The Dispositional Genome: *Primus Inter Pares*

Christopher J. Austin

According to the proponents of Developmental Systems Theory and the Causal Parity Thesis, the privileging of the genome as "first among equals" with respect to the development of phenotypic traits is more a reflection of our own heuristic prejudice than of ontology - the underlying causal structures responsible for that specified development no more single out the genome as *primary* than they do other broadly "environmental" factors. Parting with the methodology of the popular responses to the Thesis, this paper offers a novel criterion for 'causal primacy', one that is grounded in the ontology of the unique causal role of dispositional properties. This paper argues that, if the genome is conceptualised as realising dispositional properties that are "directed toward" phenotypic traits, the parity of 'causal roles' between genetic and extra-genetic factors is no longer apparent, and further, that the causal primacy of the genome is both plausible and defensible.

What is it about the causal nature of the *genome* that singles it out, over and above all the other causal factors present within the cellular architecture, as *primus inter pares*? According to the proponents of 'Developmental Systems Theory', the simple answer is *nothing*. Although it is certainly true that at least since the time when the molecular basis and structure of the genome was discovered and detailed it has occupied a privileged theoretical role in every subsequent respectable scientific ontology, according to the 'developmentalist' perspective, "…the empirical differences between the role of DNA and that of [the surrounding cellular architecture] do not justify the metaphysical distinctions currently built upon them"[1]. The denial of these "metaphysical distinctions", on the part of the developmentalist perspective, is based upon the affirmation of the *causal parity thesis*, which could be stated as follows: there is no genuine, principled, ontological distinction between the causal contribution of genetic factors and those of non-genetic factors that establishes one type as *causally primary* with respect to the specified production of proteins, and thus the formation of phenotypic traits.

In this paper I argue that, armed with a proper understanding of the nature of the causal structure of the genome, the thesis of causal parity is false. The argument I offer is based on the plausible supposition that the genome realises dispositional properties. I claim that if the unique causal role of dispositional properties is properly understood, a powerful response to the causal parity thesis is at hand. Armed with that understanding, we are afforded a novel conception of *what it is to be* 'causally primary' with respect to some effect. That conception of causal primacy, I argue, is well-suited to provide an answer to the developmentalist argument, and to declare the genome as causally primary with respect to the formation of phenotypic traits.

1. *Dispositional Properties and their Genetic Realisation*

Dispositional properties are properties that, for a lack of a better word, *dispose* their bearers towards being in some state. They do so in virtue of being properties which are causally productive of those states once

[1] Griffiths & Knight (1998: 254)

they have become "activated" by the occurrence of some other state of affairs – in the philosophical parlance, dispositional properties have 'stimulus' conditions which activate the causal production of their 'manifestation' states. In the realm of philosophy of physics, 'negative charge', for instance, is often taken to be a dispositional property, one which disposes its bearers to 'repel with such and such momentum' (manifestation) upon the occasion of 'meeting with a like-charged particle' (stimulus).

These properties, in a certain sense, act as "switches", in that they are a kind of causal channel through which the influence of certain states of affairs is mediated through in order to bring about some other state of affairs. When, and *only* when the switch is flipped (by the occurrence of the appropriate stimulus conditions), the "target" state of affairs (the manifestation) is brought about. In other words, the possession of a dispositional property *alone* does not ensure that the state of affairs the property disposes its bearer for actually comes about – it must be "activated" by the occurrence appropriate stimulus conditions. Importantly however, even "un-activated" dispositional properties are still "directed toward" their manifestation-states - eternally un-broken vases are nevertheless 'fragile' – and they remain "ready" to perform their role of causal mediation from stimulus to manifestation irrespective of their activation.

