

Contingency's Causality and Structural Diversity

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Abstract

What is the relationship between evolutionary contingency and diversity? The evolutionary contingency thesis emphasizes dependency relations and chance as the hallmarks of evolution. While contingency can be destructive of, for example, the fragile and complex dynamics in an ecosystem, I will mainly focus on the productive or causal aspect of contingency for a particular sort of diversity. There are many sorts of diversities: Gould is most famous for his diversity-to-decimation model, which includes disparate body plans distinguishing different phyla. However, structural diversity construed more broadly spans scales, such as organization in and among cells, structural arrangements and biomechanics on various scales, and even the profile of ancestor-descendent relationships or community structure of interactions within ecosystems. By focusing on stochastic processes in contingent evolution, I argue that contingency causes structural diversity. Specifically, I focus on the plurality of structural types of cells, genetic codes, and phyla diversity as case studies.

“...[W]e would like to have some reason for believing that...evolution had actually resulted in only one outcome: one mechanism of inheritance in sexual organisms, one mechanism of development on eukaryotes, one foraging strategy in herbivores. But why would we expect evolutionary outcomes to be so constrained?”

--John Beatty (1994, 52) *Theoretical Pluralism in Biology*

1. Introduction

John Beatty once argued that we should not expect evolutionary outcomes to be highly constrained by selection or some singular mechanism (1994, 52). While Beatty's work suggests a pluralism concerning evolutionary products and a view of evolution as unconstrained, Hull (1987, 178) worried that pluralism could be destructive of science. That tension motivates my thesis in this paper: I argue that contingency has a generative role in the production of diverse outcomes. In other words, one should not assume that the products of evolution, such as the genetic code, cell types, modes of inheritance, or even individuals in selection, are constrained in such a way that they often resist change and limit new variants. One important upshot is that contingency, by causing diversity of a certain sort, results in a pluralism. Specifically, contingency causes structural variations, which distinguish a plurality of types. Many philosophers have adopted pluralism about biological categories, such as species, individuals, and so forth. I'll argue that such pluralism is to be expected because evolutionary contingency generates it. Reasons to expect pluralism from an evolutionary contingency framework is a strong undercurrent and systemic motivation throughout this paper. Admittedly though, to say that contingency *causes* diversity sounds strange at first pass, yet it is an indispensable move in the argument from contingency to pluralism. And so, this paper mainly consists in working through the diversity I have in mind and how contingency causes it.

On the one hand, Beatty (1995, 47) proposed the evolutionary contingency thesis (ECT) to include generalizations that are distinctly biological and as such they describe contingent outcomes of evolution. However, here I do not discuss laws and generalizations in biology directly, rather I understand ECT more broadly. ECT now also includes topics from contemporary literature that tackle a variety of concerns, such as how the pattern of contingent evolution unfolds (Beatty 2006, Turner 2011, Desjardins 2011), the sources or causes of that contingent profile (McConwell and Currie 2017), and evidence for or against contingency as the hallmark of evolution (Powell and Mariscal 2015). Generally, the profile of contingent evolution consists of a set of events whose trajectory depends on prior events, sometimes their order or path, and unbiased processes, such as mutation, drift, species sorting, and external disturbances. Here I aim to understand the contingency thesis's explanatory reach. I anticipate a dilemma concerning contingency as causal, which is given careful analysis in section 3. As we'll see, the dilemma sets up the process-based approach to evolutionary contingency explored in the present paper. The process-based approach to contingency I adopt is in a similar vein as mine and Adrian Currie's (2017) previous work, which contrasts with the traditional approaches primarily working with modal properties. I discuss this in Section 2.

On the other hand, concepts of diversity have a complex history that cross branches of biology, such as evolutionary theory and ecology. One main message of Maclaurin and Sterelny's work is that a single measure of diversity in a biological system is untenable (2008, 7). There is historical attention concerning diversity as a central theme of ecology, the varieties of measurement, and different sorts of diversities (Magurran 1988), as well as a recent focus on explaining patterns of species abundance and diversity (Bausman, 2016). From this, any

connection between diversity and contingency, or why it matters, is not entirely obvious.

Though perhaps we can return to Gould.

Gould argued for a diversity-to-decimation model, which includes disparate body plans distinguishing different phyla. Diversity qua disparity is a technical notion referring to differences among anatomical designs (Wills et al. 1994, Foote 1993, Maclaurin and Sterelny 2008). Although Gould outlines a decimation model where *Baupläne* disparity decreases through time, as we'll see, it's not obvious that Gould's model necessarily discounts episodes or explosions of diversity even after periods of decline. This is the case when diversity, specifically structural diversity, is treated more broadly than anatomical design. Gould's treatment of contingency as a destroyer of diversity, is only one part of the story. Instead, I ask, is there a positive or productive connection between evolutionary contingency and diversity? Indeed, as we'll see, there are grounds to consider that connection as causally productive.

The paper is organized as follows: first I introduce the contingency literature starting with Gould and followed by Beatty's 2006 distinction between two senses of contingency. I use their work as a foundation to start thinking of contingency as causal by working through a dilemma in Section 3. The dilemma is explored in detail because it motivates the process-based approach to evolutionary contingency I adopt in this paper. That approach emphasizes processes that cause contingent evolution rather than evolution's modal character.¹ Thereafter, while noting various types of diversity in Section 4, I analyze case studies for evidence of contingent

¹ Another way to think about process-based approaches versus primarily modal approaches to evolutionary contingency is this: 'thin' accounts of contingency appeal primarily to modal properties of evolutionary trajectories, whereas 'thicker' accounts include more specific causal details about the processes responsible for those modal properties.

evolution and structural diversity. I finalize in Section 5 by reconciling Gould's decimation (or the "contingency as destructive") model with the present thesis that evolutionary contingency produces (rather than only destroys) diversity.

2. Contingency

Understanding the extent of contingency in evolution and the nature of evolutionary history is a significant research programme. Stephen Jay Gould famously argued for the prevalence of chance and dependency relations in life's history. He used contingency to challenge evolutionary inevitability and progressiveness, in addition to panselctionism—the view that natural selection, especially at microscales, could capture patterns found over large spans of time. While in this section I focus on Beatty's two senses of contingency found in Gould's work, I'll first start with a brief introduction to the topic more generally.

It is well-known that the Burgess Shale quarry contains remarkable soft-part preservation in some of the earliest known fossils from the Cambrian period (570 mya). Gould counted over 20 Baupläne designs (i.e. body plans) or assemblages of morphological features establishing different phyla, of which only 4 survived (1989, 106). "The rare soft-bodied faunas of the fossil record are precious windows into the true range and diversity of ancient life" (1989, 24).² However, Gould did not think that life unfolds in predictable pathways of progress and ever-increasing anatomical disparity (276). He challenges his reader to imagine replaying life's tape: if survivors of the Burgess Shale are superior in their adaptive features, then they should win on

² There has been controversy over reclassification of Burgess Shale fauna (Brysse 2008). Changes in taxonomic methodology and the adoption of stem group concepts concerning Burgess creatures' relationships with modern organisms has challenged the range of Cambrian diversity. Brysse understands this shift as a methodological artifact not necessarily forged by new data.

every replay. But if those survivors are “protégés of Lady Luck or fortunate beneficiaries of odd historical contingencies, then each replay of the tape would yield a different set of survivors and a radically different history” (1989, 50). On this picture, evolutionary outcomes have mostly unique qualities that vary across replays.

More recently, Gould’s framework has been further developed (Beatty 2006, Desjardins 2011, Turner 2011). Beatty distinguishes two senses of contingency used by Gould: contingent *upon* and contingent *per se*. The former is historical and refers to dependency relations among events, such as sensitivity to initial conditions and path-dependence, which includes prior pathways that constrain future evolution (Desjardins 2011, 737). The latter, contingency *per se*, is forward-looking—future evolutionary events are unpredictable due to the causal insufficiency of any prior state (Beatty 2006, 339, Turner 2011, Gould 1989, 278).

