Venomous Dinosaurs and Rear-Fanged Snakes: Homology and Homoplasy Characterized

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Abstract

I develop an account of homology and homoplasy drawing on their use in biological inference and explanation. Biologists call on homology and homoplasy to infer character states, support adaptationist explanations, identify evolutionary novelties and hypothesize phylogenetic relationships. In these contexts, the concepts must be understood phylogenetically and kept separate: as they play divergent roles, overlap between the two ought to be avoided. I use these considerations to criticize an otherwise attractive view defended by Gould, Hall, and Ramsey & Peterson. By this view, homology and homoplasy can only be delineated *qua* some level of description, and some homoplasies (parallelisms) are counted as homologous. I develop an account which retains the first, but rejects the second, aspect of that view. I then characterize parallelisms and convergences in terms of their causal role. By the Strict Continuity account, homology and homoplasy are defined phylogenetically and without overlaps, meeting my restriction. Convergence and parallelisms are defined as two types of homoplasy: convergent homoplasies are largely constrained by external factors, while parallelisms are due to internal constraints.

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I Introduction

On one hand the biological world is wildly diverse, on the other surprisingly conservative. *Mammalia* alone boasts naked mole-rats, giraffes, whales and coendous (arboreal porcupines sporting prehensile tails); platypus, orang-utan, horseshoe bats and aye-ayes. The range of forms and behaviour is bewildering. Yet nothing is unique: whales retain vestiges of their ancestor's quadrupedal lifestyle; all mammals are hairy, milk-provisioning and back-boned. Moreover, the naked mole rat's strange eusocial lifestyle echoes that of ants and termites; the platypus' electroreception is found in many fish; the orang-utan's remarkable puzzle-solving is rivalled by kea. Point to some putatively unique trait in a lineage and I will find it in another. A central task of evolutionary biology is accounting for the diversity and conservatism of organismic form and function in an historical context. In virtue of what facts about their pasts are lineages similar and different? This paper is about the similarities. In particular, I discuss a cluster of concepts which biologists use to characterize and explain similarities between and within organisms. These concepts, 'homology', 'homoplasy', 'convergence', 'parallelism', and so on, are essential for the work of biologists and increasing amounts of philosophical ink are spilled on them. This paper presents an original approach to capturing these concepts, and a new view.

As a rough first pass, let's consider two explanations a biologist might give for some similarity between two organisms. Why do *Cetacea* have similar skeletal features to land-based mammals? Because cetaceans and terrestrial mammals share a common ancestor who had those features, and they *inherited* them. Call this 'homology'. Why do platypus and shark share electroreception? Because their ancestors lived in environments which faced similar problems – how do you locate prey in the dark? And their ancestors found similar solutions, they *independently* evolved electroreception. Call this 'homoplasy'. I have presented these as (1) contrasting *phylogenetic* concepts: two similar traits are homologous just in case they are present in the most recent common ancestor; homoplastic just in case they are *not* present in the common ancestor; (2) *explanations* of similarity in biological form. I ultimately defend a view similar in spirit, if not in detail.

I start by clarifying the paper's approach and situating it in current debate.

I focus almost exclusively on four concepts: *homology*, *homoplasy, convergence* and *parallelism*. Convergences and parallelisms are, by my account, two types of homoplasy. Roughly, if two lineages independently evolved similar traits from different starting points, or utilizing different developmental resources, they are *convergent*; if the traits evolved from similar starting points, or utilizing similar developmental resources, they are *parallel*. There are other concepts, such as 'analogy', 'atavism', 'reversal' and so on, which are relevant to the discussion at hand but, to avoid laborious definitions, I ignore.

I should say something about methodology. Many philosophers tackling homology and homoplasy take an explicitly historical perspective. They are in part interested in capturing how these concepts were understood in the 19th Century, and tracing the development of those ideas (Griffiths and Hall, for instance). This is a worthy approach, but it is not mine. I am interested in understanding homology and homoplasy in terms of their *epistemic roles*. I want to track not what scientists say about them, or how they were used in the past, but their use in a contemporary, justificatory, context. When scientists call on homology or homoplasy, what kinds of inferences and explanations are they concerned with, and what account makes the most sense of those epistemic roles?

An important dispute about homology concerns its role in *individuating* biological traits. Griffiths (1994, 2006) and Amundsen & Lauder (1994) argue that relations of homology (ancestry) are the best determinate of organismic traits. Neander & Rosenberg (Neander 2002, Rosenberg 2006, Neander & Rosenberg 2009) argue instead that selective role function is indispensable for delineating characters. In this paper I do not take a stand on this debate – I am interested in understanding homology, homoplasy and so on in terms of how they are used *once traits are individuated*. I take the two interests to be complimentary and hopefully everything I say conforms both to a homology-first or function-first approach to character delineation.

I should briefly mention two accounts of homology and homoplasy. The *taxic* or *geneological* account conceptualizes them as I have above: two traits between sister clades are homologous if and only if they are held by their most recent common ancestor. As put, this definition fails to account for *serial* homologues (my left and right hands are surely homologous, but as they are part of the same organism they cannot be homologous by this definition); and fails for homologues between ancestor-descendent pairs (surely my hands and my father's hands are homologous, but we are not in sister-clades). I think of homology as a relation of inheritance, but not as put by the taxic account. The *developmental* account of homology (Wagner 1994, Brigandt 2007) holds that two traits are homologous just in case they are the products of the same developmental process. This view is unsatisfactory because, as Hall demonstrates, homologous traits can arise from different developmental processes. The challenge facing characterizations of homology and homoplasy is to find an account which can play these various roles.

As I have said, I am interested in homology and homoplasy in terms of their epistemic roles in explanation and inference. Another approach understands homology as an *explanandum* rather than an *explanans*. For instance, Griffiths (2007) suggests that homology is an 'investigative kind': a robust phenomenon demanding explanation. He sees biological categorization as a descriptive (rather than explanatory) project which captures a phenomenon (homology). Explaining homologous patterns is a requirement of any complete biological science. He takes homology and homoplasy as I discuss them to be *explanations* of 'homology' as an investigative kind. He is a pluralist about the developmental and taxic account of homology as they are complementary explanations of homologous phenomena. The hierarchists (see below) and I present non-pluralistic accounts.

With this focus in mind, in section II I consider four epistemic uses homology or homoplasy. This grounds a restriction on accounts: on pain of undermining their epistemic roles, homology and homoplasy must be understood phylogenetically, and kept distinct. In section III I discuss a view proposed by Hall (2012, 2007, 2003) and Gould (2002) and developed by Leander (2008) and Ramsey & Peterson (2012). The position has two main tenets: (1) attributions of homology or homoplasy can only be made *qua* some level of description; (2) homoplasy is disjunctive (some homoplasies are homologous, and others not). I agree with the first, and resist the second part of the claim. Finally, Section IV presents a new view of homology and homoplasy. Importantly, this involves an account of parallelisms which both retains their homoplastic status and illuminates their epistemic role.

Before getting to the paper proper, there are two issues I want to partly sideline. First, are homologous traits are *the same* or merely *similar*? Ramsey & Peterson argue vigorously for a 'sameness' reading against Rosenberg & Neander's (and others) 'similarity' reading[1]. So far as I can tell, whatever hangs on this disagreement does not affect my arguments herein. In formulating my view I will remain agnostic to either reading. Second, one might worry that this discussion is merely semantic in the pejorative sense. For instance: does the disagreement between myself and Hall collapse into mere labelling? I believe it does not. At the very least, such labelling reflects scientific concepts, and getting this right *matters*. Second, I take it that one route to asserting ontological or epistemic claims is through *what works* in scientific practice. I think much of what follows demonstrates this.

In a nutshell, my approach is to argue for a restriction on accounts of homology and homoplasy in section II, use that to object to the hierarchical account in section III, and then build on that discussion for my own account in section IV.

II The epistemic role of homology and homoplasy

Homologous and homoplastic relationships, understood in phylogenetic terms and strictly delineated, play an important role in biological epistemology. A satisfactory account of homology and homoplasy must retain those roles. In this section I sketch four roles and explain how they motivate the restriction. For different epistemic tasks, biologists sometimes consider homology as noise and homoplasy as signal, and vice-versa. In splitting the evidential wheat from the chaff, we often divide along homoplastic/homologous lines – and this motivates keeping them distinct. One reason to care about this distinction is that an otherwise attractive view, the hierarchical, violates it.

