatory elements as resulting from the duplication and divergence of previously existing 5' enhancer sequences. Needless to say, the acquisition of new regulatory elements could have occurred in the intronic or 3' flanking regions of these genes and could have involved the transposition of new sequences or the mutation of old ones (Shimmel *et al.*, 1994); it is only for the sake of clarity that these evolutionary steps are presented as 5' duplications. Thus the only theoretical prediction made here is that the homeotic loci governing carapace, cephalothorax, and abdominal morphology in anomurans and brachyurans will differ from that of other decapods by the presence of synapomorphic regulatory sequences.

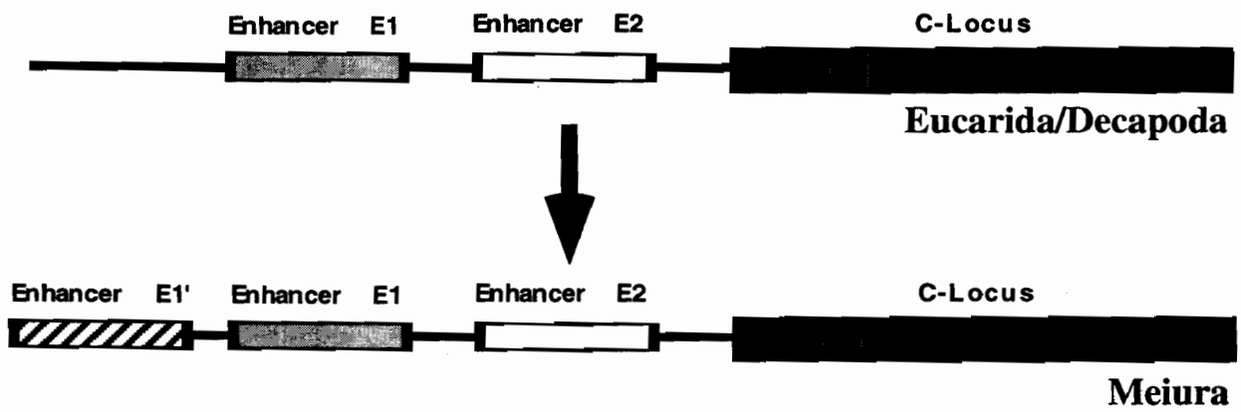

Figure 1. A model of c-loci evolution involving the acquisition of meiuran-specific (synapomorphic) regulatory elements via duplication of a preexisting enhancer sequence.

As mentioned above, not all Brachyura exibit the same degree of carcinization (Stevcic, 1971). Thus any molecular-mechanistic model of carcinization must not only explain the parallelism of carcinization but also grades of carcinization. Several pieces of information concerning the nature of eukaryotic regulatory elements and the expression of homeotic genes (discussed briefly above) can nevertheless point to parsimonious suggestions. Two pieces of information are pertinent: A) the modular, redundant nature of regulatory elements (Camprodon and Castelli-Gair, 1994) and B) the epigenetic silencing of regulatory elements (Chan *et al.*, 1994) by products of the *Pc-group* genes after the initial repression by the gap genes. If we propose that the transcription levels of the *kpx*, *cpx*, and *ple* loci are positively correlated with the extent to which the carapace, cephalothorax, and the pleon are 'carcinized', the makings of a simple evolutionary model of parallel and progressive carcinization becomes apparent. (Note: if pleon reduction is due to apoptosis of the musculature it can also be proposed that the homeotic *ple* locus regulates the extent to which and where this process takes place.)

Figure 2 presents a schematic of evolutionary changes in c-loci regulatio ased upon differences in the epigenetic silencing of the meiuran-specific regulatory elements. According to this model the *kpx* locus is positively regulated by *Antp*; *cpx* by *Antp*, *Ubx*, and *abdA*; and *ple* by *Ubx* and *abdA*. The products of the *Pc-group* genes are general repressors of the c-loci by heterochromatinizing regions of the meiuran-specific regulatory elements (Müller *et al.*, 1995). The degree to which products of the *Pc-group* genes silence the c-loci regulatory elements would be determined by the initial binding of the gap genes (presumably to prevent the binding of the transcription activating *Hox* encoded factors), which in turn is a result of the surrounding chromatin environment. Mutations altering the non-protein coding sequences flanking the homeotic gene regulatory elements or mutations in the regulatory elements themselves (Shimmel *et al.*, 1994) could prevent the initial binding of the gap gene products or the follow-up binding of the *Pc-group* products thereby allowing transcriptional activation from the previously silenced element. With this background to the model, parallel and progressive carcinization can be explained as follows:

- **Grade 1**. At this level all meiuran-specific c-loci regulatory elements are heterochromatinized (Fig. 2, grade 1), i.e., spatiotemporal transcription of the *kpx*, *cpx*, and *ple* loci that specifies carcinization of the respective regulated structures is prohibited. This is the predicted regulatory state of homeotic c-loci in the anomuran families Galatheidae, Pylochelidae, Aeglidae, Chirostylidae, Coenobitidae, Parapaguridae, Paguridae, and Diogenidae with carcinized members being obvious exceptions. The ancestral anomuran presumably had this c-loci regulatory state.