Adrian Currie and I (2017) have argued that these accounts understand evolutionary history modally. The modal pattern is an increasing trajectory of necessary yet insufficient conditions: a prior particular state might be needed (or as John Beatty puts it ‘strongly necessary’) to bring about a particular outcome, but alone is not enough: an event’s occurrence provides no guarantee for what happens next. Evolution lacks the sort of necessity that would guarantee a bridge to make future predictions. This modal pattern of dependence and insufficiency is analytically described in a way that effectively black-boxes the causal processes of evolution’s profile. That is, traditional accounts of contingency largely ignore or de-emphasize (i.e. “black-box”) the sorts of processes that cause contingent evolution, which is unsatisfactory. Against the mainstream, we emphasize the role of contingency in terms of processes. Adrian and I look at different sources of evolution—the processes causally responsible for life’s pattern. The

process-based approach is useful—we appealed to empirical differences in the sources of contingent evolutionary patterns to clarify the debates. Along a similar vein, I draw from the role of stochastic processes in contingent evolution, however, I emphasize different contexts where the process-based approach is required. In the next section, I directly reflect on the two different approaches to evolutionary contingency (modal vs. process-based) and the potential problems they raise for contingency's causality in a dilemma. Then later in section 4, by adopting a process-based approach, I discuss how contingency causes diversity.

3. Thinking of Contingency as Causal: A Dilemma to Contend With

The thesis that contingency causes diversity likely raises an immediate worry. Prima facie, to think of contingency as causal is strange because of its modal baggage. In this section I unpack two horns of a dilemma for thinking of contingency as causal. Insofar as there are at least two approaches to thinking about evolutionary contingency, a problem arises in each case:

- (1) The modal approach: If contingency is a modal property, then to think of contingency as causal is misinformed or misguided because modal properties don't cause things, much less diversity. *Contingent upon* and *Contingent per se*, as outlined by Beatty, are the modal senses of contingency analyzed here. A process-based approach avoids such strangeness by addressing the sources (i.e. the processes) of contingency.
- (2) The process-based approach: However, if one considers contingency as the stochastic processes that cause contingent evolution, then stochastic processes are doing all of the causal work. The threat is for contingency to become extraneous: Stochastic processes cause diversity, so why is evolutionary contingency needed at all?

My strategy is to address each horn in turn by not only unpacking the approaches in (1) and (2), but also navigating through contingency's causality in each case.³ In the end, I adopt and defend a process-based approach to contingency in order to argue that it causes diversity in section 4.

3.1 The Dilemma's First Horn: The Modal Approach

Contingency is traditionally a modal property considered in contrast to necessity, and sometimes situated in historical contexts (Ben-Menahem 1997). Modality can be understood in possible world frameworks (re: Lewis), along with other analytic work (Cresswell 2016). Generally, 'modality' refers to non-actual aspects of statements: modal properties situate their bearers within possibility. *Evolutionary* contingency, then, is a property of events arranged in a certain pattern or relationship to one another such that their likelihood is counterfactually understood. One aim in recent evolutionary contingency literature is to establish the prevalence of contingent outcomes compared to those that are necessitated or determined. In the latter case, evolutionary trajectories converge regardless of history (Gould 1989, Conway Morris 2003, Powell and Mariscal 2015). In other words, the aim is to discover how much history matters in evolution: do outcomes typically converge across different phylogenies, e.g. eyes for sight in humans and squids? Or does history make a difference most of the time? Under a modal framework, one might argue that diversity is itself an outcome that is either contingent (i.e. fragile and dependent on history) or robust (i.e. frequently occurring in certain environmental conditions).

³ The line between modal versus process-based approaches may not be as clear cut as presented here, though it serves as a heuristic to navigate the contingency literature landscape.

The worry is this: contingency is a modal property and modal properties do not cause things, therefore, to say that contingency is causal is misinformed, or even foolhardy. The plot thickens further as a modal approach includes the two senses of contingency identified by Beatty.⁴

First, the causal dependence sense of contingency is the backward-looking historical sense, which includes outcome sensitivity to both initial conditions and prior ordering of states, events, and processes (Beatty 2006, Desjardins 2011). So, the existence of today's giraffes (*Giraffa camelopardalis*) is likely contingent upon the existence of their ancestor *Okapia johnstoni*. That is, *O. johnstoni* was potentially necessary⁵, but alone insufficient for *G. camelopardalis* because many other factors (stochastic and otherwise) occurred in between.⁶ When a particular outcome depends upon a prior state(s) that precedes it, that outcome is contingent upon the prior state(s) (Beatty 2006, 339). That is, the relationship between states is one of causal dependence. If 'contingency' is defined as causal dependence, then to say that contingency causes diversity amounts to the following:

causal dependence causes diversity

To say that diversity is explained by causal dependence seems uninformative or even trivial:

That there are many kinds of wasps, for example, depends upon prior states of affairs, such as

⁴ I am very much indebted to Derek Turner and Joyce Havstad for articulating the dilemma to follow through in order to consider contingency as causal.

⁵ *O. johnstoni* as a necessary condition for *G. camelopardalis* does not mean "if *O. johnstoni* occurs, then *G. camelopardalis* necessarily occurs." In other words, to recognize the dependence of *G. camelopardalis* on *O. johnstoni* is to acknowledge prior needed (yet individually insufficient) conditions of a particular path leading to a particular outcome. Identifying necessary and sufficient conditions can be used to make sense of the historical dependency relation between species, but Desjardins has showed how such path dependent relationships can come in degrees of sensitivity too.

environments hospitable to wasps. However, the trouble is not necessarily triviality, if indeed one lesson from contingency-as-causal-dependence is that history constrains future evolution. Recall Gould's explanation for what makes life so wonderful: even under a million replays, it's not obvious that anything like human existence, or any other outcome of interest, would ever evolve again. This is due to the various possible starting conditions and dependencies along the way that guide and constrain pathways. On this picture, evolutionary outcomes are mostly unique across different possible replays. As Currie (forthcoming) states, "history bends possibility: once inviting routes are closed off, once remote paths become neighbourly" (203). "Historical contingency generates uniqueness" in the sense that "historically contingent end states require highly specific initial conditions and sequences of events to occur, and so shall be rare if those conditions or sequences are" (213-14).⁷ Such rarity or uniqueness is revealed upon *thinking counterfactually* where outcomes are sensitive to a complicated, and sometimes unlikely, trajectory of prior events. Therefore, replays yield a diverse set of outcomes in the sense of uniqueness across possibility space where such rarity is discovered counterfactually.

I'll stipulate up front that contingency-as-casual-dependence cannot do the causal work needed in this paper. I aim to show that contingency produces a variability that can be typed and categorized both synchronically and diachronically: actual differences in structures (i.e. arrangements, relations, or mechanisms as we'll see later), which serve to distinguish various types of biological phenomena persisting at the same time and one after another. This is developed later in Section 4. Rarity or uniqueness in terms of possibility and counterfactual

⁷ Currie (forthcoming, 204) analyzes positive and negative historicity in terms of our evidential access to the past and how it can be used for historical reconstruction. Currie draws from Wimsatt's work on generative entrenchment where dependencies accumulate and become necessary for future contingencies.

thinking does not quite capture that aim. However, the modal approach to evolutionary contingency identifies a second sense of contingency to contend with—contingency *per se*.