I first characterize the use of homology and homoplasy in inferring the traits of lineages. Both homology and homoplasy play roles here, but on different justifications. Second, I turn to the uses of homology and

[1] To an extent I attribute this to Rosenberg & Neander because Ramsey & Peterson do. I don't think many philosophers before Ramsey & Peterson have taken talk of homology as 'the same' trait, versus homology as 'similar' traits particularly seriously. In Rosenberg & Neander's paper they do make some claims about similarity: "… similarity judgements, or at least specifications of the traits or characters judged to be homologous, are prior to homology claims (330)."

homoplasy in adaptationist explanation: accounting for some contemporary trait in terms of selection pressures in the past. As we shall see, homologous relationships play a framing role, while homoplastic relationships count as evidence. Third, I discuss the role of homology in identifying novel traits. Fourth, I cover the role homology plays in inferring shared ancestry. In each case, a phylogenetic conception is utilized, and overlaps between homology and homplasy would undermine their epistemic use.

I illustrate these uses in reference to Enpu Gong and colleagues (2009)'s argument that a small, feathered species of microraptor, *Sinornithosarus*, was venomous. Gong et al see the morphological features of *Sinornithosaurs* dentition as indicative of a venomous delivery system. They have unusually long (saber-toothed) maxillary teeth, extra space on the maxillary bone which could have accommodated a venom gland, and a supradental groove along the maxilla which they interpret as "represent[ing] the location of small venom reservoirs (3)." Their justification for taking these morphological characters as indicative of venom are analogies with venomous organisms with similar features. Opisthoglyphous (rear-fanged) snakes and squamate lizards have similar maxillary teeth and similar jaw structures. The space on the maxillary bone is interpreted as a venom gland on the basis of morphological similarities with these contemporary reptiles. Opisthoglyphous snakes and rear-fanged lizards deliver venom via poison mixing with saliva from their ducts. The force of the bite delivers the poison, rather than the 'injection' system seen in vipers. Rear-fanged snakes and lizards typically use venom to weaken, rather than kill, prey: the poison makes the victim easier to control. On this basis Gong and colleagues make further claims about the ecology and behaviour of the microraptor, suggesting that the poison fangs are an adaptation to a bird-eating lifestyle:

> … the long fangs in *Sinornithosaurus*… evolved to penetrate a covering of feathers and… it was largely a predator on feathered taxa… (2011, 110)

And so, from analogy with extant animals we have claims about *Sinornithosaurus'* morphology, behaviour, ecology and adaptive history:

> … *Sinornithosaurus* was a venomous predator that fed on birds by using its long fangs to penetrate through the plumage and into the skin, and the toxins would induce shock and permit the victim to be subdued rapidly (1).

Gong et al may be drawing a long bow here – but it is not the truth or otherwise of Gong et al's position that I am concerned with. Rather, it is the use of the comparative method they apply.

Homology & Homoplasy in trait-inference

Homologies and homoplasies are used to infer the presence or otherwise of traits. Paleontologists draw on comparisons between extant (and occasionally other extinct) lineages to put meat on fossilized bones. In systematics the characteristics of common ancestors are reconstructed on the basis of contemporary morphological patterns. The characteristics of contemporary but inaccessible animals, such as those in the

deep seas, are inferred on the basis of comparisons with lineages we can access (see Currie & Levy (forthcoming)'s discussion of Colossal squid). As we shall see, both homologies and homoplasies can help here, but do so on the basis of different justifications.

In their 2009 paper, Gong et al do not take a stance on whether the common ancestor of microraptors and Opisthoglyphous snakes had a venom-delivery system. Let us consider the status of their inference if the relationship is homoplastic or homologous on taxic grounds. In both situations, the inference moves from some match between a morphological trait (say, long maxillaries) to another (venom) in one lineage, to project that onto another lineage where the first trait is known, but the other is not. It might help to put this in some schematic form.

Take two lineages, call one a 'model' lineage, the other the 'target' lineage. We use the model lineage to reconstruct the target lineage, based on the similarities between the model and the target. We wish to discover whether the target lineage exhibits some character – call it character$_1$, and investigate on the basis of a similarity between the model and the target lineage, in some other character – character$_2$. Here is the basic structure:

1. In the model lineage, character$_1$ and character$_2$ are coupled
2. The target lineage exhibits character$_1$
3. Projections are justified: if character$_1$ and character$_2$ are coupled in the model lineage, then they most likely will be in the target lineage
4. Most likely, the target lineage exhibits character$_2$

Opoisthoglyphous snakes have venom and long maxillary fangs, and *Sinornithosaurus* has long maxillary fangs. Assuming that we can project from Opoisthoglyphous snakes to *Sinornithosaurus*, then given that coupling in the snake, we should expect the dinosaur to be venomous. So, what does the homoplastic or homologous nature of the similarity between snake and dinosaur have to do with the inference? How premise 3 is justified turns on these relations.

If the characters are *homologous*, the traits are basal: the common ancestor of the two lineages used maxillary fangs to deliver venom – and this was inherited. The inference is justified if it is more likely that the function and morphology of the teeth remain stable across generations, rather than shift by (say) drift or exaptation. If, for instance, we have reason to think that the trait is especially labile, then we might doubt the inference. If it is entrenched, we should be happy (in Currie & Levy (forthcoming) this is called a 'phylogenetic inference' made on the basis of 'phylogenetic inertia').

If the characters are *homoplastic*, two lineages evolved the maxillary fang-venom coupling independently. The justification of the projection here does not turn on the robustness of inheritance (at least not primarily – see section IV). It rather turns on our confidence in regularity: the relationship between maxillary teeth and venom in an evolutionary context. If there is selection for Opisthoglyphous venom-

delivery, then (most likely) long maxillary teeth will evolve. Or perhaps, if long maxillary teeth have evolved, this is most likely to accommodate Opisthoglyphous venom-delivery.

The justification for the inference (premise 3) differs depending on the ancestral relationship. If a homology is proposed, then the inference is justified on common ancestry and phylogenetic inertia. If a homoplasy is proposed, then the inference is justified on grounds of a coupling between two traits given some selective regime.

In an illuminating exchange, Gianechini et al (2011) criticize Gong et al, and they reply (2011). I focus on the comparative aspects of the discussion. As stated, Gong et al cite three traits as evidence for their microraptor's venom: (1) elongated teeth which could deliver venom; (2) labial grooves suggestive of venom ducts; (3) 'ornamentation' (extra space) on the maxillary bone which could accommodate venom glands. Gianechini et al object to each piece of evidence.

To (1) they point out that "… extremely elongated maxillary teeth are also observed in other therapod taxa… without any evidence of connection with a venom delivery system (104)"; for (2) they claim "… a wide variety of therapods, pertaining to distantly related clades, exhibit labial grooves… However, none of these therapods exhibit other evidence of venomous adaptations (104-105); and finally for (3) "... an almost identical ornamentation is also present in the antorbital and "subfenestral" fossae of other dromaeosaurids… but their dentition is considerably different from that of *Sinornithosaurus* (105)." In each case, they draw attention to the presence of characters 1-3 in non-venomous lineages.

Why do Gianechini et al care so much about the distribution of traits 1-3? Presumably Gong et al refer to rear-fanged snakes in order to infer, on the basis of homoplasy, that long fangs evolve to accommodate venom. By showing that other lineages have that trait, but are not venomous, this is undermined. The counter-examples to the analogy between Opoisthoglyphous snakes and microraptors suggest we cannot project from the coupling of long fangs and venom in the snake, to the dinosaur's fangs being indicative of venom. Of course this assumes that Gong et al are thinking of the relationship as *homoplastic*: they cite the snakes as an independent data-point for the regular coupling of that teeth morphology and venom, and this is swamped by many cases of the same morphology *without* the venom.

Gong et al's response is surprising:

> Apparently… venom was present at the base of the lepidosaur [snakes and lizards] radiation, and might be expected in a sister group… Gianechini et al. (2010) make a fundamental error by assuming that archosaurs with grooved teeth had no venomous taxa in their ancestry and were not venomous themselves... We have no way to conclusively prove that any of the animals with labial tooth grooves were not venomous and, considering recent studies of lepidosaurs, might reasonably expect them to be (109)

This is ambiguous between two readings. Perhaps Gong et al claim that Gianechini and company err by thinking they infer from a *homoplastic* relationship between lepidosaurs and *Sinornithosaurus,* when they propose a *homologous* relationship instead. The common ancestor of the two clades was venomous. Another reading claims there is good reason to see poison as a particularly 'evolvable' trait in that clade. It is 'easy' for snakes, lizards and dinosaurs to evolve venom (see Brown 2013 & Sterelny blah for discussions of 'evolvability'). This suggests the trait is *parallel.* I will concentrate on the first reading and return to the latter in section IV. Taking the inference as made on the basis of homology changes Gong et al's claim dramatically. First, it is not restricted to *Sinornithosaurus*, but many of its sisters: venom should be common in these dinosaurs. Second, reference to lepidosaurs is not as an independent homoplastic data-point but as a homologous ancestral relationship. *Sinornithosaurus inherited* its venom from ancestors common with snakes. A long bow indeed!

