Evolvability in vertebrate segmentation

James E Hammond[1†], Callum V Bucklow[1], Berta Verd[1††]

[1]Department of Biology, University of Oxford, Oxford, UK.
[†]james.hammond@biology.ox.ac.uk [††]berta.verdfernandez@biology.ox.ac.uk

Abstract

The number of vertebrae in the axial skeleton of vertebrates is extremely diverse, and reflects adaptations to a diverse range of habitats and lifestyles. The capacity for heritable evolutionary change in the number of vertebrae - its evolvability - is underpinned by the process of somitogenesis, which determines the number of somites that form in the early embryo. However, despite the evolvability of somitogenesis having been crucial for the success of the vertebrates across evolutionary history, the developmental sources of evolvability in somitogenesis are still unknown. Here, we review the evolution of somitogenesis and vertebral number, and attempt to identify sources of evolvability within this important developmental process.

Introduction

The body plans of vertebrates are extremely diverse [1, 2, 3, 4, 5, 6]. A major contributor to this diversity is the number of vertebrae in the axial skeleton, which can range from as few as 7 in diminutive terrestrial frogs from the *Paedophryne* genus [7], to more than 750 in deep-sea *Nemichthys* Snipe eels [8, 9, 4]. The capacity for variation or 'evolvability' in the number of vertebrae varies between clades: in some groups this variation is low and the number can be taxonomically definitive [4]while in others, this number can be highly variable, for instance in the Ophidiidae Cusk-eels which vary from 58 to 91 vertebrae in total [8] or in African cichlids (Pseudocrenilabrinae) where vertebral counts are more modest but still vary with a range from 25 to 40 [10, 11]. This capacity has been a major driver of vertebrate evolution, as changes in vertebral number have enabled vertebrates to colonise new niches across evolutionary history [12, 13, 14]. However, the developmental origins of this evolvability remain elusive and poorly understood.

The development of the vertebrae is a well-characterised process, where the number of vertebrae is specified in early development by the number of somites [15, 16], paired blocks of mesoderm that segment the developing trunk body, and give rise to the skeletal muscle and axial skeleton [15]. Somitogenesis is a highly dynamic process, with somites arising sequentially from an unsegmented region of mesoderm at the posterior of the developing embryo, known as the pre-somitic mesoderm (PSM). The process by which somite boundaries are pre-patterned in the PSM is well-characterised, and is thought to be driven by a complex molecular oscillator known as the 'segmentation clock' that drives travelling waves of synchronised gene expression from the PSM posterior to the anterior [17, 18, 19]. The total number of somites formed is thought to be inversely proportional to the frequency of the segmentation clock [17, 20, 21], and the dynamics of the clock are known to vary across species [22, 23], for instance in the Zebrafish (*Danio rerio*) the clock oscillates approximately once every 30 minutes [24, 19], every 90 minutes in the Chicken (*Gallus gallus*) [18], every 120 minutes in the Mouse (*Mus musculus*) [25], or 320 minutes in Humans [25], suggesting that evolution of the clock may be involved in generating the diversity of vertebral number. While we are now beginning to understand the processes that give rise to such a diversity of dynamics [25, 23], the developmental sources of evolvability in somitogenesis remain elusive.

Understanding evolvability, the capacity of a biological systems to evolve heritable and adaptive phenotypes, and its relationship to the mapping between genotype and phenotype, is a major research goal within evolutionary developmental biology (EvoDevo) [26, 27, 28, 29, 30]. While the term 'evolvability' is widely credited to Dawkins (1989) [31], who studied whether a lineage's capacity to evolve and adapt under natural selection could itself evolve using *in silico* life forms, the study of how properties of development may affect the trajectory of evolution is itself older [27, 26, 32, 33]. Evolvability is thought to be determined by a variety of processes, such as the rates of mutation and recombination of genetic material [34], phenotypic plasticity in response to environmental factors [35], stochastic gene expression and resulting phenotypic heterogeneity [36], pleiotropy [37], canalisation and robustness of the genotype-phenotype map [38, 39], the complexity of the genotype-phenotype map [33, 40], and the way in which adaptive or fit phenotypes are selected by evolution [41, 42].

While much of the work on evolvability has been theoretical, there now exists a large corpus of experimental studies investigating the causes of evolvability and their evolution reviewed in [43]. Experiments involving techniques suited to microorganisms, such as experimental mutagenesis [44, 45], laboratory studies of evolution [46, 47, 48, 42] or design of synthetic gene regulatory networks [36, 49] have proven extremely powerful in furthering our understanding of evolvability. However outside of a handful of examples [50, 51, 52], there are relatively few experimental or data-driven studies of the developmental bases of evolvability in multicellular systems. This is in part due to the experimental challenges involved in performing comparative studies of development between closely related taxa, and also because in order to determine the causality of development in evolvability, realistic and accurate data-driven mathematical models are required [26, 32, 27]. With this in mind, the study of vertebral number in vertebrates provides an excellent model system for understanding the developmental sources of evolvability as there exists a rich repertoire of data-driven mathematical models describing the segmentation clock [53, 54, 55, 56, 57, 58] and an increasingly large body of comparative data [22, 23]. Here, we examine the evolvability of vertebral number in vertebrates and discuss possible sources of evolvability within

somitogenesis.

Evolvability of segment number

The number of vertebrae in the vertebrate axial skeleton exhibits a high degree of evolvability, as can be seen in the ray-finned fish (Figure 1). Here, we see repeated independent transitions between low and high counts at a family-level resolution (Figure 1), with the same pattern of repeated independent transitions in vertebral number being present within clades [8, 59], and at the infra-family scale, e.g. within African cichlids (Cichlidae, Pseudocrenilabrinae) (Figure 1) [10, 11]. Vertebral number is also known to vary between populations of the same species, e.g. in the Medaka *Oryzias latipes* which ranges from 30 to 31 vertebrae [60] (Figure 1), the Arctic Charr *Salvelinus alpinus* (58 to 64) [61], the Kōaro *Galaxias brevipinnis* (57 to 62) [62], the Northern pike *Esox lucius* (56-63)[63], or the Deer mouse *Peromyscus maniculatus* (23 to 27 caudal vertebrae) [14]. While some of this variation may be attributable to environmentally driven developmental plasticity [63, 64], in at least the Pike, Medaka, and Deer mouse such variation has been shown to be heritable [63, 60, 14]. It therefore appears that heritable changes in vertebral number can occur under presumably minor or at least evolutionarily rapid changes to the genotype, and thus that vertebral number exhibits a high degree of evolvability.

Vertebral number is also an adaptive trait: longer tails with more vertebrae permit forest populations of Deer mice to climb better than their prairie-dwelling congeners [14]. Deep sea fish show typically elongate eel-like bodies, thought to be an adaptation for energy-efficient anguilliform swimming under increased hydrostatic pressure [12]. In some cases, this is associated with extreme increases in the number of vertebrae [9]. Similarly, fossorial snakes possess smaller vertebrae relative to their body than fast-moving terrestrial or arboreal species [1], which could in principle arise via an increase in the number of vertebrae while reducing the length of the vertebrae to preserve overall body length but allowing greater flexibility, as may possibly be required for such a lifestyle. In a similar way, fast predatory fish such as Barracuda or Scombriformes are known to have relatively few vertebrae compared with other similarly elongate fish [65], which may stiffen their body axis, giving better propulsion when pursuing prey [66].

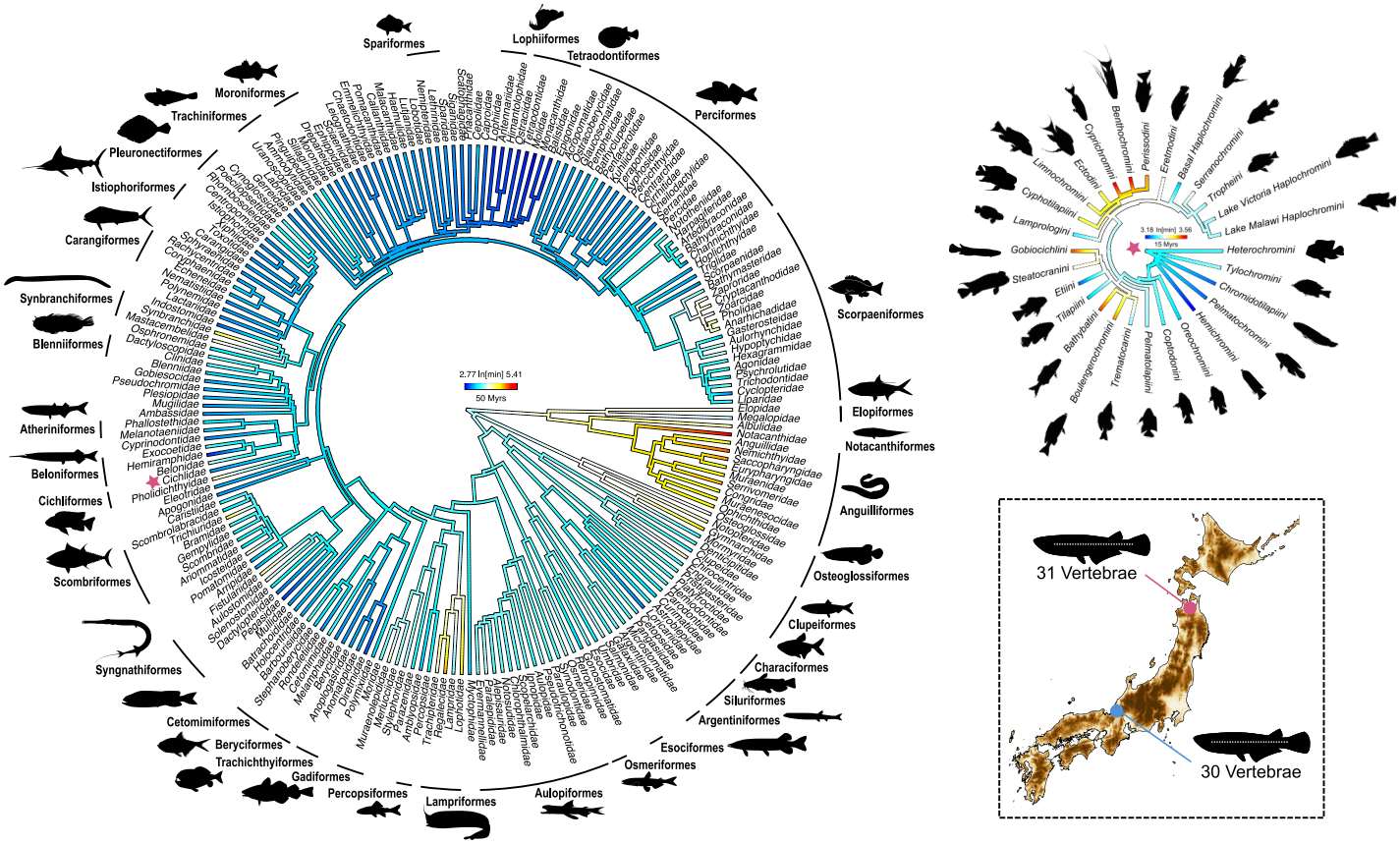

Figure 1: **Diversity in vertebral number across the bony fishes.**
Left: Family-level phylogeny of the Actinopterygii after [67], with tips coloured according to the log-transformed minimum number of vertebrae reported for that family by [4]. Silhouettes were downloaded from PhyloPic (`https://www.phylopic.org/`). **Top right:** Tribe-level phylogeny of the african Cichlidae, after [68]. Tips coloured according to the log-transformed minimum number of vertebrae from the corresponding tribe ([11]). Silhouettes were downloaded from PhyloPic or created in Inkscape. **Bottom right:** Different populations of japanese Medaka (*Oryzias latipes*) show heritable differences in vertebral number ([60]).