How might one consider the unpredictability sense of contingency or contingency *per se* as potentially causal? This sense of contingency emphasizes how an event or state might not have happened or might have happened differently. Here we are still thinking of evolutionary history counterfactually: a lack of necessity affects the probability distribution of outcomes across different replays. For example, the introduction of pelvic appendage gate patterns was potentially a causal factor in the evolution of walking and diversification in tetrapods (King et al. 2011). However, the existence of pelvic appendages is insufficient; more was needed, such as the use of a bottom substrate (i.e. a solid surface) to produce propelling force, digitated limbs, and terrestriality (King et al. 2011, 21146). The evolution of walking may have turned out very differently (or perhaps might not have happened at all) if the complexity with which the events took place over very large spans of time is considered. This second sense of contingency within a modal framework, then, amounts to causal insufficiency (see Turner 2011): any particular prior event (e.g. pelvic appendages) alone is not enough (e.g. for the evolution of walking). The evolutionary trajectory for tetrapod walking included an intricate network of other influences, pressure, and luck in addition to the evolution of pelvic appendages. However, trying to make sense of contingency's causality is particularly challenging if by stating 'contingency causes diversity' one means:

causal insufficiency causes diversity

How could causal insufficiency possibility cause diversity? The present setting of counterfactual thinking, possibility, and replays—i.e. what might have happened or not—employed by the

modal approach does not help my current quest for contingency's causality. Causal insufficiency causing diversity seems non-sensical to say the least.⁸ Rather, in the tetrapod case above, concrete causal factors were the morphology of pelvic appendages, or happenstance of location, avoiding annihilation, favourable selection, and stochastic processes.

To recap thus far, the dilemma's first horn includes problems with the modal approach. The modal approach to contingency provides two options for considering contingency's causality. On the one hand, contingency-as-causal-dependence leads us astray (i.e. to uniqueness and rarity of outcomes rather than to a diverse set). On the other hand, contingency-as-causal-insufficiency yields a nonsensical result for contingency's causality. Hope is not lost though. Next, we'll see how a process-based approach can make sense of contingency's causality, but not without its own obstacles to overcome.

3.2 The Dilemma's Second Horn: A Process-Based Approach

Here I explain the process-based approach to contingency before tackling two concerns. There are some foundational ideas in the literature for processes that cause contingent evolution. For example, Turner (2015) argues that what Gould really meant by 'contingency' was unbiased macroevolutionary sorting and passive trends more generally. At the microlevel (think genes, traits, organisms, local populations) natural selection is a process affecting the distribution of traits within populations in a way that is biased to the environment, whereas drift is an unbiased process. There are analogous processes at the macrolevel influencing large-scale lineages with species selection and unbiased sorting, which cause the persistence of some

⁸ It has been pointed out to me that if absences can be causes (e.g. not locking my door is a causal factor in my house being robbed), then why can't insufficiency as a *lack of causal power* itself be a causal factor? I'll leave the controversy over the causal power of absences and Tyler Goldschmidt's paper to the *Daily Nous*!

species over others. Unbiased sorting patterns can be statistically captured as passive, undirected (i.e. not directed by selection) trends. Turner's 2015 paper offers a starting point for what contingency means in terms of causal processes and how certain processes are responsible for contingent evolution. Additionally, Adrian Currie and I (2017) explicitly focus on the sources of contingency: the causal processes (i.e. mutation, drift, unbiased species sorting) in relation to some evolutionary pattern at a particular scale (i.e. microscale or macroscale). Rather than focus on the shape or modal profile of life's history, attention should be paid to the processes that produce the pattern in question. For current purposes, this means that contingency as a property of outcomes can be conceptually pulled apart from the sources or processes that cause contingent evolution.

Let's dig in further to the notion of a process. Consider Roberta Millstein's (2006, 679) claim concerning the difference between processes and outcomes as a starting point:

By "process" I mean a series of physical states occurring over time, whereas by "outcome" I mean the effect, or ending state at a particular point in time, of that process.

An outcome might be some pattern of gene frequency in a population that changes over time, some structural feature that serves a particular function, such as the limbs of vertebrates, an adaptive function, or even the emergence of cyclic metabolic pathways, and transmitted information through cellular signalling, etc. (da Costa and Galembeck 2016). Generally, while some outcomes are conserved through time, others undergo transformation through both divergence and change. Measuring the diversity of outcomes, then, amounts to individuation and counting according to some set of criteria. The processes responsible for outcomes can be evolutionary, developmental, biochemical, or ecological. Which means such processes might

include natural selection, mutation, drift, species sorting, organogenesis, signalling or signal transduction, cell adhesion, biosynthesis, and even geological factors like water cycles affecting wetland ecosystems, community dynamics, and so on. The process-based analysis of contingency I am presenting here secures a sensible account of contingency's causality because 'contingency causes diversity' means:

contingency-as-stochastic-processes causes diversity

That *processes* cause diverse outcomes is less controversial than either sense of contingency in the modal approach previously discussed. One need not worry about the strangeness of contingency as causal if by 'contingency' one just means some process or other. And so, to establish that contingency causes diversity calls for a positive connection between stochastic or chance-based evolutionary processes and diversity. Stochastic processes of interest might be, but are not limited to, the following three.

First, it's common to understand mutations—or random changes in nucleotide sequences unbiased to the environment—as a source of variation that selection works with. Though there has been a “neo-Darwinian prejudice against evolution through macro-mutations,” mutations of large effects can occur, for example, when deeply entrenched traits have many downstream dependencies (Schank and Wimsatt 1986, 39). Second, one can treat drift, namely the frequency of allelic (or gene variant) changes according to the random sampling of individuals in a population, as a force or process. The role of drift in evolution is a contentious notion. For instance, it is sometimes construed as a merely statistical effect and questioned as to whether random sampling could have an evolutionary effect in a population. Regardless, drift has been frequently “invoked to account for many apparently nonadaptive

patterns of variation” (Beatty 1992, 277). And third, species sorting according to Turner (2011) is the macroevolutionary analogue of random drift—certain species persist or not without directionality. As we have seen, Turner argues that “evolutionary contingency *is* the random or unbiased sorting of entire lineages” (2011, 69 my italics). To construe contingency as stochastic processes though leads out of the dilemma’s first horn, and right into the second.

What work is *contingency* doing if stochastic evolutionary processes cause diversity? The worry might be twofold: (1) If natural selection is the most significant evolutionary driver, why focus on unbiased processes? And (2) one can discuss the roles mutation, drift, and unbiased sorting have in producing diversity without needing contingency at all. So not only is the focus misguided (i.e. on stochasticity rather than selection), but contingency also becomes extraneous. Below is a response that keeps the focus on stochastic processes and still motivates the salience of contingency.

First, natural selection may not be the most significant evolutionary driver. There are good reasons to be suspicious of selection’s alleged all-encompassing power to dominantly drive evolution at all scales. Gould (2002, 505) discussed how the modern synthesis of Darwinian theory and Mendelian inheritance initially considered a plurality of mechanisms for evolutionary change. Prior to the hardening of the synthesis, the pluralistic phase “grant[ed] a greater role to randomness and nonadaptation in evolutionary *change*,” (523). Gould challenged the overreach of natural selection throughout his career, the culmination of which is reached in his last publication’s final pages. There he argued for Darwin’s own commitment to

contingency (2002, 1334). Simply put, there are well-known solid grounds to reject a narrow focus on selection alone.⁹

And second, contingency is not extraneous: *contingency matters in contradistinction to the adaptive, functional, convergent, and environmental-fit factors of natural selection*. By keeping stochastic, unbiased processes under the banner of contingency, there is something to be learned about the role of contingency at various scales. If it were the case that natural selection could act both dominantly and uncompromised by other factors, certainly some level of predictability would be at our finger tips. That is, so long as certain environmental conditions hold, natural selection as biased to the environment, should produce outcomes that converge on a function suitable to the challenge. Notably it is a large research project to establish convergences compared to divergent contingencies, which amounts to analyzing the relative significance of natural selection compared to chance in evolution. In other words, chance processes are grouped together under contingency typically in opposition to selection and other biased processes. What can be said about this oppositional relationship though?