Whatever we think of their response, it is clear that understanding it requires contrasting homoplasy and homology along taxic lines – to make sense of the dispute we need the distinction.

And so, when inferring the status of traits using the comparative method, ascertaining the homologous or homoplastic status of target and model is essential. If the relationship is homologous, the inference relies on the shared ancestry of the trait. If homoplastic, it relies upon selection's ability to shape the trait in regular ways. These inferential structures depend on retaining the kind of distinction we see in the taxic account. If venom is homologous in *Sinornithosaurus* and opisthoglypous snakes, then the reconstruction is based upon common ancestry; if it is homoplastic, then it relies on common selective environments. Blurring the line between homology and homoplasy blurs the line between these two kinds of inferences. As we shall see in section IV, some homoplastic inferences in part rely on underlying homologous developmental resources, but this should not lead us to claim they are homologies.

Homology & Homoplasy in adaptive hypotheses

Currie (2012a) discusses the evidential role homoplasy plays in adaptive hypotheses. Here I sketch this, and emphasize the role of homology. As we shall see, homologous relationships 'frame' adaptive hypotheses by setting the appropriate descriptive and temporal level of inquiry; while homoplastic relationships count as evidence *for* that data.

Gong et al claim *Sinornithosaurus'* venom and fangs were adaptations. They aided in bird hunting by weakening prey. Their lengthened maxillary fangs reach the bird's flesh through thick feathers. The inferential structure here moves from a morphological trait (Opoisthoglyphous venom) and a function (weakening prey) in one lineage (Opoisthoglyphous snakes), to infer that same function, given that same morphological trait in a different lineage (*Sinornithosaurus*) (Currie 2012a calls this an 'organism to world' analogous inference). This has the same basic structure as I describe above. However, adaptive hypotheses rely on homology and homoplasy in special ways. Note the hypothesis has competitors. The venom could have evolved for some other purpose. For instance, it has been suggested that the venom of

the male platypus has evolved due to male-male competition for mates. The venom could have evolved before the lineage actually began hunting birds. Consider what we make of Gong et al's adaptationist hypothesis if the trait is homologous or homoplastic.

If it is homoplastic, then (by hypothesis) Opoisthoglyphous snakes have *independently evolved* long maxillary teeth and venom. This counts as *evidence* of adaptive function in the dinosaur. That some other lineage evolved venom in order to weaken prey gives us *some reason* to think that this occurred in *Sinornithosaurus*. But what if they are homologous? If so, then the claim that the venom *evolved to weaken prey* depends on the function of the venom in the common ancestor. Imagine that in the common ancestor the poison evolved for a different purpose. Perhaps the venom was a sexually selected trait as has been suggested for platypus. The use of venom in *Sinornithosaurus* would then be an exaptation, rather than an adaptation as Gong et al claim. Hunting birds does not *explain* the evolution of venom, and Sinornithosaurus' bird hunting is not validly inferred from its venom.

Homology is important because claims about the adaptive function of traits depend upon *timing*. If I claim that some trait *x* evolved to solve environmental problem *y*, then a restriction on this claim is that *x* in fact evolved in the presence of *y* – and homology can test for this. If venom and long maxilliary teeth are homologous between Opisthoglyphous snakes and *Sinornithosaurus*, and the common ancestor *did not* live in a 'using venom to weaken prey' niche, then the venom didn't evolve for that purpose. And so in this sense homology 'frames' adaptive hypotheses: attending to homology helps us decide *which questions to ask* in adaptationist contexts.

Gong et al defend their adaptive claims as follows:

> Our speculation involving long fangs in *Sinornithosaurus* having evolved to penetrate a covering of feathers and the possibility that it was largely a predator on feathered taxa is unaffected by their [Gianenchini et al's] comments. Feathers make such a thick keratinous covering that shorter teeth would have been unsuccessful. In a situation in which a deep tearing wound was difficult to achieve, addition of poison may have been favoured (110).

In other words, the *selective environment* of bird-eating favours long fangs and venom. Whether or not this is true, Gong et al have missed an important point here: if indeed venom or long fangs are basal in these lineages, then their having evolved *for that purpose* turns not on the derived environment which *Sinornithosaurus* lived in, but rather the basal environment: what did the common ancestor use them for? Wings did not evolve to swim even though penguins use them for swimming. Even if Gong et al are right that *Sinornithosaur* fangs were exapted for feather-penetration, they cannot establish that the trait *evolved to* penetrate feathers.

Again, the use of the comparative method in testing adaptationist hypotheses turns on capturing the taxic conception of homology and homoplasy. An adaptationist hypothesis relies upon a correlation between

the trait's evolution and the environment it supposedly evolved in response to – and homology, but not homoplasy, tests for this as it can help us identify *when* the trait evolved. An adaptationist hypothesis relies upon the plausibility of the trait in question's evolution in response to the hypothesized environment – and homoplasy, but not homology, can support this.

Homology and novelty

Evolutionary Developmental Biology (evo-devo) mechanistically explains the evolution of *novel traits*. When new traits, such as turtle shells, the digits of tetrapods, and perhaps *Sinornithosaurus* venom evolved, which developmental changes occurred? A standard account of novelty says that some trait is novel just in case it is *not homologous* (West-Eberhard 2003, Hall 2005, Brigandt & Love 2012).

> A novelty (whether structure, function, or behaviour) is a new feature in a group of organisms that is not homologous to a feature in an ancestral taxon (Hall 2005 p549)

If *Sinornithosaurus'* venom is not homologous in basal microraptors, then it is an evolutionary novelty – and thus a target of evo-devo explanation. If it is present, then it is not novel, and so not a target. Unsurprisingly, there are conceptual issues here: how 'novel' must a feature be? Even if ancestral taxon did not have long fangs, they certainly had fangs – is the gradual lengthening of maxillaries worthy of an evo-devo explanation? I am not interested in providing a complete account of novelty here, but (following Hall) suggest that indexing the potential novelty to some level of description is at least a start. Take the scenario where *Sinornithosaurus*' venom is not present in recent ancestors, but (say) similar developmental resources are utilized. In this case, the ancestors are, say, 'proto-venomous' – they exhibit preconditions for venom's evolution. Here, there might not be novelty at the level of developmental pathways (as developmental homologues exist), but novelty at the *phenotypic* level. As stated this is not completely satisfactory (for instance, are *Sinornithosaurus* fangs novel qua 'being long', but not as 'fangs'? Should we divide the levels that finely?), but nonetheless novelties-as-non-homologues is a promising account for Evo-Devo.

Here we do not see a taxic conception of homology and homoplasy, as the relationship holds between ancestor-descendent pairs rather than sister clades, but nonetheless taking novelties as non-homologues requires that we identify homology strictly and phylogenetically. If the feature (*qua* some level of description) is present in the ancestor of the lineage in question, then it is no novelty – if it is not, then it is. Although the taxic account identifies homologues in sister clades, not ancestor-descendent pairs, it still turns on a phylogenetic and discrete account of the concept.

Homology and shared ancestry

To determine ancestral relations, homoplastic noise must be divided from homologous signal. Cladistic phylogenetics infers patterns of ancestry via statistical analysis of patterns of similarity, allowing diagnoses of homology and homoplasy. This is true in a molecular context, as seen in neontology, and in

morphological contexts as utilized by palaeontologists. Roughly speaking, if two traits are homologous they may be used to trace the common ancestry of lineages, if they are homoplastic then they may not. That penguins and terns have wings is good evidence that they are related via a winged ancestor; that terns and bats have wings is not.

Consider cases where cladistic analyses are 'swamped' by homoplasy. In deciding which traits give good signals of common ancestry, we must control for labile traits, or those with high selection pressure, as they are likely to re-evolve. Hall (2007), for instance, discusses Lockwood (1999)'s study of platyrrhine monkeys. The frequent homoplastic evolution of adaptations for climbing undermined the use of those characters in setting phylogeny:

> This is a situation where a predominant behaviour (climbing) is such a strong selective force that homoplasy becomes a dominant source of the shared similarity in data sets based on characters reflecting that behavioural/selective force (Hall 2007, pp 475).

