RECENT WORK

The Philosophy of Biology

Christopher J. Austin

1. Introduction

The biological sciences have always proven a fertile ground for philosophical analysis, one from which has grown a rich tradition stemming from Aristotle and flowering with Darwin. And although contemporary philosophy is increasingly becoming conceptually entwined with the study of the empirical sciences with the data of the latter now being regularly utilised in the establishment and defence of the frameworks of the former, a practice especially prominent in the philosophy of physics, the development of that tradition hasn't received the wider attention it so thoroughly deserves. This review will briefly introduce some recent significant topics of debate within the philosophy of biology, focusing on those whose metaphysical themes (in everything from composition to causation) are likely to be of wide-reaching, cross-disciplinary interest.

2. Evolutionary classification

In a post-Darwinian age, one of the most important and well-known changes in our philosophical thought about the biological world has been the paradigm shift wherein attention turned away from sharply and eternally defined *natural kinds* and so, away from *individuals* as a central theoretical focus, and towards vaguely bounded and contingently stable *species*, with *populations* taking the theoretical fore (Hull 1965; Mayr 1994; Sober 1980). In this shift, an organism's developmental architecture and its role in trait-building was de-emphasised in favour of analysing instead the correlational statistical trends between population traits and their corresponding genomic profiles. However, this shift also brought a renewed focus on another, more finely grained class of individuals known as *homologues* – modular organismal sub-systems responsible for the building of a particular trait that are present throughout successive generations of organism groups which exhibit broad morphological similarity among their various (intra- and inter-species) instances over traceable lineages of modification history (Scotland 2010). A prime exemplar is the *tetrapod limb*: examining the limbs of everything from bat wings to human arms seemingly reveals the existence of a single *archetypal* individual whose morphologically distinct instances are ordered

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by relations of successive variation in distinct groups of organisms over evolutionary time-scales. However, the contemporary effort to better understand these 'evolutionary individuals' has raised an important philosophical question, one which reflects the aforementioned paradigm shift: do these individuals carve at the ontological joints of the evolutionary landscape, or are they merely heuristic abstractions constructed from it? Deciding between these two perspectives – the *developmental* and *phylogenetic*, respectively – is the focus of the debate on the nature of homologues.

The phylogenetic view, the view most naturally aligned with the spirit of the populationist conception of species, has a *variation first* perspective: homologues are merely 'idealized types' constructed from comparative paleontological and anatomical studies on the morphological similarity of a certain feature among organism lineages (Cracraft 2005; Grant and Kluge 2004; Love 2009). On this view, in other words, although the morphological differences between the wing of a bat and the arm of a human can be subsumed under a certain structural type, they are so only conceptually, and only as a matter of convention – the 'unity' between these morphologically disparate forms doesn't amount to *identity*. The developmental view, taking a *stability first* perspective, sees it the other way around: the 'type' that defines a homologue consists of a set of intrinsic causal capacities which persist through, and are themselves explanatory with respect to the morphological particularities which it displays throughout its historical lineage. On this view, one and the same structure lies beneath the bat's wing and the human's arm, and the morphological disparities between them are only varied surface-level reflections of the generative potential of that underlying structure.

Although the phylogenetic approach is the historically prominent one, recent years have seen a rise in defenders of the developmental perspective, in large part due to the advent of advances in evolutionary developmental biology (evo-devo), a research programme centred on studying evolutionary transitions in organismal form *via* the modification history of the developmental mechanisms underlying homologous structures (Canestro *et al.* 2007; Davidson and Erwin 2006; Wagner 2014). However, according to the phylogenetic perspective, its rival faces an insurmountable task – that of illustrating genuine (and not merely conceptual) *unity* among the morphological variations of a homologue throughout its occurrence in a wide variety of organisms in such a way that each is an instance of a *single*, *repeated* individual; as Owen's (1848) famous definition makes clear ('[t]he same organ in different animals under every variety of form and function'), 'homology' is about *sameness*, not mere *similarity*.[1]

1 It should be noted that phylogenetic accounts have an analogous problem of sorts, given that they require a precisely defined notion of a 'character' in order to be able to subsequently pick-out/define a varied set of the *same* character over lineages. This isn't trivial – see Richards (2003).