Previously, chance influences, such as mutation, drift, and even recombination have been referred to as “degradational forces” that can hinder selection’s capacity to maintain against them (Schank and Wimsatt 1986, 44). Perhaps one can add to that list other contingent influences like external disturbances (i.e. extinctions caused by geological factors). Moreover, developmental constraints are often construed as restricting evolutionary change and adaptation, rather than, for example, positively channeling evolution (Pigliucci 2008). To take an antagonistic caricature of selection versus other forces is useful because it can affect how

⁹ See Gould and Lewontin (1979) for a critique of adaptationism.

evolutionary patterns of diversity appear to us. Such diversity is due to selection not having a clear shot: the power of selection is confounded by the forces of contingency. That is, stochastic processes, internal constraints, and even external disturbances affect the available strategies that natural selection has to cope with in addressing environmental challenges, but selection is not the only facilitator of change.¹⁰ As Brigandt (2007, 715) suggests, traditional evolutionary biology often proceeds to explain evolutionary change through natural selection acting on variation without attempting to explain *why* that variation occurs and its significance. And so, in the next section I explore stochastic influence in the production of variability, and how that variation can be pluralistically classified using a not-so-adaptively-focused approach.¹¹

In summary, this section included a dilemma: first, there is strangeness concerning contingency as causal in its traditional clothing of both contingent *upon* (causal dependence) and *per se* (causal insufficiency). Instead I adopted the process-based approach to contingency to circumvent the issue: If evolutionary contingency just *is* stochastic or unbiased processes, and stochastic processes are all concrete causes, then contingency as causal is less problematic. However, the focus on processes yields the dilemma's second horn: what role is left for contingency? I argued that contingency still matters in contrast to selectionist explanations. This is because contingency serves as a lens through which the history of life is approached by gathering matters of chance under its umbrella. Contingency as a modifier can apply to events,

¹⁰ Schank and Wimsatt (1986, 52) explore an experiment by Stuart Kauffman on gene control networks, which they argue showed significant limitations on the power of selection with changes in properties of the networks better explained by intrinsic constraints rather than selection.

¹¹ Stochastically-produced outcomes are not truly random in a way that circumvents rational explanation. Instead, such outcomes are "chance events from the viewpoint of function" (Noble 2013, 1236). It is better to think of stochastic processes as merely unbiased to the environment, rather than occurring purely without reason or pattern. This means that coherent explanations of diversity are still possible even when they draw from the role of chance.

processes, states, and so on, but even as a modal operator certain causal processes in addition to their end states and overall profiles all fall under its domain. Stochastic processes share in being non-adaptational or non-optimal modelling processes: united under the contingency framework they muddle the uniformity of selectionist models.¹² That uniformity is predominantly functional: it relies on the identification of how traits work in their respective environments regardless of their biological structure or form.

The distinction between ‘function’ and ‘structure’ is important for the cases studies in section 4. Adaptations as markers of selection are construed by the work they perform, and in terms of their responses to the environment. Similar environments yield a confined set of stable functions. This means that the differences among various structures of evolutionary mechanisms don’t matter insofar as they fulfill the same functional role. In other words, functional roles like heritability, variability, evolutionary individuality, etc. remain stable and constrained in selectionist models. Those functions remain stable despite important structural differences among the mechanisms by which they are realized.

4. Functional and Structural Measurements of Difference: How Contingency Causes Structural Diversity

So long as we distinguish among outcomes in number and have some way to measure differentiation, then a general notion of diversity begins to take shape (Maclaurin and Sterelny

¹² Does contingency just amount to any other evolutionary force other than natural selection on my account? McConwell and Currie (2017, 250-251) argue that natural selection can be a source of contingent evolution too: evolutionary outcomes can be sensitive to initial conditions such as a traits heritability for example. At the very least natural selection can be a source for historical contingency, but my emphasis on chance drives the contrast between processes under the banner of contingency and natural selection.

2008, 9). Up to this point, I have introduced contingency and argued that contingency is causal insofar as the stochastic processes shaping contingent evolution are causal. The aim of this paper is to show that contingency causes diversity, so in this section I rely on examples to demonstrate the positive influence of contingency for diversity production of a certain sort. In surveying the examples, I look for the influence of stochastic, unbiased processes, emphasize structural (rather than functional) differences, and discuss why those differences matter. However, before turning to those cases I'll introduce diversity more generally and how functional versus structural approaches can affect measurements of difference.

Different measures of diversities yield different sorts of diversities, which are, in effect, different kinds or types of outcomes. One main message of Maclaurin and Sterelny's work is that a single measure of diversity in a biological system is untenable (2008, 7). There are different sorts of diversities, such as taxonomic biodiversity in communities assessed by the number of species they contain (richness) and how evenly the individuals are distributed among species (evenness). Diversity is also assessed by functional interaction types or other ecological traits (Mougi and Kondoh 2012). There is diversity qua anatomical disparity where diversity is measured by the range of disparate anatomical body plans or structure of higher taxa, such as phyla (see Gould 1991, Wills et al. 1994, Foote 1993). Finally, but not exhaustively, there is phenetic and phylogenetic diversity of traits and organisms where degrees of similarities and differences are used to measure the diversity among shared traits (both morphological and otherwise) in phylogenetic trees, and the degree of divergence by evolutionary relatedness and common ancestry in branching-patterned cladograms. Though evolutionary systematics and cladistics are different ways to construe evolutionary relationships, they are also ways to

potentially track diversity in evolutionary history (Gould 1991, 420, Maclaurin and Sterelny 2008).

To determine whether there is diversity or not will depend upon measurement strategies and tools, which are sometimes coloured by presuppositions about the processes identified as dominant causers.

Consider the following scenario as a warm up:

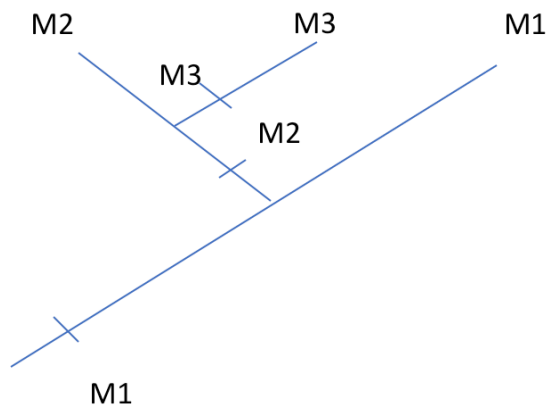


Figure 1. The evolution of sight. M1 represents mammalian eyes, M2 represents reptilian eyes, and M3 represents bird eyes. Functional and structural approaches classify these outcomes differently.

When can we say there is diversity? Because questions of diversity draw from ways to individuate and differentiate, a more traditional way to phrase the question might be: when has enough change occurred such that two different types of mechanisms can be identified, rather than one? Conversely, what needs to be conserved in order to say we have the same type of mechanism? These are tough questions and there are different tools to approach them that I'll refer to as 'functional' versus 'structural' strategies. This will help to elucidate what structural diversity amounts to. Suppose that M1, M2, and M3 represent mammalian eyes, reptilian eyes, and bird eyes, respectively. Both mammalian (M1) and non-mammalian

vertebrate (M2 and M3) eyes resemble the function of a camera in important ways. The quality of vision depends on the lens and cornea, and the cornea's shape influences visual accuracy or sharpness. Cornea shape is determined by the structure and biomechanics of collagen lamellar organization—layers of collagen fibers or ribbons.

On the one hand, with a functional approach M1, M2, and M3 can all be grouped together based on what they achieve in their respective environments. So, for example, even though the animals belong to different classes, the function of these traits in terms of their activities for some purpose (i.e. for sight) converge. Convergence is important because it shows that adaptation, and therefore natural selection, is at work in similar environments. Functional convergences are often considered as both marks against the significance of evolutionary contingency and demonstrative of natural selection's power. A functional approach might attend to the relationship of eyes with their environments, and also their parallelism or homoplastic status compared to traits in other species. By privileging adaptive function, this evolutionary history appears more constrained—the outcomes are less diverse, more convergent, and conservative when structural variation is ignored. On a functional account selection as *the* evolutionary process takes center stage. Conway Morris is well known for providing lists of convergences to serve as counter examples against the prevalence of evolutionary contingency.¹³ Functional analysis, however, is not the only way to represent and categorize evolutionary outcomes.

¹³ Convergences are also cited in support of evolutionary inevitability or predictability of outcomes (Conway Morris 2003).