Development of vertebral number and its evolution

The number of vertebrae in the adult vertebrate is determined by the number of somites that form in the early embryo [15, 16]. Somites are paired transient blocks of mesoderm that segment the trunk anterior-posterior axis, and determine the future segmentation of the body's musculature, nervous system, and axial skeleton [15]. The number of somites is partially controlled by the frequency of an intracellular oscillator known as the segmentation clock, which synchronises differentiation of cells in the pre-somitic mesoderm (PSM) and thus controls the timing of the regular budding of cells out of the PSM to form the somites [17, 18, 20].

The action of the clock is most often explained in terms of the clock and wavefront model [17, 69]. In brief, this model assumes that the phase of intracellular oscillations in gene expression are 'read-out' by development at a point in space known as the wavefront. This wavefront moves posteriorly as the embryo elongates, and thus the temporal information from the oscillator can be read out into a spatially periodic pattern [17]. The identity and nature of the wavefront is controversial but is generally thought to be under the control of FGF signalling [70, 20, 71, 72, 69], most likely via ERK signalling [73, 74, 75]. The coupling of this wavefront to elongation of the embryo is thought to be, at least in part, due to the progressive decay of FGF mRNA in PSM cells, creating a gradient of positional information [76] from the posterior to the anterior. The identity of clock components is less controversial and there is strong evidence that the clock is driven by auto-repressive feedback of genes in the Hes/Her family [18, 77, 53, 78, 79, 80]. Intracellular oscillations of Hes/Her transcription factors are noisy [81, 82], are synchronised by delta-notch signalling [83, 81, 54, 80], and decrease in frequency towards the PSM anterior [84], creating travelling waves of gene expression which travel along the PSM towards the anterior [81, 19].

The frequency of the clock thus determines the total number of somites (and therefore vertebrae) that can be formed [20, 85, 86]. However, the model also implies that the duration of somitogenesis is causal in determining the number of vertebrae in the body. Developmental control of the duration of somitogenesis is less well-understood, and it is thought to depend both on gene regulatory and morphogenetic processes [87, 20, 88, 14]. For one, the total number of somites in the embryo correlates with how much the PSM elongates over the course of somitogenesis [22, 88], which has been explained by termination of somitogenesis being induced by a threshold value of retinoic acid (RA), an anterior-posterior decreasing gradient which is secreted from the somites, and thus somitogenesis terminates when the PSM is sufficiently short [20, 89]. In this way, it is thought that maintenance of PSM length over time by morphogenesis determines the duration of somitogenesis, and with it, of the total number of vertebrae formed. What controls the maintenance of the PSM over the course of somitogenesis is unclear, however the point in time at which PSM elongation halts has been shown to correlate with the expression of Hox13 genes [90, 91, 92, 93, 94, 95], and temporal changes in Hox13 expression have been implicated in evolutionary changes in vertebral number in reptiles and mice [91, 14].

It is however unclear from the current literature whether one of these two processes is most often employed by evolution to alter an organism's vertebral number, and if so which one. To explore the relative importance of changes to clock frequency or duration of somitogenesis in evolution, we performed a literature survey, cataloguing the number of somites formed during embryogenesis and the rate of their appearance in different vertebrate species (see supplementary material). We can imagine three scenarios that would help explain these data (Figure 2A). In scenario one, diversity in somite number is solely explained by evolution of the clock frequency, and we see a positive correlation between somite number and frequency (Figure 2A, left). In scenario two, where diversity in somite number can be explained by evolution of the duration of somitogenesis alone, we would expect to see the data lie along a vertical line when we plot the number of somites against the clock frequency (Figure 2A). The third scenario is that the diversity in segment number is due to co-evolution of these two processes, and we would expect to see the data spread along each axis (Figure 2A).

Plotting the total number of somites formed in the embryo against the rate of somitogenesis (see supplementary material for methods and data), we see no clear trend, and that the frequency of somitogenesis and the total number of somites is extremely diverse across different groups of vertebrates (Figure 2B). Within our dataset, the slowest rate of somite formation is the Common snapping turtle (*Chelydra serpentina*), with one somite being formed every 19 hours [96], and the quickest is the Mexican tetra (*Astyanax mexicanus*), with one somite being formed every 20 minutes [97]. However, for appropriate comparison between species the rate of somitogenesis must be scaled against the overall pace of development, which varies across taxa [25, 23]. We note that the period of somitogenesis (i.e., the time taken to form one somite) correlates well with the time taken for the embryo to reach the phylotypic stage (defined by the first appearance of the pharyngeal arches), and the slow frequency of somite formation in species like the Common snapping turtle or the Common marmoset (*Callithrix jacchus*) can largely be explained by their overall slower pace of development (Figure 2C). This is similar to the results of [23] who observe that the period of the segmentation clock *in vitro* scales with the overall pace of development [23]. Scaling the frequency against this overall rate of development (see supplementary material for methods) and plotting against the number of somites reveals that the total number of somites correlates with frequency, but that the data are broadly heterogenous and are best explained by scenario three (Figure 2D).

Here we see that some species, such as the Japanese striped snake *Elaphe quadrivirgata* or the Corn Snake *Pantherophis guttatus* exhibit accelerated clocks and their large numbers of somites can be at least partially attributed to this fact (Figure 2D) [22]. We also see that some active predatory fish such as the European seabass (*Dicentrarchus labrax*) form few somites and have correspondingly slow clocks (Figure 2D), suggesting that perhaps this reflects selection for a stiffer vertebral column with fewer vertebrae [65, 66]. However, some species such as the Long spiky-head carp (*Luciobrama macrocephalus*) possess rapid clocks, approaching the snakes' in their frequency, but do not form as many somites (Figure 2D). This suggests that the duration of somitogenesis is shorter relative to the frequency of the clock in the Long spiky-head carp than in snakes and highlights that even in cases where the clock is dramatically accelerated, evolution of duration cannot be discounted when considering the evolution of somite number [22].

We also note, as reported by [23], that the species of mammals analysed here have roughly equivalent somitogenesis frequencies when one accounts for differences in developmental rate (Figure 2D), suggesting that perhaps differences in mammalian vertebral number are not driven by changes in the clock but rather differences in the duration of somitogenesis [14]. The evolution of the mammalian vertebral column is thought to be highly constrained by its regionalisation, with number of pre-sacral vertebrae being highly conserved [27, 98]. It is possible therefore that this invariance in scaled clock frequency reflects a constraint on the pace of the clock relative to the pace of the 'Hox timer' or the sequential activation of Hox genes in the embryo [99] - assuming such a timer also scales with the overall pace of development - thereby ensuring a constant number of vertebrae per axial region. While this is speculative, the accessibility of non-model mammalian species for developmental biology is rapidly improving [23], and it may soon be possible to verify if the developmentally-adjusted frequency of the clock is indeed a conserved feature of mammals.

This analysis also reveals that when the overall pace of development is accounted for, the Testudines (turtles, terrapins, and tortoises) have a very slow rate of somite formation (Figure 2D). The three species of Testudines in our dataset, the Common snapping turtle, the Green sea turtle (*Chelonia mydas*), and the Red-bellied short-necked turtle (*Emydura subglobosa*), all have similarly low frequencies of somite formation (Figure 2D), suggesting this is possibly an ancestral feature of the Testudines. The functionality of this is unclear, but perhaps could reflect an adaptation for decreased vertebral number and increased rigidity of the axial skeleton [100]. Study of the Testudine segmentation clock may prove to be illuminating for understanding the evolvability of vertebral number, particularly in terms of understanding the clock's capacity for change in both dynamics and regulatory architecture, as well as a comparison with snakes, which appear to have taken the diametrically opposite path in evolution (Figure 2D).

We also note that the frequency of somite formation is approximately the same in the Sea lamprey (*Petromyzon marinus*) and the Brownbanded bamboo shark (*Chiloscyllium punctatum*) (Figure 2D). Whether this is coincidental or not remains to be seen, however we consider it of note due to the position of both species at the base of the vertebrate tree of life [101, 102]. If such a trend were to hold more generally and the rate of somitogenesis is conserved between the cyclostomes and elasmobranchs it would suggest that the extant evolvability of the clock arose only later in vertebrate evolution. Data on somitogenesis is currently extremely limited within these clades and sparse in general, but as more data become available we suggest that the field take note of any conservation of somitogenesis frequency.

Overall, this analysis suggests that both the morphogenesis of the PSM and the clock co-evolve to generate diversity in segment number. We cannot identify from this dataset whether or not one process is more labile under evolution than the other. To do so, it would be necessary to have data from closely related species that had diverged in vertebral counts relatively recently.