Without wishing to deny the substantial morphological variation among their instances, developmentalists, trading on a popular move in the metaphysics of 'natural kinds' (Boyd 1999), have suggested that perhaps a homologue is best understood as a *homeostatic property cluster* (HPC) – a collection of phenotypic features which are *mainly* present in *most* of its instances, though which particular sub-set of those features are present in any particular instance will vary over evolutionary time (Brigandt 2009; Keller *et al.* 2003; Rieppel 2005; Wilson *et al.* 2007). The upshot is clear: on this conception, a homologue is not defined by *all* or any *one* such feature (nor any particular sub-set thereof), and thus the morphological variation of those features throughout its instances poses no individuative problems. Plausibly though, if the unity that HPC-homologues are meant to provide to their variously distinct morphological instances is to be taken with ontological sincerity, it must be one underwritten by a shared developmental mechanism which is present in each of those instances, and which is in some way causally responsible for the clustering of certain features throughout them (Elder 2007). However, discerning such a mechanism has proven problematic as even the most paradigmatic homologues are now known to have been underwritten by a series of distinct developmental mechanisms over evolutionary timescales – a phenomenon commonly known as *hierarchical disconnect* (Ereshefsky 2009; Hall 2003; Müller 2003).

In light of this phenomenon, some have argued that the homeostatic mechanisms which ground the 'unity amidst variation' of homologues need not solely consist of *intrinsic* properties, but might also be comprised of *extrinsic* ones – namely those which specify historical, lineage-specifying relations – e.g. lines of descent and gene flow, histories of selective pressures etc. (Elder 2007; LaPorte 2004; Wilson *et al.* 2007). These extrinsically specified mechanisms will certainly be 'present' in every instance of a homologue (given that such histories aren't variable), and be explanatory with respect to the presence of a particular sub-set of clustered features in any particular instance – but is that enough? Recall that for developmentalists, the nature of a homologue is meant to play an important role in causally determining the *structure* of that clustering over time – that is, in determining *which* particular morphological features are the possible and likely members of those clusters throughout its instances. That relevant homeostatic mechanism must, in other words, be responsible for the *developmental constraints* which give that structure its shape – a prominent theme in evo-devo research (Brakefield 2011; Hendrikse et al. 2007; Laland et al. 2015).

With that in mind, some have confronted the challenge from the phenomenon of hierarchical disconnect by identifying a homologue not with any specific molecular mechanism, but rather with a particular higher-order, idealised causal structure which many such mechanisms may realise – one which maps out its underlying mechanism's potentialities for morphological variability, and so reflects the causal contours of its developmental constraints (Brigandt

2007; Hallgrimsson et al. 2012; Otsuka Forthcoming; Wagner and Stadler 2003). On this view, while the individuation of homologues remains an intrinsic affair, and the identification of a homologue across its various instances in some way ontologically depends upon molecular mereology, the identity which unites those instances is grounded not in the uniformity of their constituents, but in the equivalency of the developmentally salient causal-*cum*-modal structure instantiated by those constituents.[2] In allowing the individuation of homologues to be conceptually unfettered from their molecular moorings, and by basing that identity in the specified potential for morphological variation, this sort of view avoids many of the aforementioned worries. However, its dependence on abstract measures of developmental potential (and the limits thereof) courts the further concern that it may in practice be rather difficult to discover and distinguish homologues in this fashion: discriminating between morphological variations which have not appeared throughout various instantiations of a homologue because its nature causally prohibits them from doing so and those which are so permitted though absent as a matter of mere historical accident, for instance, is a subtle and difficult affair (Olson 2012; Ramsey and Peterson 2012).