On the other hand, one might approach the case *structurally*. To analyze differences in biological structure might involve large scale body plans that define different phyla, for example. However, there is also structure on smaller scales beyond total body plans, though the smaller scale features may help produce and maintain them. There is structural organization among cells, internal to cells, arrangements and biomechanics on various scales, and even organization and relations in general, such as the structure of ancestor-descendent relationships or community structure of interactions within ecosystems. Chance-based sources of contingency often play a role in altering these structures. And even though mutational sources, for instance, may become adaptive downstream, the classification of structural difference does not necessarily rely on function nor should evolutionary history be addressed only in that light. Structural details matter in informing our investigation of living things: they can be used to represent, classify, and categorize biological phenomena.

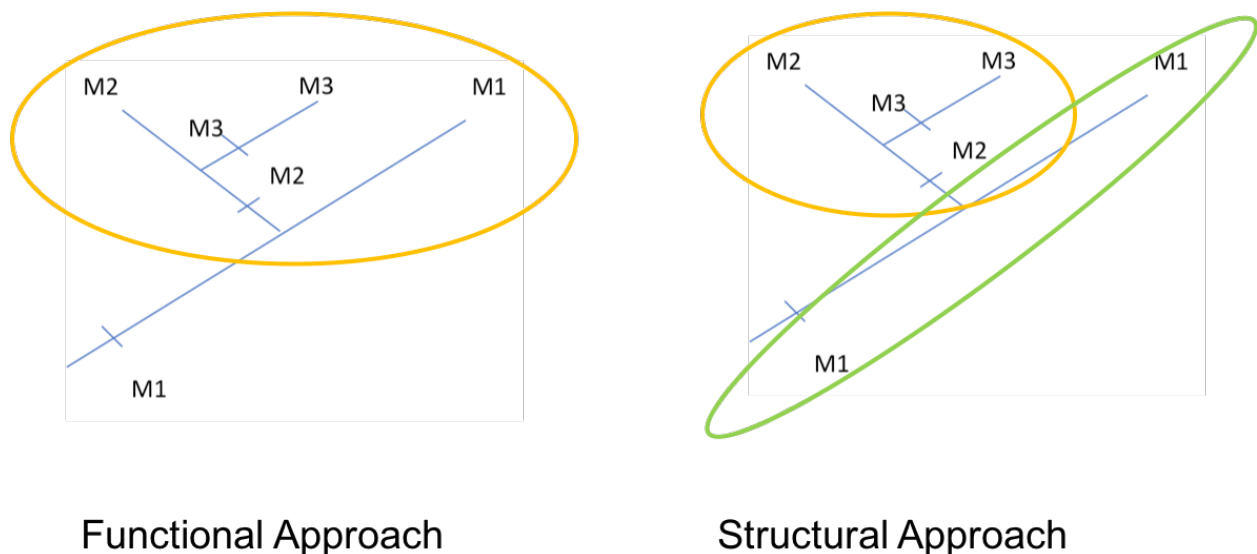


Figure 1a. A functional approach groups together mammalian (M1) and non-mammalian (M2 reptile and M3 bird) by how the traits perform in a given environment. However, while reptile and bird eyes share in structural organization, mammalian corneas are organized differently. Diversity is increased when approached structurally in this case.

Importantly, while non-mammalian vertebrate eyes share their structural organization, mammalian corneas are organized differently “suggesting a divergent evolutionary background” (Winkler et al. 2015). Recall that cornea shape is determined by the structure and biomechanics of collagen lamellar organization—layers of collagen fibers or ribbons. In reptiles and birds, the collagen sheets are broken down into ribbons and maintain a particular orientation, whereas in mammals the “layered collagen sheets are replaced by more dynamic fiber bundles” in random orientation (Winkler et al. 2015). Winkler et al. (2015) aim to understand the biomechanical structures in controlling corneal shape because of how that shape affects refractivity, thereby, affecting the sharpness of vision across species. Not only do the “mammalian corneas [exhibit] a radically different structural paradigm,” which suggests unique developmental programs, the visual experience in birds is very different from mammals—an important detail glossed over by a functional account (ibid). Birds have two to eight times increased visual acuity (or sharpness) compared to mammals, their visual fields are over 360 degrees with an increased ability to detect certain movement, and they have the potential to perceive ultraviolet light (Doneley et al. 2016). In order to classify sight as a functionally convergent trait across classes, these structural details are ignored.

Admittedly, it’s not that one approach—structural or functional—is necessarily *the* approach one must take. Assessing structural and functional (dis)similarity in order to understand evolutionary patterns is messy and complex. Love (2009, 13-14) puts it best when he says,

The typology is structural in some cases, whereas in others it is functional...and what counts as ‘structural’ and ‘functional’ also varies across disciplines. In some

cases, structural and functional considerations are mixed...what is grouped together is heterogeneous, including entities, activities, properties (size, weight, colour), processes (selection), mechanism, and time (normal stages).

The three cases explored next have structural diversities that tend to be finer-grained and revealing of differences in a way that functional accounts can overlook. Different processes produce different sorts of patterns, which means inflating the relative significance of selection in evolution yields a more constrained, functionally-glossed pattern of evolution. However, by paying attention to chance or unbiased processes and the structural variability they cause, revealed is a contingent evolutionary pattern far less constrained and much more diverse. A structuralist approach is not necessarily opposed to functionalist accounts. Instead, structuralism enriches our understanding of evolution. Admittedly, one implication of all of this is that functional analyses make evolution appear more constrained—is that the case?

It might be that *both* structure and function can be individuated more or less finely.¹⁴ Or, one might also explore what sorts of consequences structural differences can have for relevant functions just as the Losos Lab does by investigating different species of Caribbean *Anolis* lizards. However, that structure and function can be placed on descriptive continuums does not bear significant negative weight against contingency's productive relationship with structural diversity. Indeed, there might be some coarse-grained structural descriptions that in some sense have less divergent details than a corresponding functional one, or even structural convergences (such as the upper limbs of penguins and other birds) that have very different functions. So, while I suspect that structural diversities *tend to be* more fine-grained there is still

¹⁴ A reviewer for this paper raised concerns about the implication that functional analyses make evolution appear more constrained, and so they requested that something akin to that claim should be defended.

room for those types of exceptions. Additionally, Conway Morris has spent much of his career detailing functional convergences as evidence for constrained evolution. One good example is entitled “Life’s Solution: what happens when we re-run the tape of life?” (2008) where Conway Morris argues that evolution is constrained based on what he takes to be the pervasive nature of convergences defined as “near identical functional solutions” (2008, 207). Conway Morris (2003) is known for providing lists of those convergences as evidence *against* contingent and unconstrained evolution. And so, in contrast, I offer a case *for* structural diversity and unconstrained evolution.

In the following, I’ll discuss three examples that showcase how contingent evolution causes structural diversities. Any forward-looking sense of contingency has been heretofore discussed as either unpredictability (Beatty 2006) or causal insufficiency (Turner 2011). These accounts do reflect a sense of vast possibility space such that any structural flexibility found suggests future possible trajectories. However, I direct attention to the process-based approach: structural diversities are caused and distinguished by processes responsible for contingent evolution. Contingency causes diversity through the production of structural novelties and their transformation through time. I turn to three examples—the structure of the genetic code, the evolution of signalling pathways yielding cell-type diversity, and on a larger scale, anatomical body plans. I survey the examples briefly for the influence of stochastic processes and their role in producing structural (rather than functional) differences.

First, consider the genetic code, that is, the rules that govern how information encoded in DNA and RNA is translated into proteins that are comprised by amino acid sequences. Through this process certain combinations of nucleotides called ‘codons’ correspond to an amino acid in

constructing a protein.¹⁵ The rules, for example, determine which codons (or ‘nucleotide’ triplets such as U-A-C) bind to a tRNA molecule carrying the amino acid to be added next during protein synthesis. The genetic code was thought to be universal among all organisms, and a frozen accident unable to evolve further (Crick 1968). In other words, the genetic code is constrained and “impossible to improve because so many proteins depended on a fixed system” (Cornish-Bowden et al. 2014, 18).