- **Grade 2**. Here the carcinization specifying regulatory element of *cpx* is silenced; meiuran-specific enhancer sequences of the *kpx* and*ple* gene are only partially repressed by *Pc-group* proteins (Fig. 2, grade 2) permitting some dorsoventral compression of the carapace and limited reduction of the abdomen which may or may not be flexed under the sternum. Taxa which exhibited this level of c-loci regulation were the extinct Eocarcinidae, Prosopidae, and *Mithracites* (Glaessner, 1969) which presumably arose after the Anomura-Brachyura lineages diverged but which are basal to the 'higher' Brachyura. (This is the lowest grade of carcinization in the Brachyura.) Modern taxa which are hypothesized to possess this mode of c-loci regulation include the Hippidae, Albuneidae, and *Probeebei* in the Anomura and the Homolodromiidae, Dynomenidae, Tymolidae, and Raninidae in the Brachyura.

- **Grade 3**. The E1' (i.e., meiuran-specific) enhancer (Fig. 1) of the *ple* locus is completely derepressed whereas the acquired enhancers of the *kpx* and *cpx* loci exert some influence on the transcription of these genes, i.e., the gap gene products do not specify complete silencing by the *Pc-group* proteins (Fig. 2C). The spatiotemporal expression of the *kpx* and *cpx* loci allow for the dorsoventral compression of the carapace and partial lateral expansion of the carapace and cephalothorax structures. The abdomen is considerably reduced due to *ple* controlled apoptosis. Anomuran taxa such as the Lomisidae, Porcellanidae, the coenobitid *Birgus*, and the pagurids *Porcellanopagurus*, *Ostraconotus*, and *Tylaspis* (Borradaile, 1916; Stevcic, 1971) have this level of c-loci regulation. In *Birgus* this c-loci deregulation would occur rather late in ontogeny (discussed in Richter and Scholtz, 1994). Whether one places the Dromiidae in the Anomura (Spears *et al.*, 1992) or as representatives of the Archaebrachyura (Scholtz and Richter, 1995), the family is predicted to be at this level.

- **Grade 4**. The E1' enhancers of the *kpx* and *cpx* loci are fully active resulting in complete or nearly complete dorsoventral flattening of the carapace and broadening of the carapace and cephalothorax.

The *ple* locus is at the same regulatory stage as in grade 3. This is the mode of c-loci regulation seen in all Eubrachyura and the Lithodidae.