Interestingly, addressing the complexities that accompany this idealisation strategy may turn out to be an unnecessary conceptual detour as recent empirical evo-devo research has suggested that the phenomenon of hierarchical disconnect may be merely superficial, and that there might be intrinsic molecular mechanisms which ground homologue identity after all: namely, smaller subsets of the developmental mechanisms which are generatively responsible for homologue development – often referred to as 'kernels' or 'character identity networks' – which have been shown to be phylogenetically invariant throughout various intra- and inter-species instances of certain homologues (Davidson and Erwin 2006; Wagner 2007, 2014). Whether the stability of such finely grained sub-systems over evolutionary timescales is sufficient to establish metaphysical *identity* among the morphologically disparate forms of homologues, and whether the generative competency of these systems can be shown to be sufficient in causally controlling the specified development of homologues (and the various permutations of their instances) remains to be seen.[3]

3. *Organismal ontology and explanation*

Although they may diverge with respect to *how* and *why* the developmental machinery which underwrites homologues are biologically significant, both

2 Due to their definition *via* an abstract causal structure which tracks developmental modalities, a case can be made that developmental constraints are at least partly established by *extrinsic* factors – especially selective pressures. See Sansom (2009).

3 For empirical case studies on this capacity see Hallgrimsson et al. (2007), Young et al. (2010) and Rasskin-Gutman and Esteve-Altava (2014).

the phylogenetic and developmentalist perspective agree that such sub-systems function as *modular* units of ontogenic construction. But what precisely are these *developmental modules* of which individual organisms are composed? Following the outstanding successes of the reductionist paradigm in twentieth century biology, and in the wake of the advent of systems biology, the most popular answer is that organisms are fundamentally 'made of' *mechanisms* (Canestro et al. 2007; Hall 2003; Laubichler 2010).[4] This *new mechanism* movement (Bechtel and Abrahamsen 2005; Glennan 2002; Machamer et al. 2000) has two component claims – one concerning mereology, the other the nature of explanation.[5] The first claim is that organisms are composed of discrete collections of distinct 'elements' which are structurally organised and causally connected by their various isolatable 'activities'. The second claim is that organismal alterations are best conceptualised as the causal product of these activities' facilitation of a step-wise, temporally successive series of state-changes in these elements.[6] In virtue of its appeal to particular sets of entities and their interactivities, this mechanistic model is purported to provide a more causally discerning, 'ontic' form of explanation (Craver 2014; Salmon 1984) – one whose heuristic value outstrips the mere *post-hoc* predictive prowess of the once prominent deductive-nomological model (Bechtel and Abrahamsen 2005; Kaplan and Craver 2011).[7]

As intuitively plausible and widely adopted as this new mechanism movement is, both of its central claims are not without scrutiny in the contemporary literature. Consider first the mereological claim. Note that the ontological division of organisms into *discrete* mechanistic sub-systems is meant to reflect the *modularity* of their development, a feature which is now widely recognised to be a *sine qua non* of their participation in the evolutionary process (Callebaut and Rasskin-Gutman 2005; Erwin and Davidson 2009). In virtue of what then are these collections of entities and activities discrete? In other words, what defines their *boundaries*? An intuitive answer is that some *causal relevance criterion* will do this work: because every member of a mechanism must be causally connected to some other, the ontological border of a

4 For a good overview of the philosophical distinctions between 'machines' and 'mechanisms', their historical interplay within the history of science, and what's 'new' about the new mechanism movement, see Allen (2005) and Nicholson (2012).

5 There are mechanists who go beyond the explanatory claim and hold that biological causation itself *just is* mechanism-mediated influence, but this is an extreme view that I don't have time to consider here. For an overview, see Williamson (2011), and for a critique; see Casini (2016).

6 The degree to which these models generalise 'outwards' is a subject of contention, with some going so far as to propose a mechanistically-mediated causal model for the process of natural selection. For discussion, see Skipper and Millstein (2005) and Barros (2008).

7 Although it's an intuitive notion, determining precisely what an *ontic* mode of explanation amounts to and what distinguishes it from *epistemic* modes isn't without its difficulties – see Wright (2012) and Illari (2013).

mechanism plausibly traces the edges of its interactionist web.[8] One way that this criterion has been metaphysically cashed out is *via* Craver's (2007) *mutual manipulation model*, built from Woodward's (2002) influential account of causation. The model proposes a simple test for mechanism membership: any alteration in the activity *of an entity belonging to a mechanism* must result in an alteration in the activity *of the mechanism*, and vice versa.