However, not only are there alternative codes existing currently, there’s evidence of genetic code flexibility due to stochastic processes. Therefore, “it cannot be strictly fixed” (ibid 19). For example, there are small differences known in non-plant mitochondria and other systems such as intracellular bacteria, which have both alternative genetic codes and DNA structure. Mitochondrial code differs from the standard genetic code in its translation of certain codons (Jukes and Osawa 1990). In fact, Elzanowski and Ostell (2016) have compiled a list of more than twenty different genetic codes.¹⁶ Those codes include different rules for arrangements and relationships among codons and their corresponding nucleotide sequences. Additionally, while typical nuclear DNA is a double helix structure, mitochondrial DNA is a closed circular double stranded DNA molecule. In other words, there is something like a coarse-grained morphological disparity at the molecular level: the double helix structure of nuclear DNA is structurally different than the closed circular structure of mitochondrial DNA. *The genetic code as a singular universal phenomenon is only considered as such when*

¹⁵ Stop signals halt translation into proteins.

¹⁶ Elzanowski and Ostell’s list of genetic codes can be found at:
<https://www.ncbi.nlm.nih.gov/Taxonomy/Utils/wprintgc.cgi?mode=c>

differences among codes are ignored in favour of their functional similarities, such as “single direction reading” (see Kubyshkin, Acevedo-Rocha, and Budisa 2017 for more on similarities across coding events).

In order to establish that contingency causes code diversity we’d need to investigate the origin and evolution of the various genetic codes and the stochastic processes involved. I can only speculatively gesture at the direction one could take. Mitochondria are thought to be of separate evolutionary origin before the endosymbiotic event eventuating eukaryotic cells—the mitochondrion is the remnant of the engulfed bacterial cell. That two different types of DNA structures exist *within eukaryotic cells*, which are each guided by their own respective genetic code variants, is at minimum a structural diversity likely caused by a highly contingent endosymbiotic event wherein an archaeon engulfed a bacterium (Martin et al. 2015).

Moreover, any genetic code flexibility demonstrates future evolutionary potential for change into a new type, but how could changes to the codes possibly occur? In codon reassignment, the meaning of the codon is altered and thought to be lethal in causing mistranslation of genetic information. However, genetic code flexibility is revealed in *E. coli* (Mukai et al. 2010). Though the processes responsible for genetic code flexibility are not entirely understood, there is work on mutational processes (i.e. one source of contingency) to prepare *E. coli* for the codon reassignments associated with changes in DNA structure (ibid). We should be open to such flexibility marking the potential for change, even in evolution’s most basic (i.e. small-scale) structural components, such as genetic codes. That genetic mechanisms control the breeding habits and life cycles of organisms does not necessarily exempt them from change. Darlington (1939) argued that the genetic systems central to evolution were

themselves subject to change and constantly evolving. And Beatty (1980) has argued that meiotic mutants, such as nondisjunction, are evidence of evolutionary flexibility in the meiotic mechanism, and of the reproductive process itself that's integral to heredity. My point is that similar inferences can be made about evident flexibility in the genetic code.

Second, let's zoom out to a slightly larger scale: cell type diversity. While cell type classification can be a difficult task, traditionally it was achieved by identification of morphological (or structural) variability, such as inclusion size, shape, staining properties, organelles, and cytoskeletal structure (Vickaryous and Hall 2006, 426). More recently, gene expression and chemical constituents of tissues have been used, along with modern imaging techniques, to identify dendritic¹⁷ stratification patterns, for example (Arendt et al. 2016). Signalling pathways allow cells to communicate their identity to others, which may have facilitated the evolution of multicellularity. Cell-to-cell communication occurs through intercellular signalling pathways and other extracellular means where signals are transmitted, signals which are important for multicelled life. Pathways involve "systems of proteins that act in an orchestrated fashion, [and] mediate the response of a cell toward internal and external signals" (Soyer and Bonhoeffer 2006, 16337).

Because cell-type diversity is achieved through several different pathways (Vickaryous and Hall, 441), the contingent evolution of those pathways can be explored.¹⁸ There are various

¹⁷ Dendrites are short branching extensions of nerve cells, but there are dendritic cells that are part of mammalian immune systems named for their tree-like or dendritic shapes.

¹⁸ One reviewer suggested that even though cell type diversity is realized through multiple pathways (and I suggested the contingent evolution of those pathways be explored), this could be an example of a different type of convergent evolution. Perhaps the idea is that while one can distinguish between structural types based on contingent features and mechanisms, one might also distinguish between different functional types based on their prior histories (e.g. convergent evolution, parallelism).

pathways by which cell types arise each with definitional challenges analogous to the various species concepts.¹⁹ However, there are patterns of diversity used to identify corresponding cell types (Vickaryous and Hall 2006). Babonis and Martindale (2017) investigate the evolution of signalling pathways that facilitate cellular communication by tracking pathway phylogenetic history. They aim to understand the evolution of complex multi-component signalling pathways and the evolution of novel cell communication systems in general (2017, 14). The evolution of more complex signalling pathways, such as complexity-as-pathway-size, was likely needed for multicellularity to occur in order to navigate the terrain of different cell types within close vicinity. Soyer and Bonhoeffer (2006, 16337) show that such complexity can occur without any selective pressure for it, and that final pathway size tends to be lower and less complex when pathways evolve under strict selection criteria. They argue that pathways have an intrinsic tendency to become more complex resulting from mutational events on pathway response, mutations that yield the addition of new interactions or proteins. They discuss how the evolution of these pathways occur due to the “nature of mutational events affecting pathway structure” with only a nominal role for selection pressure on function (ibid). This suggests that the signalling pathways responsible for diverse types of cells, distinguished structurally, are caused mostly by stochastic processes and, as such, contingently evolve.

Third and finally, let's turn to largescale structural transformation in the origins of diverse novel body plans: diversity as anatomical disparity. There is disagreement over the amount of

¹⁹ Cell-type classification might call for philosophical analogues of various species concepts: Vickaryous and Hall (2017, 426) hope for unique cell markers to demonstrate unambiguous identity of cells, but despair about identity complications, such as how some cells can come to share features with recently encountered cells, and how identification is often up to the investigator.

disparity in life's early history (Wills et al. 1994, Foote 1993, Ridley 1993), especially by appeal to stem groups and changes in taxonomic methodology (see Brysse 2008, Briggs and Fortey 2005, Erwin 2016). Disparity is a technical sense of diversity closely associated with contingency because it articulates a diverse set of starting points. Diversity here is measured by the range of disparate anatomical body plans or structure of higher taxa, such as phyla.

Gould argued that the Cambrian explosion had a vast array of structural designs in this sense because Cambrian creatures were “so damned curious, different from one another and dissimilar from surviving lineages...life's greatest radiation” (1991 413, 416). However, even though typology is central to comparative morphology concerning form and structure commonalities and differences across taxa, structural types need not be just coarse-grained descriptions of anatomical traits. Structural types can be used to explain why some pattern is present or why certain traits are shared by a taxon (Brigandt 2017, 5).²⁰ It's also incorrect to think that structural typologies can't include variability and change (ibid, 2, 6). Not all changes are responses to environmental change or entry into the Simpson-esque pre-existing adaptive zones (Cavalier-Smith 2017, 12). Small mutations can cause huge changes, and in the right “organismal, phylogenetic, developmental, and ecological context, they [i.e. mutations] can make new phyla” (ibid). This certainly suggests that body plan design—the bodily forms or structures that distinguish higher taxa, are less constrained: phyla diversification was potentially caused by mutational change. Cavalier-Smith argues that the origin of sponges, for example, were “internal non-responsive innovations that worked” and that key mutations early

²⁰ Levin et al (2016) argue that phyla can be distinguished by mid-developmental transitions, which serves as an example of how structural typing can include fine-grained patterns and not just abstract anatomical descriptions.

in development can radically change animal phenotypes (ibid). In other words, “self creation of radical novelty can dramatically alter” and even positively channel selection producing ancestors with “unprecedented evolutionary potential” (ibid). If Cavalier-Smith is correct, then mutational processes—a source of contingent evolution—directly caused structural diversity on a very large scale.