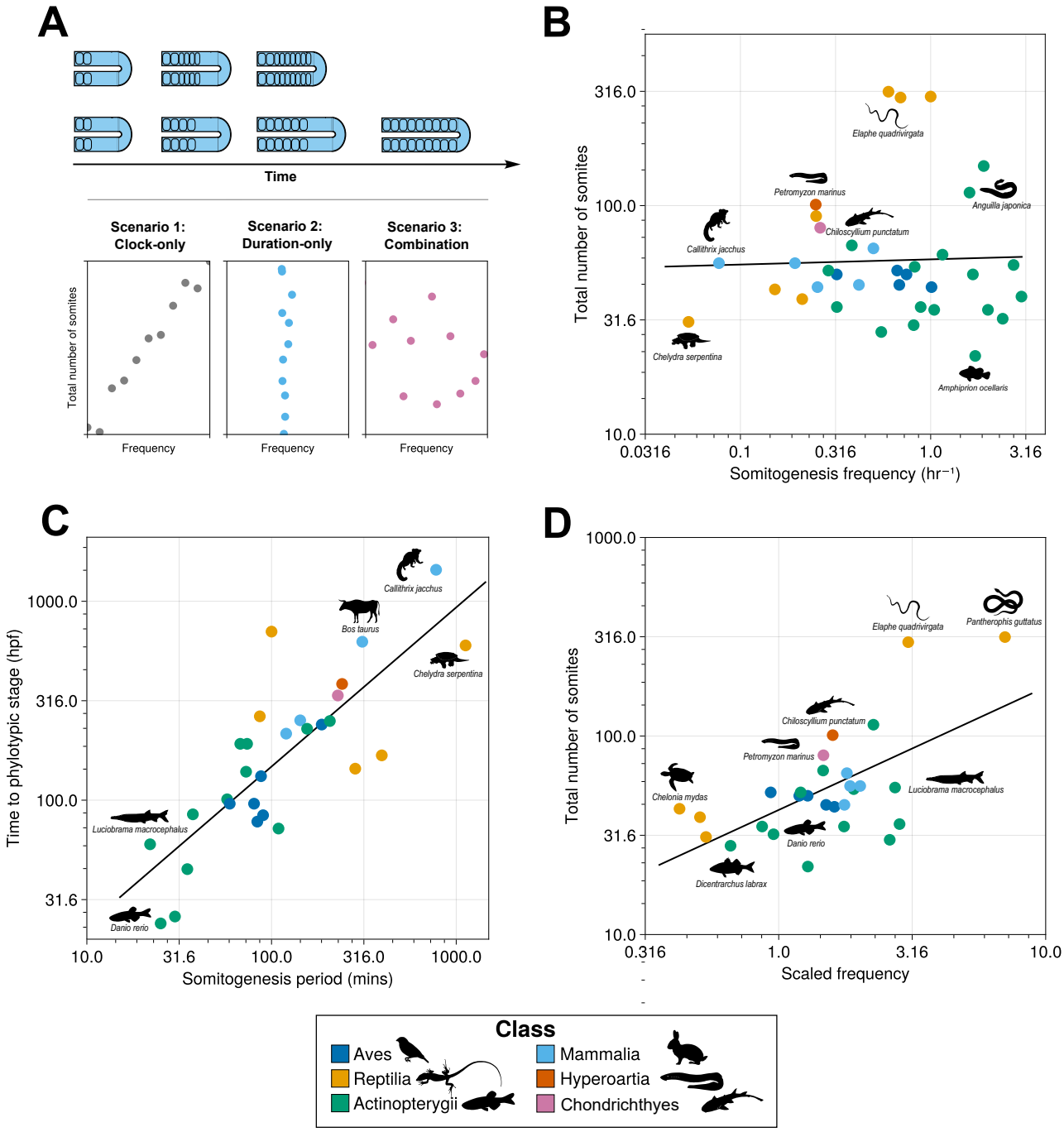

Figure 2: **Diversity in somitogenesis dynamics across the vertebrates.**
A Top: the same number of somites (black rectangles) can be achieved by changes in either frequency (top) or duration (bottom) of somitogenesis. **Bottom:** Scenarios for plotting the total number of somites formed during embryogenesis against somitogenesis frequency. If most of diversity is due to only evolutionary changes in frequency, the data should lie along a linear trendline (left, black). Conversely if most of diversity is due to only changes in duration, the data should lie along a vertical line (middle, blue). If diversity is created by a combination of the two, the data is heterogenously spread across the space (right, mauve). **B** The total number of somites formed during embryogenesis, plotted against the frequency at which somites are formed. A linear trendline is fitted from regression of the log-transformed data with trendline $y = 0.023x - 1.765$ with the summary statistics $p = 0.8866$, $R^2 = 0.001$, suggesting that there is no correlation between the dependent and independent variables. **C** The time taken to reach phylotypic stage (defined as presence of four pharyngeal arches), plotted against the somitogenesis period. A linear trendline is fitted from regression of the log-transformed data with trendline $y = 0.8x - 0.568$ with the summary statistics $p << 0.01$, $R^2 = 0.645$, suggesting a statistically significant relationship. As the magnitude of this trendline $0.8 \approx 1$, the relationship between the raw data is approximately linear. **D** The total number of somites formed during embryogenesis, plotted against the somitogenesis period divided by the time taken to reach phylotypic stage. A linear trendline is fitted from regression of the log-transformed data with trendline $y = 0.619x + 1.628$ with the summary statistics $p = 0.0006$, $R^2 = 0.368$, suggesting a statistically significant relationship with poor explanatory power. Code and data with references, for all figures, can be found in the supplementary material.

Developmental sources of evolvability

While the segmentation clock is thought to be driven by auto-repressive feedback on Hes/Her gene expression, the clock contains a large number of oscillatory and non-oscillatory components [103]. It is possible that this has been a rich substrate for evolution. The formation of non-DNA-binding Hes/Her dimers is known to accelerate clock oscillations [85, 78], and therefore duplication of Hes/Her family genes and their expression in the PSM could potentially result in changes to the frequency of the clock. The Hes/Her family contains many paralogs within the vertebrates [104], and Hes/Her family genes have been shown to have undergone repeated duplications across the metazoa [105, 106, 107, 108], suggesting that perhaps the appearance of novel paralogs or the neo- or sub-functionalisation of paralogous genes in this rapidly-duplicating family may be a major source of evolvability for clock dynamics. Indeed, the identity of Hes/Her oscillatory genes in the clock can vary between species of teleosts [105], suggesting a role for neo-functionalisation. Similarly, altering the copy number of delta genes has been shown to increase the emergent frequency of the clock as a result of elevated clock coupling between cells [109]. Lastly, the presence of paralogs can also create redundancy within the clock, such as in the case of the *her1* and *her7* genes in zebrafish [78]. It is possible that such redundancy has allowed neutral exploration of genotypes in evolution, allowing the discovery of novel, highly divergent clock dynamics and expression [38]. We can expect the role of gene duplication in the evolvability of somitogenesis to become clearer as the availability of comparative data increases.

It is possible that a similar property exists for the morphogenetic processes underpinning elongation of the PSM. Across different vertebrate species elongation of the PSM is known to be driven by cell division [88], as well as ingression of nascent PSM progenitor cells from surrounding tissues [110, 88, 111], and dynamic changes in cell motility [112, 113, 111, 114] and cell density [112, 115, 116]. The degree to which any one of these processes contributes to elongation of the PSM appears to vary across species [22, 88], which could suggest that diverse PSM elongation dynamics can be generated by the combinatorial effects of these processes. If this were the case, then the existence of a set of uncoupled morphogenetic processes such as these could be regarded as a source of evolvability for somitogenesis, however whether such processes are uncoupled is as yet deeply unclear and the development of cell-based models of PSM elongation [112, 115, 58, 117, 118] will reveal whether this is the case, and with it, the scope for neutral exploration of the mechanisms underlying PSM elongation by evolution.

The topology of the segmentation clock gene regulatory network may also be a source of evolvability, specifically the existence of two oscillatory circuits: one being the Hes/Her auto-repressive loop, and the other the negative feedback of delta-notch signalling onto itself via intercellular signalling [53]. Using a simple model, Lewis [53] demonstrated that the relatively long time delay created by intercellular signalling allows oscillations with much lower frequency to be created when auto-repression of Hes/Her expression is decreased, and speculated the covariation in these two processes could perhaps give rise to different clock frequencies in evolution [53]. However, simulations suggest the existence of a parameter space with irregular oscillations as the relative contribution of delta/notch to the circuit increases, and thus the evolution of this system may actually be constrained in this regard [53]. Inferring the relative contribution of each circuit in determining the clock's frequency is a hard problem as it requires experimental parametrisation of this model, and to our knowledge there have not been comparative studies examining the contributions of each circuit nor their change in evolution.

The delay in intercellular communication via delta-notch is also likely to exert constraints on the evolution of the clock gene regulatory network, and vice versa. In order for stable synchronous oscillations to be formed the delay in intercellular signalling must satisfy a relationship with the frequency of auto-repressive intracellular oscillations of Hes/Her genes where signals are sent and received by cells in the correct phase of the clock [53, 119]. The delay in intercellular signalling is thought in part to be controlled by the number of intermediary molecules, such as lunatic fringe (Lfng), involved in the signal transduction pathway [120], and therefore the number of intermediary components in signal transduction may be constrained by the frequency of Hes/Her oscillations. We also can expect the converse to be true, and that the frequency of Hes/Her oscillations is constrained by the existing signalling delay. However, the collective frequency of oscillations is also set by the value of the delay [53, 119], and so it may be that evolutionarily neutral regions of parameter space exist where the delay and the frequency may co-vary without perturbing the overall frequency. Only with further theoretical study will it become clear to what capacity the delay and frequency of the clock mutually constrain one another's evolution.

It is unclear how the behaviour of the oscillatory components of the FGF/Wnt pathways [121, 122, 103, 123], which are capable of changing their expression dynamics in evolution [103] might affect segmentation clock dynamics. Indeed, in Mouse at least it appears that the dynamics of Wnt and Notch signalling are coupled, with functional significance for somite polarity [123]. However, there is limited comparative data on whether this is a conserved feature of vertebrates and as such we cannot predict in what way such regulation may affect the evolution of the clock. Additionally, we note that as PSM morphogenesis is itself under the regulation of Wnt and FGF signalling [112, 110, 113, 114, 75], the evolution of morphogenesis and the dynamics of the clock may be prone to co-vary.

Vertebral number has been shown to also depend on maternal effects [124, 125], as have the period of somitogenesis and its duration [124]. The total duration of somitogenesis has also been shown to correlate with the amount of yolk available to the embryo [88], and the number of somites been shown to be lowered in conjoined twin embryos of the Lake trout *Salvelinus namaycush*, leading to a suggestion that the number of vertebrae depends on the quantity of yolk available to the embryo [126]. Maternal contribution to the offspring can vary with the age of the mother [127], suggesting this as a possible source of phenotypic heterogeneity in otherwise genetically homogenous populations, however it is unknown whether age-specific effects occur in the vertebral number of offspring.

Vertebral number is also known to exhibit environmentally-induced phenotypic plasticity, with vertebral number responding to changes in salinity, light, and temperature [128, 63, 64]. Due to their external mode of development, this has been most extensively studied in the teleost fishes.