Controlling for swamping illustrates the importance of keeping taxic homology and homoplasy separate in phylogenetic analyses. Again, an account of those concepts must respect that distinction.

A restriction on accounts of homology and homoplasy

A satisfactory account of homology and homoplasy respects a strict divide between homology and homoplasy along taxic lines: if, between two sister clades, the trait in question is present in a common ancestor, then it is homologous; if it is not present in a common ancestor, then it is homoplastic. It must also retain a phylogenetic conception between ancestor-descendent pairs. A satisfactory account of homology and homoplasy is (1) phylogenetic and (2) strict.

The taxic account here is taken as *sufficient*, if not *necessary*, for determination of homoplasy and homology. For instance, taxic homology as stated will not help us determine homology for the purposes of identifying novelties, as this does not concern sister-clades. Note that by 'strict' here I mean *no overlaps* – it might be okay for there to be a *vague* boundary between homology and homoplasy (traits being neither homologous nor homoplastic), but we must avoid *overlaps* – where a trait is *both* homoplastic and homologous.

I have given three cases where the strict taxic division between homology and homoplasy must be retained to make sense of the epistemic use of the concepts, and another case where *something like* that distinction must be retained:

- *Trait Inference*, where the use of homology is justified on grounds of ancestry, and use of homoplasy is justified on grounds of similar selective regimes
- *Adaptationist Explanation*, where homology serves to 'frame' explanatory targets and homoplasy counts as corroborating evidence for hypotheses

- *The Identification of Novelty*, where a trait which lacks a homologue in an ancestor is novel (and thus worthy of evo-devo explanation)
- *Phylogenetic Reconstruction*, where homologies count as data-points for common ancestry, while homoplasy is noise

These are spread widely across biological practice: from molecular genetics, to systematics, evo-devo, evolutionary biology, and so on… any account which doesn't capture these uses of homology and homoplasy should be rejected.

In the next section I turn to a view on homology which falls afoul of this restriction as it takes some kinds of homoplasy as homologous. I will respond to the arguments presented for the view, before adapting my own version.

III The Hierarchical View

The 'hierarchical view' is an attractive take on the nature of homology. I take the main defender of the view to be Hall, but it is also expressed in Gould (2002), Ramsey & Peterson (2012) and Leander (2008). I take the view to have two main tenets: (1) homology and homoplasy can only be defined *qua* some 'level' of biological organization; (2) 'homoplasy' is not a unified category: those which are due to underlying developmental homologies, such as parallelisms and atavisms, are a form of homology, whereas true convergence is not. The view usually holds that so-called 'deep-homologies' – the retention of various 'master-control' regulatory genes (such as *pax6* and *Foxp2*) and their reuse across disparate clades – are importantly different from 'shallower' developmental continuities. This claim typically underlies the split between convergence and parallelisms.

I am a hierarchist insofar as I agree with the first claim, but will demonstrate that the second claim is untenable. I also dispute the clean distinction between 'deep homologies' and other developmental constraints – preferring instead a context-dependent account. I focus on Ramsey & Peterson, as they provide the most general and rigorous version of the view. My argumentative strategy is simple: because the second part of the hierarchical view does not keep homology and homoplasy separate, it ought to be rejected. I also undermine the positive reasons for taking parallelisms as continuous with homology, and convergence separate. There are of course differences between these various accounts (for instance, Hall sees the space between homologies and parallelisms as a continuum, while Ramsey & Peterson do not), but the differences should not matter for the purposes at hand.

The hierarchy view begins with the insight, made forcibly by Hall, that homologous relationships at different levels can be decoupled: "Homology at one level, for example a feature such as a limb, need not correspond to homology at other levels; the developmental processes that produce the limb, or the genetic cascades underlying those processes (Hall 2003 pp 416)." The upshot of this is that, in principle and frequently in practice, judgements of homology and homoplasy are independent:

> …to identify the hierarchical level of homology and homoplasy being specified, we should always speak of 'homologous as limbs, homologous as digits, homologous as developmental process, homologous as a gene network. Etc.,' and ditto for 'homoplastic as…,' (pp425)."

I am convinced and point the reader to Hall's examples. Any claim of homology or homoplasy must be indexed to some level of description – *qua* 'being winged' bats and birds are homoplastic; *qua* 'being quadrupedal' bats and birds are homologous. This is extremely important for the epistemic uses I sketched in II: for the inference from the form and function of snake venom to carry over to *Sinornithosaurus*, it had better be the case that the homoplasies are at the 'right level'. If the homoplasy is physiological – say the use of lengthened maxillary fangs to deliver venom, but not 'ecological' – used for eating birds, then the homoplasy *is not* evidence for the ecological hypothesis (although of course it could have an indirect bearing via supporting physiological reconstruction).

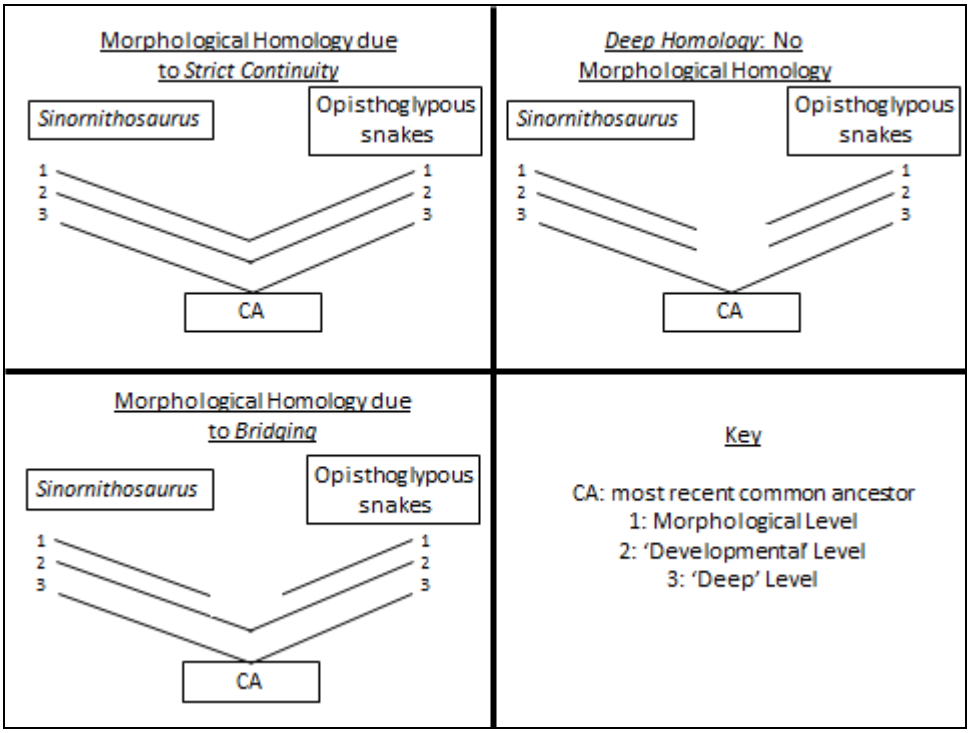
Hierarchists also see homoplasy as disjunctive; some homoplasies are properly understood as homologous, and others are not. This is in part driven by a distinction between 'deep homology' and more directly shared developmental resources. Before responding to arguments for this way of dividing the space, it will be helpful to sketch Ramsey & Peterson's articulation of the view.

For Ramsey & Peterson, two traits are homologous when they meet two criteria. Trace the closest historical line between the two lineages. The common ancestor of *Sinornithosaurus* and Opisthoglyphous snakes lived in the deep past. The line between them shall be temporally long, tracing back to the most recent organism which unites the sister-clades. The line between my father and me will be much more direct: one step from myself to he. Some trait of the organisms is homologous just in case two conditions hold: (1) *continuity*: each step between the organisms and their most recent common ancestor must have the trait *or* be bridged by continuity homology one level lower; (2) *correspondence*: the traits must be *the same* trait (numerically identical) in each organism along the path.