Though *prima facie* plausible, a bi-directional boundary building test based on counterfactual discrimination may be problematic in the biological realm (McManus 2012). For instance, in one direction of dependency, it may be too restrictive, and generate false negatives: the holistic, mechanism-level activity of complex biological systems is often impervious to minor alterations in the activities of their constituents – a phenomenon known as *robustness* (Kitano 2004; Whitacre and Bender 2010). In the other direction, it may be too permissive, and generate false positives: a large swathe of organismal features (both morphological and behavioural) bear counterfactual dependence relations to extra-organismal, environmental stimuli, as evidenced by the well-known phenomenon of *phenotypic plasticity* (Fusco and Minelli 2010; West-Eberhard 2003).[9]

Having a sufficient condition for membership is crucial, as mechanisms are individuated by their composition – namely, by their mereological constituents (entities) arranged in a specific spatio-temporal and causal structure (activities). Because individuation criteria are linked to persistence conditions (as the latter fail to be met whenever the former do), this entails that any particular mechanism *persists* just as long as its specific mereological composition does. But here too a seemingly plausible tenet of the mechanistic conceptualisation of organismal sub-systems has been subject to scrutiny. As mentioned above, biological systems are notoriously *robust* to perturbation and can continue appropriately functioning in the event of losing some subset of their mereological make-up either in virtue of (i) their possession of a redundant duplicate sub-set which takes up the lost set's causal role within the system (MacNeil and Walhout 2011; Zhenglong et al. 2003), or (ii) elements within the system forming novel causal connections which collectively compensate for the loss of that set's role to retain functioning (Edelman and Gally 2001; Mason 2010). The worry here is simple: according to their composition-based individuation criterion, the mechanist appears committed to the truth of the counter-intuitive claim that, metaphysically speaking, no

8 Recent attempts to conceptualise the 'developmental hourglass' may lend credence to this idea: in this period, either there is *no* modularity (because no boundaries of causal connectivity exist), or there is *just one* module (because there is a single interactionist web). See Galis and Metz (2001), Kalinka et al. (2010) and Stergachis et al. (2013).

9 Due in part to the widespread acknowledgement of the importance of this sort of phenomenon, the more general question of what exactly it is for an *organism* to be properly *bounded* is both vibrant and open. See Pradeu et al. (2011) and Bouchard and Huneman (2013).

organismal sub-system genuinely *persists* through such robustness phenomena, as the loss of a constitutive element, or the acquisition of a novel causal connection among those elements marks the appearance of a novel mechanism. Intuitively though, this is the wrong result – one and the same system *is* robust, and its exhibiting that robustness ought not amount to its dissolution.

Some mechanists see the worry as misplaced, noting that the compositional stability that individuates mechanisms is merely a heuristic necessity applicable only to *models* of mechanisms: the biological realm is not mereologically dissected into frozen collections of unalterable clockwork, even if our models of that realm must be (Bechtel 2015; Brigandt 2013; Craver 2006; Levy and Bechtel 2013). Others have taken a more ontological angle, arguing that the individuation of mechanisms ought to include their active role in delimiting a certain range of ontological dynamism among their constituents and activities: given that the characteristic function of biological systems is often (if not always) expressed *via* a kind of controlled fluctuation of their compositional elements and the causal connectives between them over time, a directive principle of constitutional dynamism must be incorporated in to our 'mechanism' concept (Bechtel and Abrahamsen 2010; Brigandt 2015; Kaplan 2015).

The judgement that this latter move is a rather desperate attempt to salvage a sinking mereology has fuelled the resurgence of the alternate understanding of the nature of these systems found in *process ontology*, according to which what is robustly stable in the biological world are not particularised collections of causally structured entities, but the more general *patterns of activity* in which various such collections participate over time (Dupre 2013; Henning and Scarfe 2013; Jaeger and Monk 2015).[10] On the process perspective, the essence of a biological system is the *dynamical flow* which constitutes its proper functioning and which *persists* throughout the veritable Heraclitean flux of the underlying elements which realise that activity at any time (Cahoone 2013; Jaeger and Monk 2015). For its defenders, only a process ontology is capable of accurately modelling both the developmental and evolutionary phenomena associated with biological systems as their flexible, problem-solving nature effectively outstrips the conceptual categories that a mechanistic mereology affords.