On account of these facts, dispositional properties are *individuated* by their particular stimulus/manifestation pairs, and they are often characterised as 'functional' properties – properties that are defined by a particular *causal role*. Being defined by a particular causal role might be captured by the slogan: "you *are* what you *do*". Think of familiar functionally defined systems: *anything* that plays the causal role of luring in and trapping mice *is* a mouse-trap, anything that plays the causal role of quantitatively adjusting according to temperature *is* a thermometer, etc. To return to the realm of properties, any property that "plays the causal role" of causing its bearer to shatter upon receiving a rough blow *realises* the property of fragility. Importantly, when we designate a particular structure/process/etc. as playing a particular causal role, we operate at a certain level of abstraction – one that eschews the specific details of the unique casual pathway *by which* that role is performed and focuses on the general, resultant state *for which* that pathway is in place. This means that when we attribute a dispositional property to a particular entity/system, we are very often (if not always[2]) forming an abstraction that "reaches over" a wide, multi-stage causal gap from stimulus conditions to manifestation state. This sort of abstraction is admissible only when the causal pathway from stimulus to manifestation is *stable* – that is, when the causal process that begins with the former state and reaches all the way to the latter is reliably repeatable.

There are immediate conceptual correlates in the structure of genetic causation with respect to protein production, and I think it is quite plausible to claim that the causal pathway that begins with the first spark of transcriptional activity and ends with the completion of translation activity can be understood as one that runs from 'stimulus' to 'manifestation'. Recall the causal processes that underlie the genome's specified production of proteins – *transcription* and *translation*. In order for a particular gene to begin the process of transcription, whereby a complementary strand of mRNA is produced, various causal factors have to be in operation – the most primary of these being the multi-protein enzyme RNA Polymerase and its activity of binding to the 5' end of that gene and beginning the complex process of "unwinding" along it in a 'transcription bubble'. RNA Polymerase's binding at the 5' site of a gene is a causal requisite for that gene's beginning to produce a protein. Once an mRNA strand has been produced, and a ribosomal complex binds to it, individual, specified tRNA macro-molecules carrying

[2] The general agreement by most metaphysicians that all dispositional properties, as a matter of principle, are capable of being "interrupted" *after* receiving their stimulus and *before* they come to their manifestation – a consequence of the so-called "problem of masks" - is, I think, plausibly construed as the assumption that every dispositional property's manifestation lies causally downstream from its stimulus conditions.

particular amino acids then bind to that strand and begin assembling into what will be the completed polypeptide sequence – that is, the finished protein.

What is important about these processes is that although there are a number of complicated and complex interactions that occur in both transcription and translation, there is a reliable and stable causal pathway that begins with the binding of RNA Polymerase and ends with a translated protein product. Not only is that pathway a stable one but it is also one whose every step causally depends upon the step that preceded it, and not just in the mundane sense that 'causes' precede 'effects': there is a specified, template-based instance of symbolic translation that stems from the transcribed gene to the causally downstream translated protein. The causal process that begins with the binding of RNA Polymerase to a particular gene and ends with the translation of a particular strand of mRNA in to a specific protein is then both *stable* and can be *functionally* defined – because of its determinative stability, we can safely abstract from the complexities of the various interconnected causal pathways to the larger, singular causal process.

This, I think, is all that is required to begin to conceptualise the genome as realising dispositional properties. If it is the case that upon certain conditions being met, the genome becomes causally responsible for the causally downstream production of a certain effect, this looks to be an instance of the genome *realising* a dispositional property. And that is exactly what I claim to be the case: each particular deoxyribonucleic macro-molecule whose collection of codons "code for" the amino-acid sequence of a particular protein *realises* a particular dispositional property that is picked-out (primarily) with reference to its manifestation state – i.e. the polypeptide chain/protein that it produces/specifies/"codes" for[3].