'Correspondence' fits into the analysis based on Ramsey & Paterson's view that homologous relations must be relations of *identity* or sameness, as opposed to a similarity-relation. Remaining agnostic (for the purposes of this paper), I will replace it with a more liberal restriction in section IV. By 'continuity', there are two ways that traits may be homologous. *Strictly*, with no gaps between ancestors, and via *bridging*, where there is a 'gap' between the ancestor's holding the trait, but there are homologues present one level lower. Imagine that the common ancestor of Opisthoglyphous snakes and *Sinornithosaurus* was not venomous, but 'proto-venomous' – the developmental requirements for venom are present, just awaiting the right trigger. In both lineages those triggers are fired and venom evolves. By the taxic account, their venom is not homologous – but by the hierarchy account they are – as the apparent gap is bridged by developmental homologues.

The indexical nature of homology and homoplasy, and the lumping of some homoplasies as homologous falls out of these criteria. Consider the figure below (based on Ramsey & Peterson), which provides three

possible scenarios for the ancestral relationships between *Sinornithosaurus* and Opisthoglypous snakes. For simplicity's sake, the figure only considers three levels of possible homology – the morphological, the 'developmental' (the proximate developmental mechanisms responsible for a trait's morphological expression) and the 'deep' (upstream developmental mechanisms which effect the expression of morphology indirectly).



In the top left we see homology due to 'strict' continuity: long maxillary teeth are present in the most recent common ancestor of *Sinornithosaurus* and Opsithogylpous snakes, and are retained in both lineages. This is a standard homology by the *taxic* account. In the bottom-left quadrant we see a case where the common ancestor did not have long maxillary teeth, but both descendants evolved them utilizing the same resources at the developmental level. By the taxic account, this would be counted as homoplasy – but for the hierarchists, the developmental level 'bridges' the gap in level 1. This is a case of *parallelism* and counts as homologous for a hierarchist. Finally in the top-right quadrant we see a failure of bridging – both lineages evolved the trait after the split from their common ancestor, and utilized similar *deep* resources (regulatory genes), but there is no continuity at the developmental level to bridge the gap. This is a case of *convergence* by their lights.

And so from Ramsey & Peterson's picture we get (1) indexing: homology can only be decided *qua* some level; (2) homoplastic disjunction: 'parallel' evolution is homologous, 'convergence' is not; (3) a split between 'deep' homologies and other developmental resources. It is clear that to deny 2 and 3 in this framework I must deny *bridging* and retain *strict continuity*. Adopting strict continuity would count the lower left and upper right cases as non-homologous, and deny the kind of separation we see between developmental and deep homologies. Ramsey & Paterson's continuity criterion comes from their denial

of an *independence thesis*: that the identification of homology and homoplasy at different levels are independent. I argue that the independence thesis is necessary: first, reasons hierarchists provide for rejecting it are unconvincing; second, it clashes with the requirement that the taxic distinction between homology and homoplasy be respected.

Hall attacks the idea of a unitary homoplastic category directly. It is unsatisfactory, as "… [homoplasies are] neither united by independent evolutionary history, nor by different developmental mechanisms forming the feature in different taxa. Consequently, homoplasy as a category is unsatisfactory, whether one thinks about homoplasy from a developmental or a phylogenetic point of view (2003, 418)." For Hall, then, homoplasies as a category must be united from either a developmental or phylogenetic context. I will criticize both in turn.

His rejection of a developmental basis for uniting convergence and parallelism is rooted in the distinction between deep and shallow developmental homologies. If deep and shallow developmental homologies are saliently different, then this could undermine taking homoplasy unitarily. In standard parallel cases, we see similar characters evolve in two closely related lineages with *the same* (homologous) developmental resources being transformed in mechanistically similar (even identical) ways; while in standard convergent cases, we see similar characters evolve using *divergent* developmental resources. Different homoplasies could have different mechanistic explanations – and this undermines taking them as continuous. This is unsatisfactory for two reasons. First, because phenotypic homologies can be dependent upon divergent (non-homologous) developmental resources there is an analogous case for splitting homologies in two: by Hall's own lights some *homologies* have different mechanistic explanations. Second, this assumes that the parallel/convergent divide ought to depend on the status of developmental homologies, which I deny in the next section.

Hall takes homoplasy as divergent on phylogenetic grounds because parallelisms, like homologues, are due to modification of some pattern of descent, while convergence is truly independent:

> [homology & parallelism] reflect phylogenetic conservation or retention of features in organisms with common descent, independent of whether development has diverged, and… [convergence] reflects similar features resulting from independent evolution (2003, 423, italics removed)

This follows from the idea that parallelisms are not truly *independent*. In parallel cases, development stymies the apparent independence, while in convergent cases it does not. And so:

> … when we are attempting to separate homology from homoplasy mechanistically, we are not dealing with a *dichotomy between homoplasy as parallelism/convergence* and *homology as common descent*… Rather we are dealing with common descent with modification, and, more specifically, with *common descent with varying degrees of modification* (2007, 476).

So Hall sees parallelisms and convergences as divergent phylogenetically because both homology and homoplasy are *explained by* common descent, while convergence is not. I agree that common descent is an important part of the explanation of many homoplasies, but deny that this is sufficient to think of parallelisms as a kind of homology. As I demonstrate, we can retain a strict divide between homoplasy and homology while incorporating the different explanatory resources required for parallelism and convergence. In addition, it is not obvious why this approach, which delineates homology and homoplasy in terms of how they are explained, ought to take precedence over the epistemic roles I have discussed. Moreover, I am suspicious of the idea that *any* convergence on earth is truly independent. For all we know, the basic structure of DNA underlying this generation-event places important constraints on morphological paths. If so, then all homoplasies are parallelisms by Hall's reasoning.

Another reason of Hall's (see 2007) turns on the idea that homoplasy can be evidence of shared ancestry. I previously mentioned 'swamping' cases (such as climbing in plattyhrine monkeys). In such cases we might see the homoplasy as evidence of relatedness. Although the homoplasies do not allow us to work out the patterns of inheritance between the individual lineages, it could be taken as good evidence of shared ancestry in the group overall:

> Studies such as these [Lockwood's] reinforce homoplasy as evidence of shared ancestry, even if that shared ancestry is embedded in the distant past (Hall 2007 475-476).

Hall takes a parallel homoplasy (such as climbing), as good evidence of some group's relatedness, whereas a truly convergent homoplasy is not. And so, for the purpose of identifying closely related groups, the strict homology/homoplasy distinction as captured by the taxic account fails. This is some reason to reject it, and moreover one which speaks to the methodology of this paper.

It is not obvious to me that Hall is right that homoplastic clustering is evidence of shared ancestry, but even if it is I have two responses. First, why is this particular use important, compared to the cases I used to drive my defence of retaining the taxic division? Hall might be right that significant homoplasy signals a group's ancestral clumping, but in order to actually work out the specific relationships between them, we need to divide the homologous from the homoplastic. If we collapse some homoplasies into homology, then we can identify related groups, but we cannot hypothesise specific patterns of descent. It strikes me that the utility of parallelisms here is too weak to drive Hall's claim.

Second, we can read frequent homoplasies as evidence of shared ancestry in virtue of the homoplasies being evidence for *homologies at some lower level*. If many microraptors homoplastically evolved Opoisthoglyphous venom, I would take this as evidence of them being closely related. But why? Because, given those homoplasies, they almost certainly share underlying developmental homologues – and *those* are evidence of shared ancestry. In other words, homologies are evidence of shared ancestry directly, while homoplasies are only evidence derivatively.

What direct reasons are there for rejecting the independence thesis? Ramsey & Peterson provide an argument which, if correct, would require us to abandon independence. The argument points to cases where the *expression* of the phenotype in question is not always actualized, leading to mischaracterizations of non-homologies if independence is retained.

> Consider the unique dominance displays performed by alpha males in a number of species. If we were to take two alpha males in one of these species and ask whether their dominance displays are homologous, operating under a strict rule of continuity, we would likely infer that the behaviors were not homologous. The reason is that there is a good chance that some of the ancestors (including the most recent ancestor) were not alpha males (since although the alpha males often do the bulk of the breeding, they do not do all of it). We thus need to appeal to interlevel dependency in our accounts in order to identify the behaviour of the alpha males as homologous (266)

Because the immediate ancestor does not express dominance, it appears that under a strict continuity rule, we are required to take it as homoplastic. By taking common developmental resources into account, as hierarchists do, we correctly identify dominance as homologous. This should arise for any heritable trait with conditional expression: plenty to justify denying the independence thesis.