The environmental effect of temperature here is perhaps counter-intuitive, as the frequency of somitogenesis increases with increasing temperature [129, 130, 24], whereas it is typically observed in nature that lower temperatures yield fish with more vertebrae [131, 60, 63, 64], and indeed the relationship between embryo temperature and vertebral number is thought to be U-shaped, where the maximal number of vertebrae occurs at extremely low or extremely high temperatures and at intermediary temperatures the lowest number of vertebrae are formed [128, 132]. The developmental basis of such a trend is unclear. Furthermore, the effect of temperature on somitogenesis is thought to be buffered by the invariance of somite length [24], likely due to a scaling in wavefront and clock molecular kinetics [133]. It is likely therefore that this scaling relationship, or any scaling relationship that exists between the frequency and duration of somitogenesis, breaks down at extreme temperatures.

While our understanding of somitogenesis and the segmentation clock has benefitted from a large body of theoretical work, it is still difficult to definitively identify sources of evolvability within somitogenesis. This is in large part due to a still incomplete understanding of the gene regulatory architecture underpinning the segmentation clock and the wavefront, as well as the computationally complex problem of understanding how cellular behaviours such as motility and division give rise to the elongation of the PSM, and the added difficulty of parameterising such models using experimental data. Furthermore, comparative studies of somitogenesis have largely been limited to vertebrate model species that are significantly diverged [105, 22, 103], and there have been few comparative studies examining how vertebral number and the components and dynamics of somitogenesis co-vary over short evolutionary timescales [14, 134], so it is difficult to suggest from observational data where the sources of evolvability may lie.

Modularity of the segmentation clock and PSM elongation

Modularity, the dissociability of developmental processes and pathways by evolution, is thought to be a major source of evolvability across the tree of life [28, 135], though evolvable systems can also exist without modular organisation [136]. The evolvability of diverse phenotypes such as butterfly eyespots [137], beak shape in the Galapágos finches [138], arthropod limbs [139, 140], arthropod segmentation [51], and the regionalisation of the vertebrate skeleton [8, 99], is thought to be underpinned by modularity of development.

In a recent study, we predicted that the clock and morphogenesis of the PSM exhibit modularity in zebrafish, and thus, that this might heighten the evolvability of vertebral number [117]. We predicted that the modularity is dependent on properties of morphogenesis such as tissue length and density, but more so on properties of the clock such as the strength of delta-notch signalling and the coupling delay [117]. Due to a lack of comparative experimental data we were only able to predict that this is true in zebrafish, however we see no reason to expect why this property is restricted to zebrafish and not a conserved feature of the vertebrates, particularly in light of the apparently combinatorial effects of somitogenesis pace and duration in driving diversity of vertebral number (Figure 2).

Understanding the selective or neutral ways in which modularity arises in evolution is an open question in evolutionary biology [28, 30, 141]. To our knowledge somitogenesis is one of the few developmental systems where modularity has been predicted to depend on such a small set of experimentally tractable parameters, and thus we suggest that a quantitiative study of the evolution of the segmentation clock and the PSM may be extremely insightful for this question.

Conclusions and Future Work

While much is known about somitogenesis and the developmental control of vertebral number, the developmental basis of its evolvability are still unclear.However, many of the properties of the segmentation clock and PSM morphogenesis can be placed in the context of previous theoretical work to predict possible sources of evolvability. The reasons for this lack of understanding are twofold, and reflect a lack of comparative studies with closely-related species, as well as of experimentally parameterised models of somitogenesis in different species. We note that technological advances are increasingly allowing the field to undertake comparative studies of somitogenesis [23], and suggest that these technologies be employed to study closely related groups of organisms to understand the relative lability of the various components of somitogenesis in evolution. We also suggest that the study of the reptiles, particularly a comparative study of the Serpentes and Testudines, could be illuminating in understanding the extreme limits of somitogenesis and elucidate which changes to somitogenesis can generate extreme diversity.

Methods

Mapping of vertebral counts to phylogenies was completed in R (v4.2.0) [142]. Ranges for 271 Teleostean orders and families were collated primarily from Nelson's 'Fishes of the World' [4] and for the tribes and subtribes of Pseudocrenilabrinae from previously reported data [10, 11]. We pruned the time-calibrated, bony fishes phylogeny published by [67] to the families present within our vertebral counts dataset in the R package, ape (v5.7.1) [143]. To reduce each family to a single branch and to account for the estimated age of each respective family, the tree was pruned to the longest (i.e. most basal) branch within each family, an approach we utilised again to prune the Pseudocrenilabrinae phylogeny published by [68] to each respective tribe and subtribe. Log-transformed minimum total vertebral and ancestral states were mapped and inferred using the function contMap, part of the phytools (v2.1.1) package [144]. R code for generating the figures can be found in the supplementary file *mapping_vertebral_counts.R*.

The data in Figure 2 reflects either reported values for the period and frequency of somitogenesis, or values that have been inferred by linear regression of the reported number of somites over time (see supplementary table 1). Linear regressions between frequency and the number of somites, or period and the time to phylotypic stage, were performed in Julia v1.8.2 using the function GLM.lm. Julia code for generating the figures can be found in the supplementary file *somitogenesis_pace.jl*.

Bibliography

[1] Ralph Gordon Johnson. "The Adaptive and Phylogenetic Significance of Vertebral Form in Snakes". In: *Evolution* 9.4 (1955), pp. 367–388. ISSN: 00143820, 15585646. URL: http://www.jstor.org/stable/2405473 (visited on 09/24/2024).

[2] LE Lindell. "The evolution of vertebral number and body size in snakes". In: *Functional Ecology* (1994), pp. 708–719. DOI: 10.2307/2390230.

[3] Johannes Müller et al. "Homeotic effects, somitogenesis and the evolution of vertebral numbers in recent and fossil amniotes". In: *Proceedings of the National Academy of Sciences* 107.5 (2010), pp. 2118–2123. DOI: 10.1073/pnas.0912622107.

[4] Joseph S Nelson, Terry C Grande, and Mark VH Wilson. *Fishes of the World*. John Wiley & Sons, 2016.

[5] Yimeng Li et al. "Divergent vertebral formulae shape the evolution of axial complexity in mammals". In: *Nature Ecology & Evolution* 7.3 (2023), pp. 367–381. DOI: 10.1038/s41559-023-01982-5.

[6] L. Roberts and J. Head. "Independent origins of vertebral complexity in tetrapods". In: *Research Square* (2024). DOI: 10.21203/rs.3.rs-4508905/v1.

[7] Eric N Rittmeyer et al. "Ecological guild evolution and the discovery of the world's smallest vertebrate". In: *PLoS one* 7.1 (2012), e29797.

[8] Andrea B. Ward and Elizabeth L. Brainerd. "Evolution of axial patterning in elongate fishes". In: *Biological Journal of the Linnean Society* 90.1 (Jan. 2007), pp. 97–116. DOI: 10.1111/j.1095-8312.2007.00714.x.

[9] William Beebe and Jocelyn Crane. "Deep sea fishes of the Bermuda oceanographic expeditions. Family Nemichthyidae". In: *Zoologica : scientific contributions of the New York Zoological Society* 22 (1937), pp. 349–383. URL: https://www.biodiversitylibrary.org/part/184688.

[10] Michael K Oliver. "African cichlid fishes: morphological data and taxonomic insights from a genus-level survey of supraneurals, pterygiophores, and vertebral counts (Ovalentaria, Blenniiformes, Cichlidae, Pseudocrenilabrinae)". In: *Biodiversity Data Journal* 12 (2024), e130707. DOI: 10.3897/BDJ.12.e130707.

[11] Callum Bucklow et al. "A whole-body micro-CT scan library that captures the skeletal diversity of Lake Malawi cichlid fishes". In: *Scientific Data* 11 (Sept. 2024). DOI: 10.1038/s41597-024-03687-1.

[12] FC Neat and N Campbell. "Proliferation of elongate fishes in the deep sea". In: *Journal of Fish Biology* 83.6 (2013), pp. 1576–1591.

[13] Qi-Ling Liu et al. "Rapid neck elongation in Sauropterygia (Reptilia: Diapsida) revealed by a new basal pachypleurosaur from the Lower Triassic of China". In: *BMC Ecology and Evolution* 23.1 (2023), p. 44. DOI: 10.1186/s12862-023-02150-w.

[14] Evan P Kingsley et al. "Adaptive tail-length evolution in deer mice is associated with differential Hoxd13 expression in early development". In: *Nature Ecology & Evolution* 8.4 (2024), pp. 791–805.

[15] Bodo Christ and Charles P Ordahl. "Early stages of chick somite development". In: *Anatomy and embryology* 191 (1995), pp. 381–396.

[16] Elizabeth M Morin-Kensicki, Ellie Melancon, and Judith S Eisen. "Segmental relationship between somites and vertebral column in zebrafish". In: *Development* 129 (16 2002). DOI: 10.1242/dev.129.16.3851.

[17] J. Cooke and E.C. Zeeman. "A clock and wavefront model for control of the number of repeated structures during animal morphogenesis". In: *Journal of Theoretical Biology* 58.2 (1976), pp. 455–476. DOI: 10.1016/s0022-5193(76)80131-2.

[18] Isabel Palmeirim et al. "Avian hairy gene expression identifies a molecular clock linked to vertebrate segmentation and somitogenesis". In: *Cell* 91 (5 1997), pp. 639–648. DOI: 10.1016/s0092-8674(00)80451-1.

[19] Daniele Soroldoni et al. "A Doppler effect in embryonic pattern formation". In: *Science* 345.6193 (2014), pp. 222–225. DOI: 10.1126/science.1253089.

[20] Céline Gomez and Olivier Pourquié. "Developmental control of segment numbers in vertebrates". In: *Journal of Experimental Zoology Part B: Molecular and Developmental Evolution* 312.6 (2009), pp. 533–544. DOI: 10.1002/jez.b.21305.

[21] Christian Schröter and Andrew C Oates. "Segment number and axial identity in a segmentation clock period mutant". In: *Current Biology* 20.14 (2010), pp. 1254–1258.

[22] Céline Gomez et al. "Control of segment number in vertebrate embryos". In: *Nature* 454.7202 (2008), pp. 335–339. DOI: 10.1038/nature07020.

[23] Jorge Lázaro et al. "A stem cell zoo uncovers intracellular scaling of developmental tempo across mammals". In: *Cell Stem Cell* 30.7 (2023), pp. 938–949.

[24] Christian Schröter et al. "Dynamics of zebrafish somitogenesis". In: *Developmental dynamics* 237.3 (2008), pp. 545–553. DOI: 10.1002/dvdy.21458.