Those unimpressed with the mereology of the new mechanists have also expressed scepticism regarding its other central conceptual pillar – namely, the claim that biological phenomena associated with organismal sub-systems are best explained mechanistically. There's little doubt that the conceptualisation

10 Interestingly, Waddington (1957), the main proponent of a process ontology and a corresponding non-mechanistic, topological explanatory tool-kit for the biological realm was deeply influenced by the modern philosophical progenitor of that ontology, Whitehead (1925).

of such phenomena as the causal products of step-wise, temporally successive series of state-changes in structurally organised sets of elements is one which outperforms its deductive-nomological theoretic predecessor: it delivers an ontologically discerning model whose predictive prowess is both nuanced and comprehensive. However, though the mechanistic model has been richly explanatory with respect to a wide variety of biological phenomena, recent years have seen a rise in interest in the predictive pedigree of a class of models which are strikingly *non-mechanistic*: they are not constituted by spatio-temporally structured sets of localised centres of causal interactivities, and their explanatory power is not derived from tracing the spatio-temporal transition of the state values of a set of discrete elements. Instead, these models typically represent ontogenic systems *holistically*, viewing them as a kind of higher-order, causally unified entity, and describe their primary structure *quantitatively*, often *via mathematical* relations, especially non-linear, partial differential equations. In contrast to the mechanistic explanatory strategy, these models do not offer *compositional* explanations for biological phenomena, ones which appeal to particularities of the system's mereological make-up and their arrangement, but rather *formal* ones, grounded in mathematical, abstract structural features of the system as a whole (Winther 2006).

A well-known paragon of the successful utilisation of this non-mechanistic strategy is *dynamic systems theory*, wherein organismal sub-systems are modelled as abstract, multi-dimensional state spaces bounded by a set of axes which represent genetic expression levels and composed of coordinates which represent possible total system states (i.e. possible system-wide genetic expression values).[11] In these models, each point within a system's state space is not only vectorized towards its neighbours, but also assigned a certain stability measure (represented by an additional, upward axis) that reflects the probability of the system to transition from its current total state to a neighbouring one. The resulting state-space is a structured topology of high peaks and low-lying valleys where the characteristic functioning of the system is modelled as the tracing of a continuous series of temporally successive transitions through the coordinates of that space – from the 'heights' of regulatory *instability* to the 'depths' of regulatory *stability* (Davila-Velderrain *et al.* 2015; Huang 2012; Wang *et al.* 2011). Importantly, on this model, the particularities of that functioning are *explained* by the topological features of state space: appeals to the curvature gradient of its slopes, the directed descent of its valleys, and the high walls of its basins all feature in geometric-*cum*-kinetic explanations of the specificities of the system's dynamics with respect to

11 I've limited my discussion here to dynamic systems theory, but there are plenty of other non-mechanistic explanatory models which have recently been the subject of discussion and debate: so-called 'design principles' come to mind – see Braillard (2010), Green (2015), Brigandt et al. (Forthcoming).

everything from *robustness* (Huang 2009; Huneman 2010; Kitano 2004) to *developmental constraints* (Jaeger et al. 2015; Wang et al. 2011).[12]

While those advocating for a process ontology have expectedly embraced and claimed for their own the impressive non-compositional explanatory power of these non-mechanistic models, the reaction from the new mechanism perspective has been varied. On one extreme, a prominent response has been that, contrary to appearances, these dynamical models possess no *genuine* explanatory power. This denial is based on the claim that these models *misrepresent*: they capture a merely 'phenomenal' feature of a system, one derived from and dependent upon the particularities of its underlying dynamic structure. The worry is that such a model is incapable of meeting a plausible requirement for being genuinely explanatory – namely, having the capability of being utilised to discern the *causal foundations* of a system's production of that phenomenal feature (or any others) in a principled fashion (Craver 2008, 2014).[13] Significant predictive utility notwithstanding, any model that represents a system *abstractly* is one which fails to properly carve it at its causal joints, and is thus incapable of being utilised to genuinely *explain* the occurrence of any phenomena it may otherwise usefully serve to predict.