Together the above survey of genetic codes, cell types, and phyla disparity serves as a lesson. Instead of thoroughly developing a singular case, my goal was to introduce a small variety of cases under the contingency framework. Each token case serves as a historical datum for the causal relationship between processes in contingent evolution and structural diversity found on a fine-grained, non-functional scale. The diversities of structural types above are distinguished from one another through stochastic means, which causes biomechanical variation and flexibility. By paying attention to structural differences, so long as contingent evolution continues, one can take this a step further: evolution is transformative, less optimal, and less constrained.

Overall, diversity has many dimensions. It is ill-advised to ask questions about diversity in isolation from the greater context in which they are embedded: background understandings of evolution colour the way diversity is approached. The evolutionary contingency thesis serves as an explanatory framework for diversity in biology: In the three case studies, chance-based processes cause structural diversity and the contingent evolution of those different mechanisms. One can then start typing the variability on that basis, i.e. classifying based on

structural differences, such that a plurality of biological phenomena follows.²¹ McConwell (2017) argues for a plurality of individuality types in selection based on chance processes and structural differences in variation and heritability mechanisms. I envision that something similar could be more fully developed with each of the three examples above. Recall a motivating undercurrent for working through contingency and diversity: If classifying variability into types or kinds distinguished structurally yields a pluralism, then pluralism in the biological domain is *explained by* the evolutionary contingency thesis. As I stated in the introduction, Hull (1987, 178) raised a concern about pluralism. He once proposed that remaining content with a plurality of concepts, such as species concepts for example, “might be admirably openminded and liberal, but it would be destructive of science” (ibid). Hull maintained that it was the best scientific tradition to opt for “one perspective and [push] it for all its worth” (ibid). I very much disagree. It’s not about remaining content as if to settle with biological plurality when that plurality is caused by contingent evolution.

²¹ A reviewer asks why I have not addressed simple cases: “Darwin would have said that structural variation within pigeons was due to the chance processes of accidental mutations. This process would help to increase variability in the population, and this is how we (sometimes) model mutation in population genetics, as introducing variation...there is an obvious case in which contingency causes diversity.” I avoid those cases because my aim is not to discuss accidental mutations as merely introducing variation for selectionist frameworks of evolution. My aim is to show how contingency, by causing structural diversity, results in pluralism. We sometimes find a plurality of x in the biological domain (kinds of heredity, for example), however, I aim to provide an *explanation for* that pluralism by appeal to evolutionary contingency as an explanatory framework.

5. Reconciling Productive Contingency with Gould's Diversity to Decimation Model

Lastly, recall that Gould did propose a diversity-to-decimation model, which included an initial explosion of diversity with many different body plans eradicated through the course of evolution (1991, 1993). Gould spent much of his career arguing for contingency's destructive power, yet in this paper I have argued that contingency is productive—it causes diversity. As I'll illustrate in this final section, my view is not in tension with Gould's. Gould's model does not necessarily discount episodes or explosions of diversity on different scales, even after periods of decline. Rather, contingency can be *both* destructive and productive. Let's take a closer look.

As mentioned previously, Gould was concerned with diversity as disparity, a technical notion referring to differences among anatomical designs (Wills et al 1994, Foote 1993). Rather than steadily increasing diversity with only a few forms at the base, Gould's model consists of a diverse set of starting points:

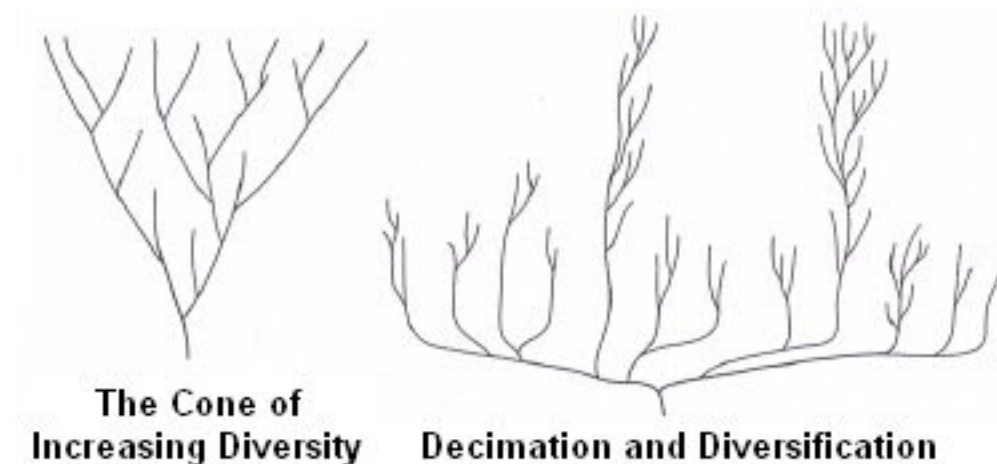


Figure 2. The traditional cone of increasing diversity with a less diverse base and increasing branching effects that can be classified together (see Brysse 2008, 308 on stem, crown, and total groups) vs. Gould's decimation model with mass radiation followed by extinctions of large groups.

Destruction is built into Gould's model, and arguably informed by his claims of evolutionary lottery and luck in the face of extinction for both local and mass scenarios. The role for contingency can be damaging of diversity unless active stabilization takes place for the "architectural depth" of structural characters (1991 417-18). Even Briggs and Fortey (2005, 108), both critics of Gould, also discuss how contingency factors into evolution and its diversity in a negative way.²² They state that contingency is involved "to the extent that the loss of many taxa during mass extinctions, particularly those attributed to asteroid impacts, is randomly determined" (ibid). On this picture, contingency is negative and backward-looking: in tracking evolutionary patterns, one finds them chipped away by fragile dependencies due to matters of chance both evolutionary and otherwise. While the previous section included case studies for contingency's productivity, consider the following as an example of contingency's destructive power.

There are cases where external disturbances are detrimental to mutualistic interactions, which are structural relations among organisms and species (Garcia-Algarra et al 2017). "Interaction networks play an essential role in the stability of ecosystems...as well as in the maintenance of biodiversity" (ibid). Interaction-type extinctions can cause a cascade of changes in communities and the loss of important ecological functions like pollination and seed dispersal (Jordano 2016). One popular example is the effect of drought on the extinction and breakdown of mutualisms between figs and fig-wasps (*Ficus-Agaonidae*). When such droughts are due to

²² Brysse (2008, 299) argues that changes in taxonomic methodology and the advent of stem group concepts that fit the "weird wonders" from the Burgess Shale with modern phyla, change the model of history to a less diverse base. Evolutionary systematics concerning similarity produces more phyla diversity compared to cladistics, which produces less.

weather events that cause temporary changes in the world climate, such as the El Niño, an increase in ocean temperature occurs due to complex interaction between wind and waves. In these cases, the very same entities present in those mutualisms no longer cooperate due to external disruption. Drought caused a break in the production of fig inflorescences, which is a special casing lined by small flowers that fig wasps enter to pollinate and lay eggs. Fig-wasps depend on the production of inflorescences, the lack of which resulted in local extinction of some pollinator species. This is, in effect, a story of contingency because the evolution of cooperative mutualisms is dependent on both historical and stochastic processes relevant to geology, meteorology, and oceanography. The evolutionary history of this complex relationship reveals a fragility: a sensitivity to disruptions that effectively shape the evolutionary profile of the fig-wasp mutualism. Presumably, catastrophic events causing mass decimation work similarly—there is sensitivity to disruptions that affect largescale evolutionary patterns and the organization of higher taxa too.

So, if contingency is destructive by constraining and demolishing pathways, how does the productively causal aspect of contingency square with the decimation model? I'll show that not only is diversity production compatible with the decimation model, but contingency's destructive power also contributes to diversity. Each will be taken in turn before concluding the paper.