[25] Mitsuhiro Matsuda et al. "Species-specific segmentation clock periods are due to differential biochemical reaction speeds". In: *Science* 369.6510 (2020), pp. 1450–1455. DOI: 10.1126/science.aba7668.

[26] Brian C Goodwin. "Development and evolution". In: *Journal of Theoretical Biology* 97.1 (1982), pp. 43–55. DOI: 10.1016/0022-5193(82)90275-2.

[27] J Maynard Smith et al. "Developmental constraints and evolution: a perspective from the Mountain Lake conference on development and evolution". In: *The Quarterly Review of Biology* 60.3 (1985), pp. 265–287.

[28] Günter P Wagner and Lee Altenberg. "Perspective: Complex adaptations and the evolution of evolvability". In: *Evolution* 50 (3 1996). DOI: 10.1111/j.1558-5646.1996.tb02339.x..

[29] Marc Kirschner and John Gerhart. "Evolvability". In: *Proceedings of the National Academy of Sciences* 95.15 (1998), pp. 8420–8427.

[30] Massimo Pigliucci. "Is evolvability evolvable?" In: *Nature Reviews Genetics* 9.1 (2008), pp. 75–82.

[31] Richard Dawkins. "The evolution of evolvability". In: *Artificial life*. Routledge, 1989, pp. 201–220. ISBN: 9780429032769.

[32] George Oster and Pere Alberch. "Evolution and bifurcation of developmental programs". In: *Evolution* (1982), pp. 444–459. DOI: 10.1111/j.1558-5646.1982.tb05066.x.

[33] Michael Conrad. "The geometry of evolution". In: *BioSystems* 24.1 (1990), pp. 61–81. DOI: 10.1016/0303-2647(90)90030-5.

[34] Rodrigo S Galhardo, Philip J Hastings, and Susan M Rosenberg. "Mutation as a stress response and the regulation of evolvability". In: *Critical reviews in biochemistry and molecular biology* 42.5 (2007), pp. 399–435. DOI: 10.1080/10409230701648502.

[35] Mary Jane West-Eberhard. "Developmental plasticity and the origin of species differences". In: *Proceedings of the National Academy of Sciences* 102.suppl_1 (2005), pp. 6543–6549. DOI: 10.1073/pnas.0501844102.

[36] Zoltán Bódi et al. "Phenotypic heterogeneity promotes adaptive evolution". In: *PLoS biology* 15.5 (2017), e2000644. DOI: 10.1371/journal.pbio.2000644.

[37] Carolyn A Wessinger and Lena C Hileman. "Accessibility, constraint, and repetition in adaptive floral evolution". In: *Developmental biology* 419.1 (2016), pp. 175–183. DOI: 10.1016/j.ydbio.2016.05.003.

[38] Andreas Wagner. "Robustness and evolvability: a paradox resolved". In: *Proceedings of the Royal Society B: Biological Sciences* 275.1630 (2008), pp. 91–100. DOI: 10.1098/rspb.2007.1137.

[39] Anton Crombach et al. "Gap gene regulatory dynamics evolve along a genotype network". In: *Molecular biology and evolution* 33.5 (2016), pp. 1293–1307. DOI: 10.1093/molbev/msw013.

[40] Sam F Greenbury, Ard A Louis, and Sebastian E Ahnert. "The structure of genotype-phenotype maps makes fitness landscapes navigable". In: *Nature Ecology & Evolution* 6.11 (2022), pp. 1742–1752. DOI: 10.1038/s41559-022-01867-z.

[41] Isaac Salazar-Ciudad and Miquel Marín-Riera. "Adaptive dynamics under development-based genotype–phenotype maps". In: *Nature* 497.7449 (2013), pp. 361–364. DOI: 10.1038/nature12142.

[42] Jia Zheng, Ning Guo, and Andreas Wagner. "Selection enhances protein evolvability by increasing mutational robustness and foldability". In: *Science* 370.6521 (2020), eabb5962. DOI: https://doi.org/10.1126/science.abb5962.

[43] Joshua L Payne and Andreas Wagner. "The causes of evolvability and their evolution". In: *Nature Reviews Genetics* 20.1 (2019), pp. 24–38. DOI: 10.1038/s41576-018-0069-z.

[44] Karen S Sarkisyan et al. "Local fitness landscape of the green fluorescent protein". In: *Nature* 533.7603 (2016), pp. 397–401. DOI: 10.1038/nature17995.

[45] Andrei Papkou et al. "A rugged yet easily navigable fitness landscape". In: *Science* 382.6673 (2023), eadh3860. DOI: 10.1126/science.adh3860. URL: https://www.science.org/doi/abs/10.1126/science.adh3860.

[46] Eric J Hayden, Evandro Ferrada, and Andreas Wagner. "Cryptic genetic variation promotes rapid evolutionary adaptation in an RNA enzyme". In: *Nature* 474.7349 (2011), pp. 92–95. DOI: 10.1038/nature10083.

[47] William C Ratcliff et al. "Origins of multicellular evolvability in snowflake yeast". In: *Nature communications* 6.1 (2015), p. 6102. DOI: 10.1038/ncomms7102.

[48] Irit Levin-Reisman et al. "Antibiotic tolerance facilitates the evolution of resistance". In: *Science* 355.6327 (2017), pp. 826–830. DOI: 10.1126/science.aaj2191. URL: https://www.science.org/doi/abs/10.1126/science.aaj2191.

[49] Javier Santos-Moreno et al. "Robustness and innovation in synthetic genotype networks". In: *Nature Communications* 14.1 (2023), p. 2454. DOI: 10.1038/s41467-023-38033-3.

[50] Marie-Laure Dichtel-Danjoy and Marie-Anne Félix. "Phenotypic neighborhood and micro-evolvability". In: *Trends in Genetics* 20.5 (2004), pp. 268–276. DOI: 10.1016/j.tig.2004.03.010.

[51] Berta Verd, Nicholas AM Monk, and Johannes Jaeger. "Modularity, criticality, and evolvability of a developmental gene regulatory network". In: *Elife* 8 (2019), e42832. DOI: 10.7554/eLife.42832.

[52] Salem Mosleh et al. "Beak morphometry and morphogenesis across avian radiations". In: *Proceedings of the Royal Society B: Biological Sciences* 290.2007 (2023), p. 20230420. DOI: 10.1098/rspb.2023.0420. URL: https://royalsocietypublishing.org/doi/abs/10.1098/rspb.2023.0420.

[53] Julian Lewis. "Autoinhibition with transcriptional delay: A simple mechanism for the zebrafish somitogenesis oscillator". In: *Current Biology* 13.16 (2003), pp. 1398–1408. DOI: 10.1016/s0960-9822(03)00534-7.

[54] Ingmar H. Riedel-Kruse, Claudia Müller, and Andrew C. Oates. "Synchrony dynamics during initiation, failure, and rescue of the segmentation clock". In: *Science* 317.5846 (2007), pp. 1911–1915. DOI: 10.1126/science.1142538.

[55] Alan J Terry et al. "A spatio-temporal model of Notch signalling in the zebrafish segmentation clock: conditions for synchronised oscillatory dynamics". In: *PLoS One* 6.2 (2011), e16980. DOI: 10.1371/journal.pone.0016980.

[56] Susan D Hester et al. "A multi-cell, multi-scale model of vertebrate segmentation and somite formation". In: *PLoS computational biology* 7.10 (2011), e1002155. DOI: 10.1371/journal.pcbi.1002155.

[57] James Cotterell, Alexandre Robert-Moreno, and James Sharpe. "A local, self-organizing reaction-diffusion model can explain somite patterning in embryos". In: *Cell systems* 1.4 (2015), pp. 257–269. DOI: 10.1016/j.cels.2015.10.002.

[58] Koichiro Uriu et al. "From local resynchronization to global pattern recovery in the zebrafish segmentation clock". In: *eLife* 10 (2021). Ed. by Didier YR Stainier and Ingmar Riedel-Kruse, e61358. DOI: 10.7554/eLife.61358.

[59] Rita S Mehta et al. "Elongation of the body in eels". In: *Integrative and Comparative Biology* 50.6 (2010), pp. 1091–1105.

[60] Kazunori Yamahira and Takeshi Nishida. "Latitudinal variation in axial patterning of the medaka (Actinopterygii: Adrianichthyidae): Jordan's rule is substantiated by genetic variation in abdominal vertebral number". In: *Biological Journal of the Linnean Society* 96.4 (2009), pp. 856–866.

[61] CE Adams and PS Maitland. "Arctic charr in Britain and Ireland–15 species or one?" In: *Ecology of Freshwater Fish* 16.1 (2007), pp. 20–28.

[62] Robert M McDowall. "Variation in vertebral number in galaxiid fishes (Teleostei: Galaxiidae): a legacy of life history, latitude and length". In: *Environmental Biology of Fishes* 66.4 (2003), pp. 361–381.

[63] Petter Tibblin et al. "Causes and consequences of intra-specific variation in vertebral number". In: *Scientific Reports* 6.1 (2016), p. 26372. DOI: 10.1038/srep26372.

[64] Calum S. Campbell et al. "Evolvability under climate change: Bone development and shape plasticity are heritable and correspond with performance in Arctic charr (Salvelinus alpinus)". In: *Evolution & Development* 23.4 (2021), pp. 333–350. DOI: https://doi.org/10.1111/ede.12379. eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/ede.12379. URL: https://onlinelibrary.wiley.com/doi/abs/10.1111/ede.12379.

[65] Andrea B. Ward and Rita S. Mehta. "Axial Elongation in Fishes: Using Morphological Approaches to Elucidate Developmental Mechanisms in Studying Body Shape". In: *Integrative and Comparative Biology* 50.6 (Apr. 2010), pp. 1106–1119. ISSN: 1540-7063. DOI: 10.1093/icb/icq029. eprint: https://academic.oup.com/icb/article-pdf/50/6/1106/1755482/icq029.pdf. URL: https://doi.org/10.1093/icb/icq029.

[66] Yordano E Jimenez et al. "Flexibility is a hidden axis of biomechanical diversity in fishes". In: *Journal of Experimental Biology* 226.Suppl_1 (2023), jeb245308.

[67] Ricardo Betancur-R et al. "Phylogenetic classification of bony fishes". In: *BMC evolutionary biology* 17 (2017), pp. 1–40. DOI: 10.1186/s12862-017-0958-3.

[68] Matthew D McGee et al. "The ecological and genomic basis of explosive adaptive radiation". In: *Nature* 586.7827 (2020), pp. 75–79. DOI: 10.1038/s41586-020-2652-7.