Happily, denying independence is not our only route around this problem. I instead embrace a *dispositional* account of characters. We are not merely interested in the phenotype *actually expressed*, but in the *range* of expressions under a variety of conditions. Something like:

Lineage *x* possesses character *y* just in case, under the right environmental/developmental conditions, *x* would express *y*

This is not intended to be a complete *analysis* of when a lineage possesses a character (that would be a difficult task indeed) but rather a sketch of a restriction – whatever story we want to tell about character possession, it had better be dispositional. 'Silver-backed' is a trait expressed by alpha males in gorilla populations, but we need not take this as homoplastic between silver-backed cousins of non-silver-backed ancestors as *under the right conditions, those parents would have expressed 'silver-backed'*. A dispositional account of characters gets around Ramsey & Peterson's concern without denying independence. Moreover, surely a dispositional account of phenotype is independently advantageous. There is an important difference between, say, a male clownfish who has the developmental potential to become female in the right conditions, and a defective male who cannot. These differences are important for explaining their divergent fitness. Likewise, a non-dominant gorilla who, given changes to his harem's hierarchy, would grow silver on his back is different from one who (through some defect) would not – *even if* neither actually becomes dominant. In explaining phenotype expression, we are interested in more than what is actually expressed, but also in what could be expressed – and this leads us to a dispositional view.

There is no good reason for denying the independence thesis. Different homoplasies have different developmental bases, but that is not sufficient to undermine independence. Some (but not all) homoplasies are evidence for shared ancestry – but they cannot tell us about particular ancestors and tell us this in virtue of signalling developmental homologues. We can deal with problem cases like conditional-phenotype-expression by appealing to a dispositional account of characters. Moreover, there are important reasons to retain the thesis.

A worry which emerges from denying independence is the spectre of subjectivism, which creeps into homology and homoplasy identification due to 'level counting'. To know if a trait is homologous or not, I must refer to levels other than the target. I must determine which level is 'one lower' than the target. How can we *determine* the levels which are referred to? Without a non-arbitrary story of determining which levels ought to be represented, the convergence-parallelism distinction becomes arbitrary. In addition, What is to stop me from adding or removing levels? In figure 1, am I *forced* to represent the case as three levelled? If I removed the developmental (2nd) level, then the case in the upper right quadrant would count as parallel as the 'deep' level would bridge. I could add levels as well. By retaining independence, we no longer need to refer to other levels in identifying homologies and homoplasies – and so such subjectivism doesn't emerge from that avenue at least. We might still refer to other levels in *explaining* homology and homoplasy of course. As we shall see, my account will favour a causal/explanatory reading of parallelisms which *proceeds from* rather than being *prior to* judgments of ancestral relations.

Most importantly for this paper, rejecting the independence thesis clashes with the restriction from section II. By that claim, homology and homoplasy by the taxic account must be, at the very least, *sufficient* (if not necessary) for determining the relationship between two traits. This is justified on the grounds that the taxic distinction is required to make sense of many evidential uses in the comparative method. By the taxic account, homology and homoplasy are determined purely phylogenetically. Two traits are homologous just in case the trait is present in the most recent common ancestor. By the hierarchy account, this knowledge is not sufficient to determine the traits' status – as it cannot decide between the upper-left and lower-left quadrants of figure 1. Phylogenetic information alone is insufficient; we must also look at other levels. This collapses homology and some homoplasies together – obscuring the important, and different, roles they play in the comparative method.

IV The Strict Hierarchy View and Parallelisms

So far, I have argued for observing a strict delineation between homology and homoplasy along broadly taxic terms and argued, *pace* the hierarchy view, that we ought to retain the independence thesis. In this section, I outline the view which keeps the indexing of hierarchy, but also independence: call it *strict hierarchy*. By this view, judgements of homology and homoplasy can only be made *qua* some level of description; but judgements of homology and homoplasy at some level are independent of judgments on any other levels. Following that I turn to parallelisms.

Strict Hierarchy

The 'strict hierarchy' view is similar to the hierarchy view, but denies the dependence thesis. It also replaces Ramsey & Paterson's 'correspondence' requirement with something more liberal.

Two similar traits are homologous if and only if, when drawing the shortest path of relatedness between the holders of those traits (i) there is strict continuity; (ii) there is correspondence of salient similarity-relations

'Strict Continuity' is simply Ramsey & Paterson's first requirement without bridging. This version of 'correspondence' differs from theirs in that it does not require a *sameness* relation. I suspect that quibbling about whether homologies need to be 'the same' or 'similar' is beside the point – but not just *any* similarity is sufficient. I opt to leave what counts as a 'salient' similarity relation for later work, but they will be whichever similarities are important for determining homology. Two reasons Ramsey & Paterson provide for taking homology as a relation of identity rather than similarity is first, that some homologues are not particularly similar (swim bladders and lungs, for instance) and second, that homologous relationships are transitive like identity relations, while similarity relations are not necessarily so. Although I require correspondence in terms of 'similarity', the restriction to salience is supposed to answer these worries. It is true that many homologues are not similar, but they are similar *in the relevant respects*. Both lungs and swim bladders, for instance, have a 'transformative' similarity (see Griffiths & Brigandt 2007) and a similarity in ancestry. If homology relationships are logically similar to identity relationships, then the similarities which count will reflect this – perhaps only transitive ones will do. Much more remains to be said on this issue, but I leave that for future work. This view answers some of the issues we have seen with the standard hierarchy view.

First, this gives us the right story about cases one, two and four from section II. In inferring traits, supporting adaptive explanations and phylogenetic reconstruction we require homology and homoplasy be divided along taxic lines. By strict hierarchy, the traits of two sister-clades will be homologous if held by their most recent common ancestor (*qua* some level of description), and homoplastic if not. The taxic divisions necessitated by those inferential and explanatory uses are retained.

Second, we get the right story for the identification of novelties. There, we require a strict delineation between homology and non-homology in ancestor-descendent pairs. By strict hierarchy the evolution of venom in *Sinornithosaurus* would count as a novelty just in case that trait is not present in the *Sinornithosaurus*' immediate ancestor. Allowing for some grey areas in terms of 'immediacy' and 'proto-traits' (presumably poison evolved gradually), this meets the needs of evo-devo.

Third, we needn't concern ourselves with 'level-counting' in determining homology and homoplasy. Although traits are homologous or homoplastic *qua* some level of description – and we might disagree about appropriate explanatory targets – once we have a target in focus, we need only concern ourselves with phylogenetic relationships to determine homology and homoplasy.

There is still a major challenge facing this account: serial homologues. I take it my left and right hands are homologues, but there is no phylogenetic relationship between them I could appeal to, to make them so by this account. One option, in the spirit of Griffiths' pluralism, would be to agree that in terms of phenomena, within-organism (serial) and between-organism homologies are continuous; both are part of the same 'investigative kind'. But we *explain* them with separate resources; some other account of homology is required for that task. Another route, as Ramsey & Peterson attempt, is to build on the current account to incorporate serial homologues. I leave a full exploration of these options for later work.

Parallelisms

The main difference between a strict hierarchist and views like Hall's is the classification of parallelisms. For the standard hierarchist, parallelisms are homologous as they are *bridged*: homologous resources one level below are responsible for the apparent homoplasy. The strict hierarchist does not distinguish between parallelisms and convergences as so far stated: all are homoplastic, neither is homologous. However, the difference between parallelism and convergence is important. For instance, cases of parallel evolution can provide a stronger basis for postulating robust regularities across closely related clades (Currie 2012a, Gould 2002). For one thing, closely related lineages are more likely to share developmental resources, and are thus more likely to respond similarly to selective pressures. Hypotheses about the evolutionary effect of environment on evolution are less likely to be stymied by developmental noise. The thought is that lineages which share developmental constraints are more likely to respond similarly to environmental pressures, making adaptive generalizations easier. A final task for this paper, then, is to distinguish parallelisms from convergences.

Keeping with the methodology so far, we should ask: why do we *care* about the distinction between parallel and convergent homoplasy? What epistemological work does it do? The usual answer is the perennial question of externalism vs internalism in shaping evolutionary form (Godfrey-Smith 1996): Gould's concern about the role or otherwise of natural selection in macro-evolution (Gould 1997). For the externalist, environmental factors play a central role in constraining the pathways open to evolution; the internalist emphasizes internal constraints, such as development. Some externalists cite wide-spread convergence as evidence for their thesis (Conway-Morris 2003, McGhee 2011). But this turns on the convergences' independence from internal constraint: if homoplasies are explained by restrictions due to the developmental resources evolution has to work with, then homoplasy is no evidence for externalists.