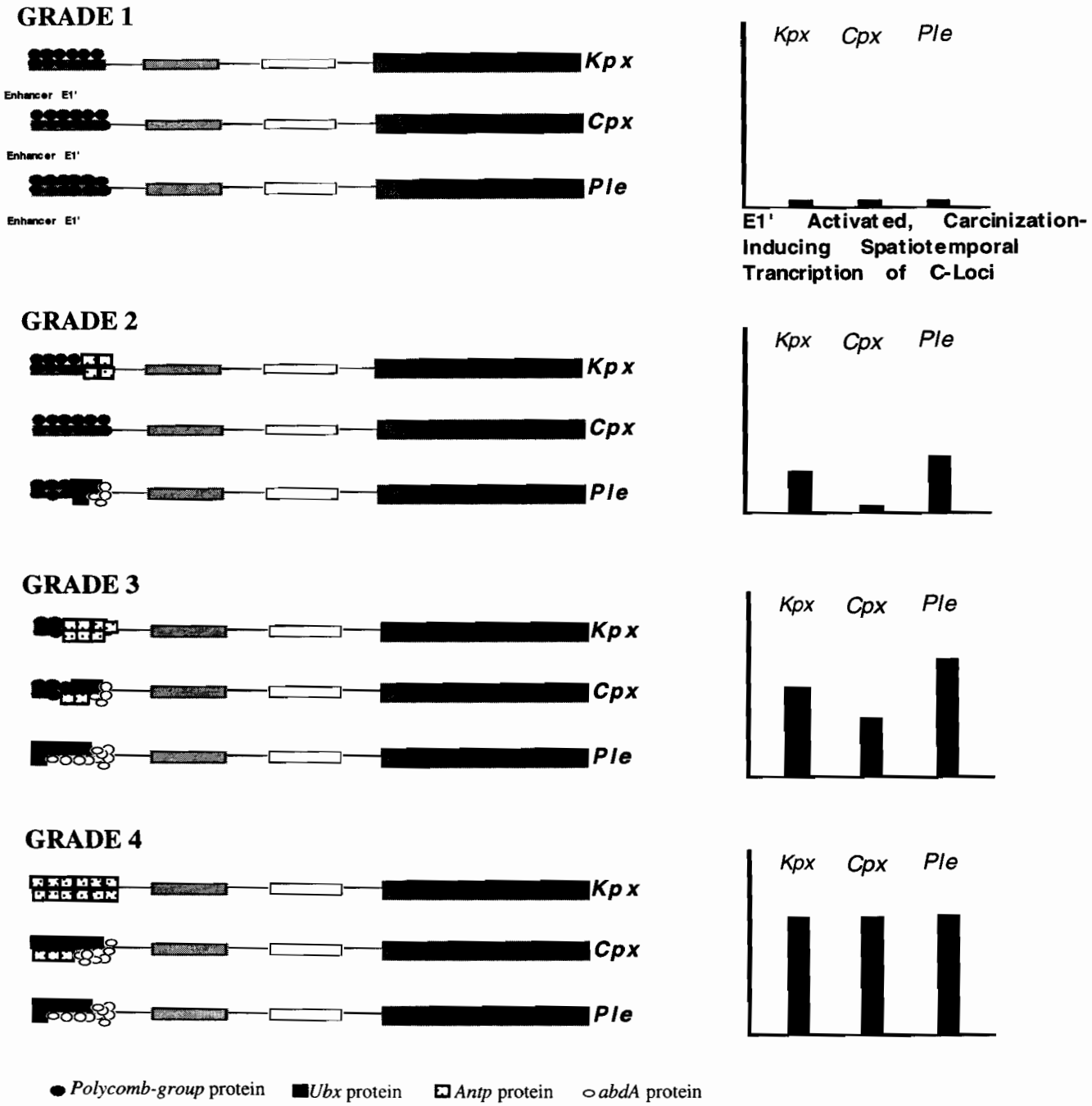

Figure 2. A model depicting grades of carcinization as entailing transistions in the state of the meiuran-specific regulatory elements (enhancer E1') for hypothetical c-loci *kpx*, *cpx*, and *ple* .

Carcinization as a parallelism can thus be explained (in terms of gene regulation) as simple changes in the degree to which c-loci enhancers are heterochromatinized (in a gap gene determined manner) by *Pc-group* proteins. The evolution of Lithodids from a pagurid (Cunningham *et al.*, 1992; Richter and Scholtz, 1994), a grade 1 to grade 4 transition, can therefore be mechanistically explained by changes in the binding affinities in gap gene/*Pc-group* proteins or altered DNA sequence/chromatin environments around c-loci at many loci need not be required for carcinization to occur.

This model is based on the genetic evidence available concerning morphological and phylogenetic transformations in well studied taxa such as *Drosophila* (De Salle and Carew, 1992). This model makes several predictions which can be tested. First, it predicts that c-loci are a) homeotic and b) few in number.

Determining the expression patterns of homeotic genes (perhaps using antibodies to known homeotic proteins in a manner similar to that employed by Averof and Akam, 1995) in the developing carapace, cephalothorax structures, and abdomen of carcinized and noncarcinized meiurans and other reptant decapods should provide clues as to the specific genes involved. This aspect of the model would be falsified if it were demonstrated that many loci are involved in carcinization and/or these loci are nonhomeotic in character (e.g., loci involved in cell-cell recognition). Modifier loci are of course expected to have a role in specifying the particulars of the carapace, cephalothorax, and abdomen. The asymmetrical abdomen of some female lithodids (Richter and Scholtz, 1994) would be an example of sex-limited modifier gene action.

Second, carcinization involves changes not in c-loci number but in c-loci regulation. The finding that carcinized meiurans possess c-loci not found in noncarcinized meiurans would directly falsify this model. And third, this model predicts that the *Pc-group* loci influence the degree to which carcinization takes place. That is, carcinization as a genetic potentiality waiting to be expressed h , its molecular basis in c-loci regulatory elements. The manifestation of this genetic potentiality is thus based on the interaction of *Hox* and *Pc-group* loci products with c-loci regulatory elements. Monitoring the expression of *Pc-group* proteins in carcinized *versus* noncarcinized decapods would be a way to investigate this prediction. Ideally (although far-fetched) mutations in some taxon like *Birgus,* mutations where the 'hermit crab habitus' is retained into maturity, would serve for testing the role of *Pc-group* loci in suppressing the carcinized phenotype.

Trivial (nonfalsifying) findings would entail identifying not 3 but 4 or 5 homeotic c-loci or finding that changes in c-loci regulation involved silencer elements, promoters, or changes in chromsomal position. Needless to say, determining that the Anomura and the Brachyura have different c-loci or even modes of c-loci regulation would necessitate rejection of this hypothesis.