A more measured response has stemmed from the general recognition that a model's being in some sense *abstract* doesn't entail its inability to accurately discern and capture a system's *causal* structure, and therefore its inability to genuinely possess explanatory power (Reutlinger and Andersen Forthcoming). An elucidation of this idea is the claim that if these abstract, formal models are genuinely explanatory with respect to their phenomena, they are so in virtue of their features being appropriately mapped on to an underlying mechanistic structure – that is, just in case their mathematical variables correctly correspond to sets of entities, and their equations accurately capture the causal relations among them (Bechtel and Abrahamsen 2010; Kaplan and Bechtel 2011; Kaplan and Craver 2011). According to this perspective, the empirical success of the explanatory principles employed in higher-order, dynamical models doesn't pose a serious threat to the reign of the mechanistic explanatory paradigm, as any heuristic advantage that the former type of model might have is one ultimately awarded to it by the merits of the latter.

However, perhaps the most popular response to the empirical successes of non-mechanistic models has been to embrace *explanatory pluralism* – the view that there are many equally valid ways in which models might explain

12 DST is now rather widely applied in analyses of everything from sub-organismal cell-fate (Bhattacharya et al. 2011; Verd et al. 2014) to the evolvability of organism populations (Jaeger and Monk 2014; Striedter 1998).

13 Though see Franklin-Hall (2016) for some doubts on whether mechanistic models themselves are capable of sufficiently meeting this requirement.

biological phenomena. Most who adopt this position view abstract, non-compositional models as conceptually complimentary to their mechanistic counterparts: they are non-overlapping magisteria, each offering unique explanatory virtues either to distinct types of phenomena, or else to distinct facets of the same phenomena (Brigandt 2013; Green *et al.* 2014; Mekios 2015; Thery 2015). It may be, for instance, that the formal structures of these abstract models, constructed as they often are from mathematical-*cum*-topological relations, license an explanatory prowess of a distinctly *stronger* sort: instead of merely providing causal explanations of particular states of the systems they represent, they may also elucidate certain modal features of those systems which explain the constraints on all of their *possible* states (Breidenmoser and Wolkenhauer 2015; Huneman 2015; Lange 2013). Moreover, if the formal structures of these non-mechanistic models are capable of correctly capturing central features of the causal architecture of biological systems (as suggested above), the adoption and subsequent refinement of such models may even heuristically aid in the process of mechanism discovery and elucidation (Baetu 2016; Fagan 2012; Zednik 2011).[14] Explanatory pluralism's popularity doubtlessly derives in part from this characteristically conciliatory approach: it permits non-mechanistic explanation to be both possible and uniquely powerful without its being methodologically privileged.

4. *Developmental information*

Deciding which ontological framework best captures the nature of the organismal sub-systems causally responsible for specified morphological development, as well as within which modelling schema that responsibility is best situated is of central importance. However, quite independent of the outcome of those decisions is another, more fine-grained and arguably, foundational metaphysical issue to be adjudicated concerning the nature of the fundamental causal-*cum*-structural feature of nearly every significant biological system, and so the proper definition of one of the most fundamental concepts in biology – *information*. But for a concept so ubiquitous and indeed, essential in the elucidation of both developmental and evolutionary processes, it is one upon which there is a great divergence of opinion.

By far the most widely discussed and controversial application of the concept of *biological information* is to the relation between gene and form, where the genome is said to 'contain information about' the morphological profile of an organism. Very roughly, that relationship is thought to exist because, according to the standard conception of information introduced by Shannon (1948), the states of the genome (its particular members and their

14 See MacLeod and Nersessian (2013) for an interesting case-study in this kind of complimentary research activity.

order) are robustly correlated with the states of that profile (its possession of certain phenotypic features and their particularities). This correlative co-variance of state values is meant to demarcate the specialised and *unique* role that the genome plays in establishing the 'developmental program' of organism-building, and thus, its privileged place in the conceptual hierarchy of the causal structure of ontogenesis.