A more prosaic reason for caring about parallelisms versus convergences is described in Currie 2012a. Cases of convergence are cited to support specific adaptive hypotheses (as opposed to the role of natural selection *generally*). Gong et al's speculation that *Sinornithosaurus'* long fangs are an adaptation for biting through feathers could be supported by pointing to a homoplasy. Perhaps some other lineage uses its fangs similarly. How convincing is this? That depends. If the relationship is parallel, we should expect the

two lineages to be more similar than if the relationship is convergent. *Prima facie*, the more (relevantly) similar the lineages, the more likely it is that differences or similarities between them will be due to selective regimes. Citing a parallelism (as opposed to a convergence) to defend an adaptationist model controls for developmental noise by lessening the scope of the inference. Given the ubiquity of appeals to convergence in evolutionary biology, this gives good reason to care about the distinction. Note that I am *not* claiming here that parallelism is better evidence than convergence. Indeed, a convergent trait between very distantly related lineages is remarkable and could be very strong evidence for adaptation. The point is that inferring *between* parallelisms is *more secure* (or at least more tractable) than between convergences. This is because the phylogenetic and developmental similarity between parallelisms controls for noise from these sources[2].

So, we want an account of parallelisms and convergence which brings out the important aspect of the distinction: a *convergent* homoplasy is largely constrained by external factors, factors like the environment do the explanatory work; a *parallel* homoplasy is largely constrained by internal factors, developmental constraints, or phenotypic limitations, do the explanatory work.

As we have seen, hierarchists define parallel homoplasies in terms of development: if there is a *bridge* on the developmental level uniting two homoplastic traits, then it is parallel. There are other accounts which explicitly link the role of development to defining parallelisms. Powell (2007) for instance, draws the distinction between parallel and convergent in terms of putative parallel developmental resources being *screened off* (in Salmon 1984's sense) by upstream processes. Pearce (2012), Wake et al (2011) and Gould (2002) also provide development-based accounts. Powell (2012) and Currie (2012a) both suggest that parallelisms and convergences can be divided by whether the underlying developmental pathway is a difference-maker.

In the account provided here, I follow up on the 'difference maker' concept and do *not* tie the concept to development.

Why not just development?

[2] Part of the justification of this turns on an empirical claim about the relative similarity, and level of constraint, between convergences and parallelisms. Following Gould and Griffiths, I think that functional continuities are relatively coarse, while ancestral continuities are finer grained - convergent similarities are 'shallow', while parallel similarities are 'deep'. If that is right, then because developmental continuities tightly constrain the space of possible phenotype expression, I should be quite confident in inferring between model and target. Moreover, shallow similarities probably belie less constraint in phenotype expression, meaning that projectability from model to target is less secure (see Currie 2012b for discussion of the 'shallowness problem'). This claim, then, is merely *ceteris paribus* and based on the empirical claim that *most of the time,* functional similarities are shallower than ancestral ones. Thanks to an anonymous referee for pushing me on this point.

As outlined above, most accounts of parallelisms are explicitly tied to the idea of development. I certainly don't want an account which *excludes* homoplasies being due to developmental factors, but think there is good reason to want something broader.

First, the parallelism-convergence distinction is interested in *constraint* – and I suspect constraint can come from places other than development. Phenotype, it seems to me, can constrain evolution as surely as development. Why, for instance, are there comparatively few large mammals dwelling on islands compared to birds? Surely this is due to the comparative difficulty large mammals face when trying to *reach* islands, and this is due to their phenotype. This doesn't look like selection: there is no selection pressure on animals to reach islands. As a developmental constraint lowers the likelihood of some new mutation arising in a population, a 'phenotype constraint' lowers the likelihood of a population reaching some environment (to be shaped by whatever environmental pressures are there)[3]. The island example is a legitimate case of constraint based on phenotype. It is schematic, and making this point fully would require much more detail, but I take it to provide proof of possibility and at least motivate a non-developmental take (see Pearce 2011b for further discussion of non-developmental constraints)[4].

Second, in some scientific contexts we lack epistemic access to developmental information. Palaeobiologists, in particular, could make good use of a parallel-convergent distinction, but have only the most indirect ways of knowing the developmental homologues between their targets. If the relation between *Sinornithosaurus* and rear-fanged snakes is indeed homoplastic, how are we to determine whether this is due to underlying developmental constraints? Scotland (2011) criticizes Gouldian accounts of the distinction on a similar basis: we rarely have access to the developmental information required – and moreover phenotype cannot act as a proxy (because development and phenotype are frequently decoupled). An account which accommodates taking parallelisms as due to *either* development *or* phenotype (or whatever else fits the bill) gets around this problem as we are free to hypothesize about internal constraint without determining developmental relationships. We should prefer an account which is more easily usable by palaeobiologists and still meets the needs of neontologists. The difference making approach I suggest does *not*, of course, make things easy for palaeontologists. But it does make things somewhat *easier*: developmental information is *included* in the set of possible constraints, but other routes

[3] But surely a lineage's phenotype is as it is in part *due to* its genotype? Why don't phenotypic constraints collapse into developmental constraints? Here's why: in cases of developmental constraint, the developmental system's inability to achieve certain forms constrains morphospace. In phenotypic constraint, it is the phenotypic failings to (for instance) fly to islands which explain why certain evolutionary paths are closed. Naturally part of the proximate explanation of why any particular phenotype is as it is appeals to developmental systems, but 'being flightless' is multiple realizable across such systems. In cases of phenotypic constraint, it is phenotype, not development, which *explains* the contrast (thanks to Kim Sterelny and an anonymous referee for pressing this point)

[4] A referee points out that structural constraints (for instance, those which appeal to geometry) could, if they are classed as internal, count as parallelisms without any developmental at all. How we precisely delineate internal and external constraints, and what to say about structural constraints, is left for further investigation.

of investigation are also open. Being free to call upon a wider set of concerns in establishing parallelism makes the concepts more amenable to use in palaeobiological contexts.

Third, and most speculatively, we might want to extend concepts of parallelism outside of usual biological contexts. Most obviously, cultural evolution might make use of the parallelism/convergence distinction – and tying the account to development restricts such extensions. The nature of the hierarchical account conflicts with tying parallelisms merely to one level-relations and surely there are potentially interesting homoplasies at each putative level.

Why difference making?

I approach homoplasy as an explanatory and inferential concept: it explains biological similarity and supports biological hypotheses. On one reading of Gong et al, the lengthened maxillaries between *Sinornithosaurus* and rear-fanged snakes are homoplastic: they independently evolved similar traits. But what explains their convergence? Presumably some combination of internal and external constraints (c.f McGhee 2011, Griffiths 1994, Sansom 2003). It could be that something about *Sinornithosaur* and snake physiology, morphology or development makes the evolution of long fangs likely: the homologous intrinsic properties of the lineages constrain occupiable morphospace. It could be instead that common environmental pressures carry the majority of the burden: snakes and *Sinornithosaurus* both underwent strong enough selection to force them into similar parts of morphospace. Parallelisms and convergences pick out this continuum. The parallel or convergent nature of the homoplasy makes a difference to which inferences are supported. If the two lineages are too different in terms of internal constraints, then this can stymie the effect of common selective pressures. If the similarity is largely due to tight internal constraints, the inferential basis will *ceteris paribus* be stronger, although the scope of the inference will be restricted to lineages with those internal constraints.

Difference-making accounts of explanation and causation lean on the thought that some counterfactual dependencies are *special*: they make a *difference* to the occurrence of the phenomena we are interested in. Roughly, some variable x is a difference maker to some output y just in case, if we were to intervene on x, while holding all other variables fixed (other than those downstream of x), this would change y (see Woodward 2003). Say that Gong et al are correct in their speculation that *Sinornithosaurus* preyed on early birds. Their claim that lengthened maxillaries evolved in order to pierce through feathers and deliver a poisonous bite can be read as a counterfactual claim. If *Sinornithosaurus* did not prey on birds, then they would not have lengthened maxillaries. What makes this interesting is the idea that selection for a bird-eating niche is a *difference maker* for that morphological trait. If we hold *Sinornithosaurus* history fixed, but tweak bird hunting, would they have shorter maxillaries? If their teeth would still be long, then Gong et al's speculation is false. If they would have had shorter teeth, then we are onto something.