[An alternative hypothesis of simple genetic changes undergirding carcinization, not to be explored here, would be the repression of meiuran-specific c-loci regulatory elements by the *Antp*, *Ubx*, and *abdA* products (Carrol, 1994). Parallel and graded carcinization would thus result from mutations in the meiuran-specific regulatory elements which gradually and independently released the c-loci from *Hox* repression.]

Canalized Evolutionary Potential, Regulatory Circuits, and Synapomorphies

The concept of a canalized evolutionary potential (Saether, 1983; Brundin, 1986) was introduced to account for the tendency of related clades to evolve similar morphological features (i.e., parallelisms). Inherent in this concept is the interpretation of rampant parallelisms, occurring within sister taxa, as indicative of a common genomic substrate. To quote Saether (1983):

> *There appears to be a good analogy between genetics on the species level and phylogenetics on the supraspecific level* (p. 355) ... *parallelisms... are expressions of the canalized evolutionary potential of a monophyletic group.* (p. 356)

This canalized evolutionary potential or, as I have termed it, 'genetic potentiality', refers to genetic networks which are synapomorphic for a lineage but which are variably expressed in members of the clade. Sluys (1989) referred to such genetic potentialities as 'underlying synapomorphies' in the sense that rampant parallelisms and the gene networks which underlie them are as valuable as traditional synapomorphies in determining phylogenetic relationships only more difficult to discern. This thinking runs contrary to the current wisdom in phylogenetics where parallelisms are to be avoided if at all possible. Homoplasies can nevertheless tell us much about the phylogenetic process and, in particular, the genetic-ontogenetic initiating conditions of complex structures.

The finding that key regulatory loci and gene regulatory machinery are conserved across phyla lends support to the idea that parallelisms and convergences may be nothing more than the reactivation of dormant regulatory circuits (Zuckerkandl, 1994). Assuming that these regulatory circuits are immanent to the genome, clades sharing a common genomic background would be expected to have a high incidence of the same circuits being switched on during their evolutionary tenure. According to Zuckerkandl's view, synapomorphies *sensu stricto*, parallelisms, and convergences are but different degrees of the same thing. The distinction between synapomorphies, parallelisms as underlying synapomorphies, and convergences would thus be based on the degree to which a regulatory pathway is expressed and the phylogenetic distribution of the pathway. Synapomorphies as such are expressed in all members of the sister clades for which the derived character states are characteristic. Underlying synapomorphies, in contrast, are variably

expressed between members of the sister clades although the rampant parallelisms are restricted to the sister clades. Convergences, e.g., the emergence of isomorphic taxa during the radiation of the placental and marsupial mammals, can likewise be interpreted as the expression of plesiomorphic regulatory networks that arose in the ancestor to the protomammal. The reader should note that to base synapomorphies or plesiomorphies -- i.e., aspects of *Baupläne* and *Unterbaupläne* -- in gene regulatory networks is not an attempt to reduce the morphological level to the DNA level. It is only to suggest that these regulatory networks act as initiating conditions, indeed 'enablements of potentialities', during ontogenesis (Salthe, 1993).

The concept of carcinization as an underlying synapomorphy is compatible with both neodarwinian and structuralist conceptions of phylogenesis. For example, Mayr (1988) wrote:

Groups of closely related species have exceedingly similar genotypes. There is great potential among such species to develop independently the same synapomorph characters. (p. 387)

and

In many, if not most, phyletic lines there is an indication of trends...It is now quite obvious that such trends are the necessary consequence of the unity of genotype which greatly constrains evolutionary potential. (p. 435)

Shubin (1991) similarly wrote:

The existence of a "bauplan", and a small number of process that may perturb it, makes the existence of evolutionary parallelism and convergence highly likely. This, in fact, is the case and the parallelism that is observed is generally predictable from the "bauplan" itself... (p. 415)

Working from the structuralist standpoint (Goodwin, 1989) one could similarly argue that the generative rules for carcinization are an emergent aspect of the Meiura, rules which are stabilized by *Hox* and homeotic genes. Thus carcinization as an underlying synapomorphy may appear unorthodox but it is in principle compatible with both synthetic and nonsynthetic interpretations of morphological evolution.

One last point. Loci which are cryptic for millions of years would be expected to undergo mutations and drift out of functionality (Marshall *et al.*, 1994). Loci, particularly regulatory loci, may nevertheless have different roles in different tissues and during different ontogenetic periods. Thus cryptic c-loci in paguroids, for example, could remain functional if these same loci have other roles uncoupled from carcinization as the enhancer elements in the model proposed here suggests (Fig. 1).

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