There are at least three points that support the compatibility of contingency's productive and destructive aspects, two of which are found in Gould's own work. First, Gould (2002, 948) criticizes the speed with which recovery occurs after catastrophic events—this was of course his view that evolution proceeded episodically and with a punctuated character, rather than

gradual. It is in his description of evolution's tempo and mode one finds a hint of positive diversity production. Gould thought that "episodes of recovery from maximum decimation at the extinction to full reestablishment of previous levels of diversity occur more quickly" until the next mass extinction of global biota (ibid). The diversity-to-decimation model, then, can be viewed as an all-encompassing picture of life, and also as a *more local, and repetitive, snapshot in time*: diversity bounces back even if to a lesser extent. Second, Gould's model was about anatomical disparity, which on his model exceeded modern levels. However, species diversity in the Cambrian was much lower than now despite having a greater range of body plans. Contingency can span scales: on the decimation model, an increase in diversity is only controversial depending on the sort of diversity in question.

Third, and albeit a little more speculatively, while identifying causes of the Cambrian diversification event is a contentious issue, the actual body plans themselves may have been generated by chance. Recall the Gouldian balance of both external environmental factors causing selection for adaptive traits and internal-type forces that constrain inheritance and development. History and chance have positive roles in generating evolutionary pathways that reflect both inherited patterns and environmental pressures. According to Gould, contingency destroyed the Cambrian body plans, which constituted a subset of workable but lucky survivals among a much larger set that "either never arose, or lost their opportunities, by historical happenstance" (2002, 1161). However, Gould (1989, 53) was also critical of direct and simple preCambrian ancestors of phyla in the Cambrian explosion. Drawing from more recent research, a major factor in the radiation was alterations to the way Hox genes (i.e. genes responsible for basic segment morphology) were put together, their expression, and

downstream signaling effects (Penumaka 2011, 47). This might mean that in early history before pathways were entrenched, mutations could alter organism development in a way that *produced*, rather than only destroyed, actual body plan diversification.

And finally, consider a version of Will Bausman’s (2016) useful metaphor tailored to explain how contingency’s destructive power can still produce diversity.²³

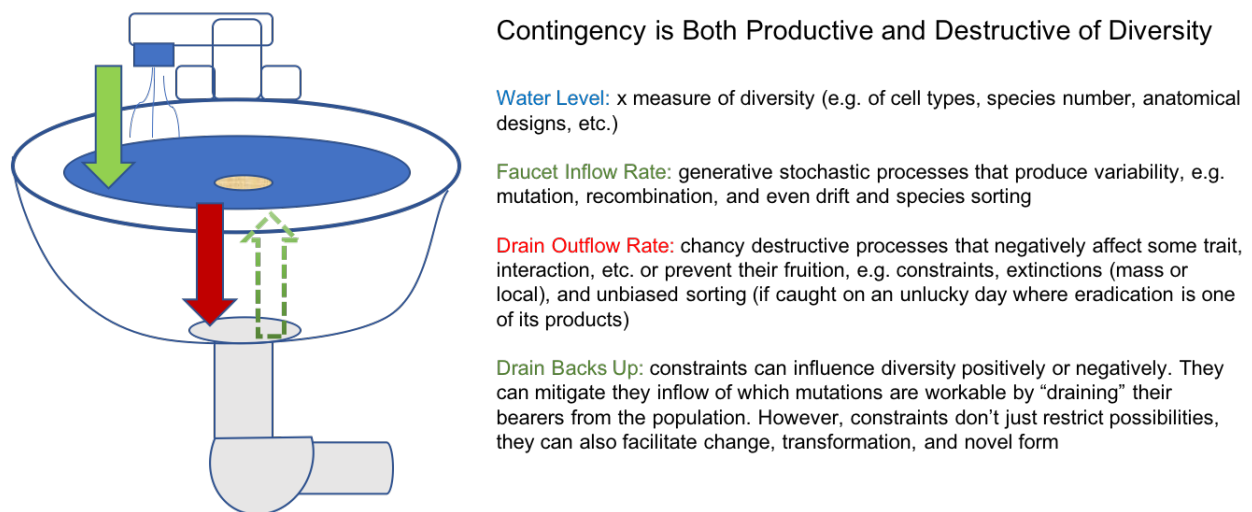


Figure 3. Contingency both produces and destroys diversity, but insofar as diversity is just a measurement of variability according to some criteria (e.g. just as one might use different tools to measure liquid volume), production and destruction really just are two sides of the same coin.

I view this metaphor as a tool: another way to approach how contingency can be both destructive (as Gould argued for) and productive (as I have argued for). Imagine the water level in your sink as analogous to a certain measure of diversity—cell types, species number, ecological interaction types, even anatomical disparity, etc. The inflow rate of the faucet

²³ Bausman (2016, 8, 224) explores neutral theory—a theory of patterns of kinds of species in an ecological community. The primary process is ecological drift: demographic stochasticity or random birth and death similar to the MBL model that explains evolutionary patterns stochastically and without selection. He discusses how species can maintain an equilibrium despite speciation and extinction (as a product of drift), as analogous to how the water level in your sink is explained by the inflow rate from the faucet and the outflow rate of the drain. Solving how species number remains constant is like “solving the sink problem when the water level is constant” (21).

includes generative stochastic processes that produce variability—mutation, recombination, and maybe even drift and its macrolevel analogue (i.e. species sorting) if you catch them on a lucky day. The outflow rate of the drain includes chancy destructive components that affect the diversity measure “negatively” by taking away some trait, interaction, species, etc. or preventing them from reaching a certain level. These destructive processes might be extinctions (mass or local). Or they might be unbiased sorting processes if caught on an unlucky day where extinction and eradication are their products.

Sometimes the very same processes (i.e. unbiased sorting processes) can play both productive and destructive roles depending on their products. We might also include internal developmental constraints that both guide and prevent pathways. Destructive processes in contingent evolution alter diversity by helping to maintain an equilibrium or simply just a change in measurement outcome. Similar to how your drain can balance a high inflow rate from the faucet or affect possible water levels either positively or negatively, developmental constraints mitigate what is made possible by mutation, for instance. Constraints influence the patterns of diversity and as such are not exempt from understanding it. However, if we think that diversity measures transformational change over time, rather than *only* more-different-things-added-at -one-time, then the drain as our destructive and restrictive processes causes that sort of diversity too. Drains, from time to time, do back up after all and shift the composition of what’s in the sink. One might consider this in terms of Gould and Lewontin’s spandrels, which arose from morphological evolution driven not by selection, but by “architectural-developmental constraints” (Brigandt 2014). Brigandt (2014) also argues that constraints do not just restrict possibilities; they can also facilitate change, transformation, and

novel form. Contingency both produces and destroys diversity, but insofar as diversity is just measurement of variability according to some criteria, production and destruction really just are two sides of the same coin.

6. Conclusion

In conclusion, the aim of this paper was to elucidate what Gould left out about contingency. I demonstrated how contingency can be an engine of diversity in a way that amounts to more than Darwin's idea of mutation and chance as engines for mere variation. Gould primarily discussed contingency as a destroyer of diversity, which leaves a gap concerning contingency's productive role—a role that is more significant than merely natural selection's servant or "lackey." However, as I discussed, his decimation model does not preclude contingency's role in causing and maintaining structural diversity either. I provided a defense for thinking of contingency as causal and explored how contingency causes structural diversity. Mechanisms dependent on stochastic, unbiased processes of contingency, which distinguish different types of the following: cells, modes of inheritance, DNA structures and genetics codes, types of interactions, and body plans. Classifying based on structural differences yields not just one mechanism type, but a plurality. These structural variations are not merely inputs for selection, but evolutionary outcomes readily classified into a plurality of types regardless of environmental functions.

Importantly, the persistence of structural diversity has consequences for the tension between Beatty and Hull's work concerning pluralism and evolutionary science. Recall that Beatty questions the expectation of primarily constrained evolution. I tried to further that agenda: Rather than assuming evolution resulted in only one outcome, i.e. one set of cell types,

one mode of inheritance, or one genetic code, etc., from a structural approach one should *not* expect evolution to be so constrained. Contingent evolution diversifies structures that can be sorted into a plurality. To accept such pluralism does not amount to simply giving up before our query is settled in a way that might be destructive of science, as Hull worried about. Instead, evolution is not so constrained: there is good reason to *expect* pluralism working within the framework of evolutionary contingency.

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