Difference-making has the potential to pick out the right distinction between internal and external factors we want for the parallel-convergent distinction. After all, we want something to be parallel just in case

internal factors make a difference, and convergent when external factors make a difference. However, difference making on its own is far too broad a category: even if some factor plays a minor role, it can still count as a difference maker. Perhaps if *Sinornithosaurus* did not inhabit a bird-hunting niche their fangs would be a tiny bit shorter – then bird hunting makes a difference to fang-length, but far too insignificantly for us to declare this a convergence rather than a parallelism. I think Woodward's (2010) 'causal specificity', which draws on Waters' 'actual difference makers' (2007), and Lewis (2000)'s notion of 'influence' is a good candidate for dividing the two. My story is similar to Powell's 2012 (he draws on Waters) – but differs as I discuss below.

For any particular homoplasy, some combination of internal factors and external factors will explain the similarity (there should be a third variable: chancy, coincidental similarity, but for simplicities sake I leave this out). Lewis' notion of *influence*, which Woodward develops, is the idea that some variable (call it v_1) effects another (call it v_2) *systematically*. In Woodward's terms, there is some mapping function (*F)* which connects interventions on v_1 with one-one changes in v_2. Bird-hunting would influence fang-length in *Sinornithosaurus* if, were we to make changes to *Sinornithosaurus* hunting behaviour (reduce or increase their dependency on bird-hunting), the average length of fang would shift a specific amount for each change in bird hunting. *Specificity*, then, controls for redundancy: if a wide number of shifts in one variable make no (or little) difference to the outcome, then it is not specific. I will sketch how we might use specificity to determine the parallel or convergent nature of a parallelism.

Any particular homoplasy will be due to some measure of *internal* and *external* variables held in common between the lineages the homoplasies occur in. For some homoplasy *H,* take the set of internal variables held in common $\{i_1, i_2, i_3 \ldots i_n\}$ and the set of external variables held in common $\{e_1, e_2, e_3 \ldots e_n\}$ and determine the specificity for each variable. This will tell us how much redundancy exists for the internal set and the external set. If the specificity of set $\{i_1, i_2, i_3 \ldots i_n\}$ is *higher* than set $\{e_1, e_2, e_3 \ldots e_n\}$, then the *H* is 'parallel', if *lower* then it is a convergence, if they are equal, then it is indeterminate.

Let's run the account through a toy test case (also used in Powell 2012). The evolution of lens-eyes in mammals and cephalopods is a striking homoplasy between two distantly related lineages. Infamously, both lineages utilize homologous homeobox genes (*pax6*) in eye development. Is the development homologue sufficiently constraining for the phenotypic homoplasy to count as parallel? *Pax6* controls for eye tokens rather than types: the insertion of a human *pax6* into a cephalopod developmental system, if it had any effect at all, would result in extra *cephalopod* eyes, rather than human ones. Moreover, the gene is utilized in non-lens eyes such as those of insects. This ought lead us to conclude that *Pax6* does not constrain eye evolution sufficiently to count as parallel. And this is the result which my account provides. *Pax6* is most likely a difference maker in the evolution of eyes overall (it certainly is in the development of eyes), but it lacks specificity. In development at least, *Pax6* is extremely redundant: changes in states to the gene operate like an on-off switch. The redundancy of *Pax6* (assuming that the other internal

variables are similar) would suffice for the phenotypic homoplasy between mammal and cephalopod eyes to come out convergent rather than parallel.

Here are a few nice features of this account. First, it allows a smooth gradient between parallel and convergence – as stated, it is (near) binary but in practice I take it that a parallelism will be one where the specificity of the internal set *significantly outweighs* the specificity of the external set. Second, it doesn't require that the internal set be developmental only – it allows for cases of phenotypic restriction, and could easily be utilized in cases of cultural evolution. Third, it gives us the answers we want. 'Master-control' genes (such as *Pax6*) don't seem to be constraining *enough* to provide parallelisms – the mere utilization of *Pax6* doesn't give us any reason to think that a particular eye will develop, as it is used for a wide variety. But this is just to say that, *qua* eye-type, *Pax6* is extremely redundant.

Arednt & Reznik (2007) and Pearce (2012) think there is a general difficulty with tying accounts of convergence and parallelism to the sorts of philosophical analyses usually reserved for causation. Most model causation as *linear* – and developmental systems just don't work like that. Powell's account, for instance, relies on a distinction between 'distal' and 'proximate' developmental causes to distinguish convergences and parallelisms. This is stymied by the sheer complexity of developmental systems. In response, we might admit that scientific representations of such systems are inevitably idealized in some ways, and so might frequently be represented as linear systems. However, in principle at least, reliance on a difference-making account does not rely on such 'proximate' or 'distal' causes: all that matters is the *causal specificity* – and this does not require linear systems. It doesn't matter *how* tortured the causal route between the variables, if it can approach some functional mapping between states of the variable and the outcome, then it will be causally specific. Moreover, as my account relies on the *aggregate* specificity of internal and external factors, as a heuristic in distinguishing parallelisms from convergences we could black box such complexities.

Another objection, which Pearce (2012) points at Powell's account, is from *operationalization*. Roughly, can we expect scientists to perform the interventions which appear to be required to determine (in my case) the specificity of such internal factors? As Pearce points out, in some cases (particularly in terms of developmental systems which include feedback loops) it isn't obvious whether 'holding fixed' 'upstream' variables is possible (as they are also downstream). I hope it is obvious that I don't (at all!) expect palaeobiologists to be able to perform anything approaching an intervention to determine the relationship between bird hunting and tooth length. But this is a standard problem for palaeobiologists, and one which I think they share with neontologists and other scientists. Sometimes we simply do not have experimental access to our targets. Happily, the use of the comparative method, modelling and simulations, and other more 'indirect' routes can grant epistemic access to such counterfactuals. Gong et al's appeal to snake phenotype (when read homoplastically) is precisely such a case. They are unable to determine *directly* whether long fangs are necessary for venom (as they cannot manipulate their extinct target), but reference to other venomous lineages gives reason to think that, if we *were* able to, such a link

would be established. Even if proposals such as my own and Powell's cannot be operationalized in this sense, they can be inferred nonetheless. This complaint can be pushed further: just how can we *know* that, say, the specificity of the complete set of internal constraints affecting eye evolution across cephalopods and mammals is less than the specificity of the external constraints? A full response to this complaint would involve too detailed a discussion of biology's epistemic resources to be made here, but I repeat myself: such knowledge can be slowly patched together indirectly.

Powell draws on Water's distinction (similar to Woodward's) of 'specific' versus 'general' cause. His 2012 builds that into his account – "… a homoplasy is a parallelism *iff* some of its proximate developmental machinery is both *homologous* and *causally specific* (369)." This account faces challenges mine avoids. First, it is tied to development. Second, it relies on our determining what 'proximate' might mean. Third, why should we think the proximate cause is the most constraining (c.f Pearce 2012)? In distinguishing parallel from convergent homoplasy, we target internal versus external constraints. Is there a good reason to think that a cause's being proximate is more likely to make it more constraining? My hunch is that more often than not this is the case – but certainly not reliably, particularly in non-linear systems. However, Powell's approach and mine are clearly close kin. As Powell says, "The key to distinguishing parallelism from convergence is that it is not the *extent* of developmental homology involved, but rather the *causal type*, that counts (369)." I mostly agree – but deny that *developmental* homology is necessary, and don't restrict 'causal types' to proximate causes.

And so, a difference-making account has the potential to deliver the goods. Causally specific factors will be constraining in the desired fashion, picking out internal versus external constraint and, in principle at least, it can deal with non-linear systems.

Conclusion

I have introduced a novel set of concerns for delineating homology and homoplasy. Examining biological practice, we see that a strict, phylogenetic delineation between homology and homoplasy is required to make sense of appeals to comparative evidence in biology. I also provided a novel conception of homology and homoplasy which retains the indexed nature characteristic of the hierarchy approach, but takes the concepts as independent – we need not appeal to other levels when delineating homology from homoplasy. Phylogenetic information suffices. I finally offered an account of parallelisms and convergences which respect their biological use. By defining them in terms of their causal role, we both capture the internal vs external requirements of that use, and allow the distinction to be made without appeal to developmental resources.

There is still work to be done. Strict Hierarchy does not give us a story about serial homology, and how the account interacts with the concepts' histories, and approaches which focus on homology as explanatory target have not been explored here.

If we are to understand applications of the comparative method exemplified in work such as Gong et al's discussion of *Sinornithosaur* venom, we had better ensure that central concepts such as homology and homoplasy are illuminative of such uses.

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