[69] Cassandra McDaniel et al. "Spatiotemporal control of pattern formation during somitogenesis". In: *Science Advances* 10.4 (2024), eadk8937.

[70] Julien Dubrulle, Michael J McGrew, and Olivier Pourquié. "FGF signaling controls somite boundary position and regulates segmentation clock control of spatiotemporal Hox gene activation". In: *Cell* 106.2 (2001), pp. 219–232. DOI: 10.1016/s0092-8674(01)00437-8.

[71] M Fethullah Simsek and Ertuğrul M Özbudak. "Spatial fold change of FGF signaling encodes positional information for segmental determination in zebrafish". In: *Cell Reports* 24.1 (2018), pp. 66–78. DOI: 10.1016/j.celrep.2018.06.023.

[72] Sundar Ram Naganathan and Andrew Charles Oates. "Patterning and mechanics of somite boundaries in zebrafish embryos". In: *Seminars in cell & developmental biology*. Vol. 107. Elsevier. 2020, pp. 170–178. DOI: 10.1016/j.semcdb.2020.04.014.

[73] Ryutaro Akiyama et al. "An anterior limit of FGF/Erk signal activity marks the earliest future somite boundary in zebrafish". In: *Development* 141.5 (2014), pp. 1104–1109. DOI: 10.1242/dev.098905.

[74] Dini Wahyu Kartika Sari et al. "Time-lapse observation of stepwise regression of Erk activity in zebrafish presomitic mesoderm". In: *Scientific Reports* 8.1 (2018), p. 4335. DOI: 10.1038/s41598-018-22619-9.

[75] M Fethullah Simsek et al. "Periodic inhibition of Erk activity drives sequential somite segmentation". In: *Nature* 613.7942 (2023), pp. 153–159. DOI: 10.1038/s41586-022-05527-x.

[76] Julien Dubrulle and Olivier Pourquié. "fgf8 mRNA decay establishes a gradient that couples axial elongation to patterning in the vertebrate embryo". In: *Nature* 427.6973 (2004), pp. 419–422.

[77] Andrew C Oates and Robert K Ho. "Hairy/E (spl)-related (Her) genes are central components of the segmentation oscillator and display redundancy with the Delta/Notch signaling pathway in the formation of anterior segmental boundaries in the zebrafish". In: *Development* 129 (12 2002), pp. 2929–2946. DOI: 10.1242/dev.129.12.2929.

[78] Christian Schröter et al. "Topology and Dynamics of the Zebrafish Segmentation Clock Core Circuit". In: *PLOS Biology* 10.7 (July 2012), pp. 1–20. DOI: 10.1371/journal.pbio.1001364. URL: https://doi.org/10.1371/journal.pbio.1001364.

[79] Anna Trofka et al. "The Her7 node modulates the network topology of the zebrafish segmentation clock via sequestration of the Hes6 hub". In: *Development* 139.5 (2012), pp. 940–947. DOI: 10.1242/dev.073544.

[80] Olivier F Venzin and Andrew C Oates. "What are you synching about? Emerging complexity of Notch signaling in the segmentation clock". In: *Developmental Biology* 460.1 (2020), pp. 40–54. DOI: 10.1016/j.ydbio.2019.06.024.

[81] Kazuki Horikawa et al. "Noise-resistant and synchronized oscillation of the segmentation clock". In: *Nature* 441 (2006), pp. 719–723. DOI: 10.1038/nature04861.

[82] Alexis B Webb et al. "Persistence, period and precision of autonomous cellular oscillators from the zebrafish segmentation clock". In: *eLife* 5 (2016), e08438. DOI: 10.7554/eLife.08438.

[83] Yun-Jin Jiang et al. "Notch signalling and the synchronization of the somite segmentation clock". In: *Nature* 408.6811 (2000), pp. 475–479. DOI: 10.1038/35044091.

[84] Laurel A Rohde et al. "Cell-autonomous timing drives the vertebrate segmentation clock's wave pattern". In: *eLife* 13 (Dec. 2024). Ed. by Yasumasa Bessho and Didier YR Stainier, RP93764. ISSN: 2050-084X. DOI: 10.7554/eLife.93764. URL: https://doi.org/10.7554/eLife.93764.

[85] Christian Schröter and Andrew C. Oates. "Segment number and axial identity in a segmentation clock period mutant". In: *Current Biology* 20.14 (2010), pp. 1254–1258. DOI: 10.1016/j.cub.2010.05.071.

[86] Yukiko Harima et al. "Accelerating the tempo of the segmentation clock by reducing the number of introns in the Hes7 gene". In: *Cell Reports* 3.1 (2013), pp. 1–7. DOI: 10.1016/j.celrep.2012.11.012.

[87] Patrick PL Tam and Seong-Seng Tan. "The somitogenetic potential of cells in the primitive streak and the tail bud of the organogenesis-stage mouse embryo". In: *Development* 115.3 (1992), pp. 703–715. DOI: 10.1242/dev.115.3.703.

[88] Ben Steventon et al. "Species-specific contribution of volumetric growth and tissue convergence to posterior body elongation in vertebrates". In: *Development* 143.10 (2016), pp. 1732–1741. DOI: 10.1242/dev.126375.

[89] Gennady Tenin et al. "The chick somitogenesis oscillator is arrested before all paraxial mesoderm is segmented into somites". In: *BMC developmental biology* 10 (2010), pp. 1–12.

[90] Teddy Young et al. "Cdx and Hox genes differentially regulate posterior axial growth in mammalian embryos". In: *Developmental cell* 17.4 (2009), pp. 516–526. DOI: 10.1016/j.devcel.2009.08.010.

[91] Nicolas Di-Poi et al. "Changes in Hox genes' structure and function during the evolution of the squamate body plan". In: *Nature* 464.7285 (2010), pp. 99–103. DOI: 10.1038/nature08789.

[92] Joost Woltering. "From lizard to snake; behind the evolution of an extreme body plan". In: *Current genomics* 13.4 (2012), pp. 289–299. DOI: 10.2174/138920212800793302.

[93] Nicolas Denans, Tadahiro Iimura, and Olivier Pourquié. "Hox genes control vertebrate body elongation by collinear Wnt repression". In: *Elife* 4 (2015), e04379. DOI: 10.7554/eLife.04379.

[94] Zhi Ye and David Kimelman. "Hoxi3 genes are required for mesoderm formation and axis elongation during early zebrafish development". In: *Development* 147.22 (2020), dev185298. DOI: 10.1242/dev.185298.

[95] Lucille Lopez-Delisle et al. "CTCF-dependent insulation of Hoxb13 and the heterochronic control of tail length". In: *Proceedings of the National Academy of Sciences* 121.46 (2024), e2414865121. DOI: 10.1073/pnas.2414865121.

[96] CL Yntema. "A series of stages in the embryonic development of Chelydra serpentina". In: *Journal of morphology* 125.2 (1968), pp. 219–251. DOI: 10.1002/jmor.1051250207.

[97] Hélène Hinaux et al. "A developmental staging table for Astyanax mexicanus surface fish and Pachón cavefish". In: *Zebrafish* 8.4 (2011), pp. 155–165. DOI: 10.1089/zeb.2011.0713.

[98] Frietson Galis et al. "Fast running restricts evolutionary change of the vertebral column in mammals". In: *Proceedings of the National Academy of Sciences* 111.31 (2014), pp. 11401–11406. DOI: 10.1073/pnas.1401392111. eprint: https://www.pnas.org/doi/pdf/10.1073/pnas.1401392111. URL: https://www.pnas.org/doi/abs/10.1073/pnas.1401392111.

[99] Hocine Rekaik and Denis Duboule. "A CTCF-dependent mechanism underlies the Hox timer: relation to a segmented body plan". In: *Current Opinion in Genetics & Development* 85 (2024), p. 102160. DOI: 0.1016/j.gde.2024.102160.

[100] Tyler R. Lyson and Gabriel S. Bever. "Origin and Evolution of the Turtle Body Plan". In: *Annual Review of Ecology, Evolution, and Systematics* 51.Volume 51, 2020 (2020), pp. 143–166. ISSN: 1545-2069. DOI: https://doi.org/10.1146/annurev-ecolsys-110218-024746. URL: https://www.annualreviews.org/content/journals/10.1146/annurev-ecolsys-110218-024746.

[101] Daqi Yu et al. “Hagfish genome elucidates vertebrate whole-genome duplication events and their evolutionary consequences”. In: *Nature ecology & evolution* 8.3 (2024), pp. 519–535. DOI: 10.1038/s41559-023-02299-z.

[102] Ferdinand Marlétaz et al. “The hagfish genome and the evolution of vertebrates”. In: *Nature* 627.8005 (2024), pp. 811–820. DOI: 10.1038/s41586-024-07070-3.

[103] Aurélie J Krol et al. “Evolutionary plasticity of segmentation clock networks”. In: *Development* 138.13 (2011), pp. 2783–2792. DOI: 10.1242/dev.063834.

[104] Ryoichiro Kageyama, Toshiyuki Ohtsuka, and Taeko Kobayashi. “The Hes gene family: repressors and oscillators that orchestrate embryogenesis”. In: *Development* 134 (7 2007), pp. 1243–1251. DOI: 10.1242/dev.000786.

[105] Martin Gajewski et al. “Comparative analysis of her genes during fish somitogenesis suggests a mouse/chick-like mode of oscillation in medaka”. In: *Development genes and evolution* 216.6 (2006), pp. 315–332. DOI: 10.1007/s00427-006-0059-6.

[106] Walter L Eckalbar et al. “Somitogenesis in the anole lizard and alligator reveals evolutionary convergence and divergence in the amniote segmentation clock”. In: *Developmental Biology* 363.1 (2012), pp. 308–319. DOI: 10.1016/j.ydbio.2011.11.021.

[107] Eve Gazave, Aurélien Guillou, and Guillaume Balavoine. “History of a prolific family: the Hes/Hey-related genes of the annelid Platynereis”. In: *EvoDevo* 5 (2014), pp. 1–33. DOI: 10.1186/2041-9139-5-29.

[108] Aya Kuretani et al. “Evolution of hes gene family in vertebrates: the hes5 cluster genes have specifically increased in frogs”. In: *BMC Ecology and Evolution* 21 (2021), pp. 1–15. DOI: 10.1186/s12862-021-01879-6.

[109] Bo-Kai Liao, David J Jörg, and Andrew C Oates. “Faster embryonic segmentation through elevated Delta-Notch signalling”. In: *Nature Communications* 7.1 (2016), pp. 1–12. DOI: 10.1038/ncomms11861.