However, recent empirical research has called the uniqueness of this role into question, and with it the developmental centrality of the genome. For we now know that the wide-range of intra-specific variations on the phenotypic features which comprise an organism's morphological profile are causally correlated with variations in extra-organismal, environmental factors – this is the phenomenon of *phenotypic plasticity*, now thought to play a crucial role in the process of natural selection (Gilbert and Epel 2015; West-Eberhard 2003; Whitman and Agrawal 2009). How precisely one ought to respond to the seemingly natural implication of this phenomenon – namely, that an organism's environment also 'contains information' about its morphological development, and in just the same way its genome does – has divided philosophical opinion. Some philosophers, in endorsing what's known as *developmental systems theory*, have understood the informational parity that obtains between genome and environment as reflecting the presence of a more general and comprehensive *causal parity* according to which there simply are no ontologically privileged causal factors in the process of development (Griffiths and Hochman 2015; Oyama 2000).[15] Most have taken this to mean that the supposedly unique, more robust sense in which the genome 'contains information' about that process is either a heuristically useful fiction (Levy 2011), or else merely a conceptual artefact of an illustrative metaphor (Griffiths 2001).

But perhaps the most prevalent reaction in the literature has been to insist that the genome *must* contain information about the process of development in a way that extra-genetic factors do not, and that adequately capturing that fact will require a reformation of, or refinement on our current concept of information (at least, in its biological context). The focus towards that end has largely been centred on the genome's principal role in shaping that process – its being causally responsible for the production of the proteome, the set of macromolecules which actively regulate the orchestration of, as well as mereologically make up, morphological features. One particularly prominently explored avenue has been to claim that the information relation that exists between gene and protein is substantially more robust than can be captured by the conceptual framework of Shannon information: it is characterised by the correlative co-variance of state-values, but it also has two additional important features which are often attributed to so-called *semantic*

15 There are a variety of ways in which the notion of *parity* is fleshed-out in developmental systems literature – see Stegmann (2012) for an overview.

concepts of information. Firstly, unlike the relation of Shannon information which makes no directional distinction between 'source' and 'signal', it is *asymmetric*: as enshrined in Crick's (1958) 'Central Dogma' of molecular biology, there is a privileged 'direction of flow' of information between gene and protein such that the former *informs* the latter, but not vice-versa (Godfrey-Smith 2007). Secondly, unlike the relation of Shannon information which is an 'all or nothing' affair, it is *error-capacitive*: the information which genes contain about their particular protein products remains intact even in cases where various disturbing factors result in that content being *misrepresented* in the proteins which result from their expression, as is the case in, for instance, splicing errors (Griffiths 2006).

These two features are meant to indicate that the information which genes contain about their protein products has content richer than can be expressed by the entropy-measures of Shannon correlations, but the question then is: what precisely does this *semantic content* amount to?[16] A popular answer is that this content is a non-representational form of *semiotic* meaning: in the context of the interpretative role of the cellular machinery of transcription and translation, the triplets of nucleotide bases which comprise the genome are *symbols* for their protein products (Barbieri 2003; Godfrey-Smith 2000; Sterelny 2000).[17] Given that the correlation between the genome and its particular protein products is in an important sense *arbitrary* – that is, it is not strictly determined by the physio-chemical properties of the genome, but is instead established *via* the accidental vagaries of its selective history, this code-based semantic content is understood to be suitably *naturalistic* (Bergstrom and Rosvall 2011; Maynard Smith 2000; Sarkar 2003). Indeed, following Millikan's (1984) influential *teleosemantic* account of meaning, the typical understanding is that the 'selected-for' symbolic content the genome possesses grounds its normative character: it dictates the proper *direction* of information flow, as well as whether that information is *misrepresented* in particular cases. That said, there is reason to think that the teleosemantic account of the informational content of the genome is incapable of capturing the unique relation between it and the proteome. It's questionable, for instance, whether the informational content determined by a selected-for history is sufficiently able to capture the robust *prescriptive* element of that relation (Kjosavik 2007; Kumar 2014). Even if it were, it's unlikely that the containing of such information would single-out the genome in any privileged fashion, as teleosemantic content about the proteome seemingly

16 Semantic concepts of information are meant to feature *meaningful content* in a richer sense than, for instance, the 'natural meaning' proposed by Grice (1957) and subsequently adopted by Dretske (1981).