[110] Andrew K Lawton et al. “Regulated tissue fluidity steers zebrafish body elongation”. In: *Development* 140.3 (2013), pp. 573–582. DOI: 10.1242/dev.090381.

[111] Samhita P Banavar et al. “Mechanical control of tissue shape and morphogenetic flows during vertebrate body axis elongation”. In: *Scientific Reports* 11.1 (2021), pp. 1–14. DOI: 10.1038/s41598-021-87672-3.

[112] Bertrand Bénazéraf et al. “A random cell motility gradient downstream of FGF controls elongation of an amniote embryo”. In: *Nature* 466 (7303 2010), pp. 248–52. DOI: 10.1038/nature09151.

[113] Alessandro Mongera et al. “A fluid-to-solid jamming transition underlies vertebrate body axis elongation”. In: *Nature* 561.7723 (2018), pp. 401–405. DOI: 10.1038/s41586-018-0479-2.

[114] Arthur Michaut et al. “Activity-driven extracellular volume expansion drives vertebrate axis elongation”. In: *bioRxiv* (2022). DOI: 10.1101/2022.06.27.497799.

[115] Fengzhu Xiong et al. “Mechanical coupling coordinates the co-elongation of axial and paraxial tissues in avian embryos”. In: *Developmental Cell* 55.3 (2020), pp. 354–366. DOI: 10.1016/j.devcel.2020.08.007.

[116] Lewis Thomson, Leila Muresan, and Benjamin Steventon. “The zebrafish presomitic mesoderm elongates through compaction-extension”. In: *Cells & Development* 168 (2021), p. 203748. DOI: 10.1016/j.cdev.2021.203748.

[117] J.E. Hammond, R.E. Baker, and B. Verd. “Modularity of the segmentation clock and morphogenesis”. In: *bioRxiv* (2024). DOI: 10.1101/2024.01.08.574679.

[118] Michèle Romanos et al. “Differential proliferation regulates multi-tissue morphogenesis during embryonic axial extension: Integrating viscous modeling and experimental approaches”. In: *Development* (2024), dev–202836. DOI: 10.1242/dev.202836.

[119] Luis G Morelli et al. “Delayed coupling theory of vertebrate segmentation”. In: *HFSP journal* 3.1 (2009), pp. 55–66.

[120] Kumiko Yoshioka-Kobayashi et al. “Coupling delay controls synchronized oscillation in the segmentation clock”. In: *Nature* 580.7801 (2020), pp. 119–123. DOI: 10.1038/s41586-019-1882-z.

[121] Alexander Aulehla et al. “Wnt3a plays a major role in the segmentation clock controlling somitogenesis”. In: *Developmental cell* 4.3 (2003), pp. 395–406.

[122] Mary-Lee Dequéant et al. “A complex oscillating network of signaling genes underlies the mouse segmentation clock”. In: *science* 314.5805 (2006), pp. 1595–1598.

[123] Katharina F Sonnen et al. “Modulation of phase shift between Wnt and Notch signaling oscillations controls mesoderm segmentation”. In: *Cell* 172.5 (2018), pp. 1079–1090.

[124] Yong-Hua Sun et al. “Cytoplasmic impact on cross-genus cloned fish derived from transgenic common carp (Cyprinus carpio) nuclei and goldfish (Carassius auratus) enucleated eggs”. In: *Biology of reproduction* 72.3 (2005), pp. 510–515. DOI: 10.1095/biolreprod.104.031302.

[125] Antony J Praphu Philip et al. “Sequence of formation and inheritance of meristic variation in the post-cranial axial skeleton of Atlantic salmon (Salmo salar)”. In: *Journal of Fish Biology* (2024). DOI: 10.1111/jfb.16004.

[126] ET Garside and FEJ Fry. “A possible relationship between yolk size and differentiation in trout embryos”. In: *Canadian Journal of Zoology* 37.4 (1959), pp. 383–386. DOI: 10.1139/z59-044.

[127] Linsey M Arnold et al. "The role of maternal age and context-dependent maternal effects in the offspring provisioning of a long-lived marine teleost". In: *Royal Society open science* 5.1 (2018), p. 170966. DOI: 10.1098/rsos.170966.

[128] James A. Fowler. "Control of vertebral number in teleosts - an embryological problem". In: *The Quarterly Review of Biology* 45.2 (1970), pp. 148–167. URL: https://www.jstor.org/stable/2821324.

[129] Ian A Johnston, Vera LA Vieira, and Marguerite Abercromby. "Temperature and myogenesis in embryos of the Atlantic herring Clupea harengus". In: *Journal of Experimental Biology* 198.6 (1995), pp. 1389–1403. DOI: 10.1242/jeb.198.6.1389.

[130] Yuriy N Gorodilov. "Description of the early ontogeny of the Atlantic salmon, Salmo salar, with a novel system of interval (state) identification". In: *Environmental Biology of fishes* 47 (1996), pp. 109–127. DOI: 10.1007/BF00005034.

[131] R. M. McDowall. "Jordan's and other ecogeographical rules, and the vertebral number in fishes". In: *Journal of Biogeography* 35.3 (2008), pp. 501–508. DOI: 10.1111/j.1365-2699.2007.01823.x.

[132] Winer Daniel Reyes Corral and Windsor E Aguirre. "Effects of temperature and water turbulence on vertebral number and body shape in Astyanax mexicanus (Teleostei: Characidae)". In: *PLoS One* 14.7 (2019), e0219677. DOI: 10.1371/journal.pone.0219677.

[133] Weiting Zhang et al. "Fgf8 dynamics and critical slowing down may account for the temperature independence of somitogenesis". In: *Communications Biology* 5.1 (2022), p. 113. DOI: 10.1038/s42003-022-03053-0.

[134] Ali Seleit et al. "Modular control of vertebrate axis segmentation in time and space". In: *The EMBO Journal* 43.18 (2024), pp. 4068–4091. DOI: 10.1038/s44318-024-00186-2.

[135] Rudolf A. Raff. *The shape of life – genes, development and evolution of animal forms*. University of Chicago Press, 1996. ISBN: 0-226-70266-9.

[136] Thomas F Hansen. "Is modularity necessary for evolvability?: Remarks on the relationship between pleiotropy and evolvability". In: *Biosystems* 69.2 (2003), pp. 83–94. ISSN: 0303-2647. DOI: https://doi.org/10.1016/S0303-2647(02)00132-6. URL: https://www.sciencedirect.com/science/article/pii/S0303264702001326.

[137] Antónia Monteiro et al. "Mutants highlight the modular control of butterfly eyespot patterns". In: *Evolution & development* 5.2 (2003), pp. 180–187. DOI: 10.1046/j.1525-142X.2003.03029.x.

[138] Ricardo Mallarino et al. "Two developmental modules establish 3D beak-shape variation in Darwin's finches". In: *Proceedings of the National Academy of Sciences* 108.10 (2011), pp. 4057–4062. DOI: 10.1073/pnas.1011480108.

[139] Michalis Averof and Nipam H Patel. "Crustacean appendage evolution associated with changes in Hox gene expression". In: *Nature* 388.6643 (1997), pp. 682–686. DOI: 10.1038/41786.

[140] Terri A Williams and Lisa M Nagy. "Developmental modularity and the evolutionary diversification of arthropod limbs". In: *Journal of Experimental Zoology* 291.3 (2001), pp. 241–257. DOI: 10.1002/jez.1101.

[141] G. Wagner, M. Pavlicev, and J Cheverud. "The road to modularity". In: *Nature Reviews Genetics* 8 (2007). DOI: 10.1038/nrg2267.

[142] R Core Team. *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing. Vienna, Austria, 2022. URL: https://www.R-project.org/.

[143] Emmanuel Paradis and Klaus Schliep. "ape 5.0: an environment for modern phylogenetics and evolutionary analyses in R". In: *Bioinformatics* 35 (2019), pp. 526–528. DOI: 10.1093/bioinformatics/bty633.

[144] Liam J. Revell. "phytools 2.0: an updated R ecosystem for phylogenetic comparative methods (and other things)." In: *PeerJ* 12 (2024), e16505. DOI: 10.7717/peerj.16505.

[145] Pauline Salis et al. "The real Nemo movie: Description of embryonic development in Amphiprion ocellaris from first division to hatching". In: *Developmental Dynamics* 250.11 (2021), pp. 1651–1667. DOI: 10.1002/dvdy.354.

[146] T Kawakami et al. "Prolongation of somitogenesis in two anguilliform species, the J apanese eel A nguilla japonica and pike eel M uraenesox cinereus, with refined descriptions of their early development". In: *Journal of Fish Biology* 90.4 (2017), pp. 1533–1547. DOI: 10.1111/jfb.13249.

[147] Aleksandra Marconi et al. "Morphological and temporal variation in early embryogenesis contributes to species divergence in Malawi cichlid fishes". In: *Evolution & Development* 25.2 (2023), pp. 170–193. DOI: 10.1111/ede.12429.

[148] Hsin-Yuan Tsai et al. "Embryonic development of goldfish (Carassius auratus): a model for the study of evolutionary change in developmental mechanisms by artificial selection". In: *Developmental dynamics* 242.11 (2013), pp. 1262–1283. DOI: 10.1002/dvdy.24022.

[149] Tatsuya Kawakami et al. "Embryonic development and morphology of eggs and newly hatched larvae of Pacific herring Clupea pallasii". In: *Fisheries Science* 77 (2011), pp. 183–190. DOI: 10.1007/s12562-010-0317-4.

[150] Patricia Cucchi et al. "Embryonic development of the sea bass Dicentrarchus labrax". In: *Helgoland Marine Research* 66.2 (2012), pp. 199–209. DOI: 10.1007/s10152-011-0262-3.

[151] I von Herbing Hunt et al. "Effects of temperature on morphological landmarks critical to growth and survival in larval Atlantic cod (Gadus morhua)". In: *Marine Biology* 124 (1996), pp. 593–606. DOI: 10.1007/BF00351041.

[152] Thomas E Hall, Peter Smith, and Ian A Johnston. "Stages of embryonic development in the Atlantic cod Gadus morhua". In: *Journal of morphology* 259.3 (2004), pp. 255–270. DOI: 10.1002/jmor.10222.

[153] Tomoko Arakawa et al. "Stages of embryonic development of the ice goby (shiro-uo), Leucopsarion petersii". In: *Zoological science* 16.5 (1999), pp. 761–773. DOI: 10.2108/zsj.16.761.

[154] Zhishen Liang et al. "Spawning areas and early development of long spiky-head carp (Luciobrama macrocephalus) in the Yangtze River and Pearl River, China". In: *Hydrobiologia* 490 (2003), pp. 169–179. DOI: 10.1023/A:1023426909635.

[155] Takafumi Fujimoto et al. "Developmental stages and germ cell lineage of the loach (Misgurnus anguillicaudatus)". In: *Zoological Science* 23.11 (2006), pp. 977–989. DOI: 10.2108/zsj.23.977.

[156] Takashi Iwamatsu. "Stages of normal development in the medaka Oryzias latipes". In: *Mechanisms of Development* 121.7 (2004). Medaka, pp. 605–618. ISSN: 0925-4773. DOI: https://doi.org/10.1016/j.mod.2004.03.012. URL: https://www.sciencedirect.com/science/article/pii/S0925477304000486.

[157] HANS-ULRICH KOECKE. "NORMALSTADIEN DER EMBRYONAL-ENTWICKLUNG BEI DER HAUSENTE (ANAS BOSCH AS DOMESTIC A)". In: *Embryologia* 4.1 (1958), pp. 55–78. DOI: 10.1111/j.1440-169X.1958.tb00147.x.

[158] Gabriela Beatriz Olea and María Teresa Sandoval. "Embryonic development of Columba livia (Aves: Columbiformes) from an altricial-precocial perspective". In: *Revista Colombiana de Ciencias Pecuarias* 25 (2012), pp. 3–13. DOI: 10.17533/udea.rccp.324728.

[159] Bodo Christ, Ruijin Huang, and Jörg Wilting. "The development of the avian vertebral column". In: *Anatomy and embryology* 202 (2000), pp. 179–194. DOI: 10.1007/s004290000114.

[160] MASAHIRO YAMASAKI and AKIRA TONOSAKI. "Developmental Stages of the Society Finch, Lonchura striata var. dornestica." In: *Development, Growth & Differentiation* 30.5 (1988), pp. 515–542. DOI: https://doi.org/10.1111/j.1440-169X.1988.00515.x. eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1440-169X.1988.00515.x. URL: https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1440-169X.1988.00515.x.

[161] C. Herbert. "A times series of embryonic development stages of the Adelie penguin (Pygoscelis adeliae) from Signy Island, South Orkney Islands". In: *British Antarctic Survey Bulletin* 14 (), pp. 45–67. URL: https://nora.nerc.ac.uk/id/eprint/526667.

[162] Koh Onimaru et al. "A staging table for the embryonic development of the brownbanded bamboo shark (Chiloscyllium punctatum)". In: *Developmental Dynamics* 247.5 (2018), pp. 712–723. DOI: 10.1002/dvdy.24623.

[163] Michael K. Richardson, Jeroen Admiraal, and Glenda M. Wright. "Developmental anatomy of lampreys". In: *Biological Reviews* 85.1 (2010), pp. 1–33. DOI: https://doi.org/10.1111/j.1469-185X.2009.00092.x. eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/j.1469-185X.2009.00092.x. URL: https://onlinelibrary.wiley.com/doi/abs/10.1111/j.1469-185X.2009.00092.x.

[164] Jerrold T Haldiman. "Bovine somite development and vertebral anlagen establishment". In: *Anatomia, Histologia, Embryologia* 10.4 (1981), pp. 289–309. DOI: 10.1111/j.1439-0264.1981.tb00695.x.

[165] P. L. Chambers and J. P. Hearn. "Embryonic, foetal and placental development in the Common marmoset monkey (Callithrix jacchus)". In: *Journal of Zoology* 207.4 (1985), pp. 545–561. DOI: https://doi.org/10.1111/j.1469-7998.1985.tb04951.x. eprint: https://zslpublications.onlinelibrary.wiley.com/doi/pdf/10.1111/j.1469-7998.1985.tb04951.x. URL: https://zslpublications.onlinelibrary.wiley.com/doi/abs/10.1111/j.1469-7998.1985.tb04951.x.

[166] C Casteleyn et al. "Anatomical description and morphometry of the skeleton of the common marmoset (Callithrix jacchus)". In: *Laboratory Animals* 46.2 (2012). PMID: 22517992, pp. 152–163. DOI: 10.1258/la.2012.011167. eprint: https://doi.org/10.1258/la.2012.011167. URL: https://doi.org/10.1258/la.2012.011167.

[167] Chun-Hsiang Chang and Chern-Mei Jang. "On the Processing and Mounting of a Skeleton of a White Rhinoceros, Ceratotherium sinum". In: 2004. URL: https://api.semanticscholar.org/CorpusID:130776191.

[168] P. P. L. Tam. "The control of somitogenesis in mouse embryos". In: *Development* 65.Supplement (Oct. 1981), pp. 103–128. ISSN: 0950-1991. DOI: 10.1242/dev.65.Supplement.103. eprint: https://journals.biologists.com/dev/article-pdf/65/Supplement/103/3109051/develop_65_s_65-supplement-103.pdf. URL: https://doi.org/10.1242/dev.65.Supplement.103.

[169] S. Beaudoin, P. Barbet, and F. Bargy. "Developmental Stages in the Rabbit Embryo: Guidelines to Choose an Appropriate Experimental Model". In: *Fetal Diagnosis and Therapy* 18.6 (Oct. 2003), pp. 422–427. ISSN: 1015-3837. DOI: 10.1159/000073136. eprint: https://karger.com/fdt/article-pdf/18/6/422/2775259/000073136.pdf. URL: https://doi.org/10.1159/000073136.

[170] P. Proks et al. "Vertebral formula and congenital abnormalities of the vertebral column in rabbits". In: *The Veterinary Journal* 236 (2018), pp. 80–88. ISSN: 1090-0233. DOI: https://doi.org/10.1016/j.tvjl.2018.04.016. URL: https://www.sciencedirect.com/science/article/pii/S1090023318301345.

[171] Nawal Al-Mukhaini et al. "Embryonic staging of the Green Turtles, Chelonia mydas". In: *Zoology in the Middle East* 51 (Jan. 2010), pp. 39–50. DOI: 10.1080/09397140.2010.10638439.

[172] Yoshiyuki Matsubara et al. "Efficient embryonic culture method for the Japanese striped snake, laphe quadrivirgata, and its early developmental stages". In: *Development, Growth & Differentiation* 56.8 (2014), pp. 573–582. DOI: https://doi.org/10.1111/dgd.12157. eprint: https://onlinelibrary.wiley.com/doi/pdf/10.1111/dgd.12157. URL: https://onlinelibrary.wiley.com/doi/abs/10.1111/dgd.12157.

[173] Ingmar Werneburg et al. “Embryogenesis and ossification of Emydura subglobosa (Testudines, Pleurodira, Chelidae) and patterns of turtle development”. In: *Developmental Dynamics* 238.11 (2009), pp. 2770–2786. DOI: https://doi.org/10.1002/dvdy.22104. eprint: https://anatomypubs.onlinelibrary.wiley.com/doi/pdf/10.1002/dvdy.22104. URL: https://anatomypubs.onlinelibrary.wiley.com/doi/abs/10.1002/dvdy.22104.

Supplementary Material

Species	Class	Total somites	Period (mins)	Phylotypic stage (hpf)	Method	Source(s)
Amphiprion ocellaris	Actinopterygii	35	22	45	Reported	[145]
Anguilla japonica	Actinopterygii	37.5	114	85	Reported	[146], A. Seleit *pers. comm.*
Astatotilapia calliptera	Actinopterygii	73.7	30	192	Reported	[147]
Astyanax mexicanus	Actinopterygii	20	40	-	Reported	[97]
Crassius auratus	Actinopterygii	30	35	26	Reported	[148]
Clupea harengus	Actinopterygii	52	61	-	Reported	[129]
Clupea pallasii	Actinopterygii	72.8	54	139	Inferred	[149]
Danio rerio	Actinopterygii	25.1	32	24	Reported	[24]
Dicentrarchus labrax	Actinopterygii	109	28	72	Inferred	[150]
Gadus morhua	Actinopterygii	207	52	250	Reported	[151, 152]
Leucopsarion petersii	Actinopterygii	187	36	-	Inferred	[153]
Luciobrama macrocephalus	Actinopterygii	22	55	60	Inferred	[154]
Misgurnus anguillicaudatus	Actinopterygii	36	50	-	Reported	[155]
Muraenesox cinereus	Actinopterygii	31.6	149	-	Reported	[146]
Oryzias latipes	Actinopterygii	57.5	35	101	Reported	[156, 134]
Rhamphochromis sp. ‘Chilingali’	Actinopterygii	67.7	36	192	Reported	[147]
Salmo salar	Actinopterygii	155.8	67	228.5	Reported	[130]
Anas platyrhynchos	Aves	80.4	50	96	Inferred	[157]
Columba livia	Aves	59.4	44	96	Inferred	[158]
Gallus gallus	Aves	90	52	84	Reported	[18, 159]
Lonchura striata	Aves	87.7	45	132	Inferred	[160]
Pygoscelis adeliae	Aves	187	50	240	Inferred	[161]
Chiloscyllium punctatum	Chondrichthyes	228.5	80	336	Inferred	[162]
Petromyzon marinus	Hyperoartia	241	101	384	Inferred	[163]
Bos taurus	Mammalia	310	56	626	Inferred	[164, 23]
Callithrix jacchus	Mammalia	778	56	1440	Inferred	[165, 166, 23]
Ceratotherium simum	Mammalia	236	44	-	Inferred	[167, 23]
Mus musculus	Mammalia	120	65	216	Reported	[168]
Oryctolagus cuniculus	Mammalia	143	45	252	Inferred	[169, 170, 23]
Aspidoscelis uniparens	Reptilia	240	90	-	Reported	[22]
Chelonia mydas	Reptilia	395.2	43	168	Inferred	[171]
Chelydra serpentina	Reptilia	1123.6	31	600	Inferred	[96]
Elaphe quadrivirgata	Reptilia	86.4	297	264	Reported	[172]
Emydura subglobosa	Reptilia	284	39	144	Inferred	[173]
Lamprophis fuliginosus	Reptilia	60	300	-	Reported	[22]
Pantherophis guttatus	Reptilia	100	315	768	Reported	[22]

Table 1: **Somitogenesis period, time to phylotypic stage, and total somites, across species.** For each species the periodicity of somitogenesis, the time elapsed before one somite is formed, was either taken from a previously published value (‘reported’), or was inferred by linear regression of somites over time from published data (‘inferred’). The time taken to reach phylotypic stage, defined as the presence of pharyngeal arches, was not always reported and so in some cases this data is missing.