17 A notable exception is Shea's (2007, 2011) 'infotel' account of semantic information, which assigns the genome truly *representational* content (even *about* phenotypic traits) on account of its selected-for history.

must also be attributed to accidental and unrelated 'genetic hitchhikers' (Davies 2001; Wheeler 2007), and potentially even the environment at large (Jablonka 2002).

Those who have judged the teleosemantic account as inadequate have typically offered an external critique, arguing that it is mistaken *ab initio*: rather than attempting to derive semiotic content from the historically contingent, selective assignment of particular genome–proteome relations, we ought instead to do so from the particularities of that relation itself – that is, from the causally unique character of that relation within the process of ontogenesis. By far the most prominent approach from this perspective has been an attempt to show that it is the *causal specificity* of that relation which captures the 'content' of informational significance which is unique to the genome (Davidson 2001; Weber 2006; Waters 2007; Griffiths et al. 2015). One quite popular way of spelling that out utilises insights from Woodward's (2003, 2010) *manipulation account* of causation, showing that the state of genes causally co-vary with the states of proteins in a very 'fine-grained' fashion (in a way that other developmentally salient causal factors do not): alterations in the states of the proteome bear a systematically precise pattern of counterfactual dependence upon the alterations in the states of the genome (mutations, deletions etc.) such that minute changes in the former reliably track minute changes in the latter.[18]

A prevalent way of cashing out that idea is grounded in the *instructional* role of the genome – that is, its operation as a causally selective *ordering function* which determines the linear, structural organisation of its protein products (Bogen and Machamer 2011; Ruiz-Mirazo and Moreno 2006; Stegmann 2005, 2014; Sustar 2007). Relying more closely upon the conception of the genome as a *symbolic code*, according to the concept of what's sometimes referred to as 'Crick information' (Griffiths and Stotz 2013), the genome's semiotic content consists in the sequential ordering of its constitutive elements which serve as a causal *template* for the specific serialised patterning of the amino acids which compose proteins. While no one disputes that the genome plays this causally specific role in protein production, opinion is divided on whether that role is sufficiently robust for it to entail the genome's containing *information* in any rich sense: the correlative relation between gene and protein is a complex one, and due to a variety of pre- and post-transcriptional cellular processes, it is one which can often fail to exhibit the covariance sensitivity which an *informational* relation seemingly requires (Stotz 2006; Stotz and Griffiths Forthcoming; Wheeler 2007). However, recent computational analyses have indicated that even in particular instances of strict covariance failure the proteome unfailingly preserves a rather specific 'genomic footprint', and the possibility of tracing that template

18 Woodward's criterion for causality has its roots in Lewis's (2000) relation of 'causal influence'.

throughout the translation process may be sufficient for attributing significant semiotic informational content to the genome, though this requires a more subtle analysis (Sarkar 2003; Scherrer and Jost 2007; Stadler et al. 2009).

Whether any of these ways of cashing out the content of 'genetic information' – be they semantic or semiotic – offer a promising way forward in elucidating the now elusive link between *gene* and *form* remains to be seen. With the advent of contemporary systems biology and increasingly precise experimentation techniques, we now have a deeper understanding of the developmental architecture responsible for morphological generation as consisting not just of sets of genes, but of genetic regulatory networks – sets of genes intimately interconnected in dynamically structured systems of causal interdependencies (Busser et al. 2008; Davidson et al. 2002; Kitano 2002). It may be that the causal-*cum*-structural complexities of these networks requires any understanding of that link to take place within a novel, more distributive conception of 'information' (Jablonka 2002; Keller 2009; Pigliucci 2010; Stotz and Griffiths Forthcoming), perhaps even one wherein these networks are more akin to information *processors*, rather than *producers* (Austin 2015; Calcott 2014; Planer 2014). Whatever the way forward, getting clearer on whether and to what extent the conceptual framework of a suitably refined information theory offers the heuristic resources to understand the process of morphological development and ultimately, the process of evolution, remains a central concern for philosophers of biology.

University of Oxford
Oxford OX2 6GG, UK
christopherja@gmail.com

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