1.1 *The Causal Link between Stimulus and Manifestation*

When an entity/system possesses a dispositional property, the entity/system reliably and repeatedly reaches a particular state (the 'manifestation' state) upon the occurrence of some other state (the 'stimulus' state)[4]. It is because dispositional properties establish, for their bearers, a genuinely causal link between two states of affairs (or property exemplifications) such that the obtaining of the first state is reliably correlated with the obtaining of the second, that metaphysicians have characterised those properties as "truthmakers" for subjunctive conditionals – most notably, counterfactual conditionals. That said, the applicability of the relation of truthmaking – a "cross-categorical", non-symmetrical relation between a concrete entity and a propositional entity such that the truth-value of the proposition causally depends upon the existence of that entity[5] – to dispositional properties has had a chequered past: the well-known context-sensitivity of "variably strict" counterfactuals[6], and the inevitably torturous (and arguably *ad hoc*) insertion of *ceteris paribus* clauses, has caused many to disparage making such a connection[7].

However, although I don't wish to endorse the existence of a truthmaking relation between dispositional properties and subjunctive conditionals, one can see the appeal: counterfactuals, for

---

[3] I say "primarily with respect to its manifestation state" because it is typically the *products* of genes that we are most interested in when we are doing the business of biological classification.

[4] "Reliably and repeatedly" is meant to express that the causal link from 'stimulus' to 'manifestation' is not one of necessity, but neither is it one of contingency – for it furnishes reliable inductive inferences for the bearers of those properties. See Mumford & Anjum (2011: Chapter 8)

[5] See Armstrong (2004) for the canonical formulation.

[6] Lewis (2001); Stalnaker (1968)

[7] See Mumford & Anjum (2011) for a thorough rejection of dispositional properties as truthmakers for subjunctive conditionals

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

instance, seem properly licensed when there is a reliable and repeatable causal connection between one state of affairs and another – "if this, then that" is arguably (minimally) true just in case there is such a reliable causal pathway from *this* to *that*. The point I wish to draw out is simply that dispositional properties are responsible for establishing a causal connection between two distinct states of an entity/system, one that operates in the aforementioned fashion.

Importantly however, as many metaphysicians have now recognised, that causal connection is not a simple one-to-one relation, and the two states that are so connected are not merely binary[8] – they are instead capable of having multiple values, and so are best thought of as *variables* which can take a number of quantitatively distinct *values* (or, in the philosophical parlance, as *determinables* whose instantiations are *determinates*). Consider the dispositional property of 'fragility', defined by the stimulus condition of 'receiving a rough blow', and the manifestation 'shattering'. There are many different, quantitatively distinct instances of a 'rough blow' which a fragile entity might suffer in order for it to 'shatter', and likewise upon receiving a 'rough blow' there may result various distinct instances of 'shattering'.

So although 'fragility' is defined/individuated via the variable/determinable-level states, specificity at the "value-level" occurs during particular manifestation events in that, for every instance of 'fragility' being manifested, there is a precise *value* of the *variable* 'rough blow' (say, "receiving a blow of *this particular strength, at this particular angle*,…") and a corresponding precise *value* of the *variable* 'shattering' (say, "shattering *in this particular fashion, at this particular speed*…"). A 'fragile' entity then is one that, upon receiving any range of particular determinate *values* of the determinable, *variable*-like state of affairs "receiving a rough blow" responds by exhibiting any range of particular determinate *values* of the determinable, *variable*-like state of affairs "shattering".

The 'causal link' that a dispositional property establishes between a stimulus-state and a manifestation-state is best characterised, I think, by a species of what Lewis (2000: 190) called the relation of *causal influence*, where there is a *pattern of counterfactual dependence of alterations of the determinable manifestation value upon alterations of the determinable stimulus value*, such that there is a substantial range S_1, S_2, etc. of quantitatively distinct, determinate-level alterations of the determinable state S, and a range M_1, M_2, etc. of quantitatively distinct, determinate-level alterations of the determinable state M, such that if S_1 had occurred, M_1 would have occurred, and if S_2 had occurred, M_2 would have occurred, etc[9]. In other words, the link that dispositional properties establish is one of *causal co-variance of state-values*, and each dispositional property functionally relates a series of fine-grained, distinct stimulus states with a series of fine-grained, distinct manifestation states.

The character of this type of causal link is apparent too, I think, in the genome – and it only strengthens the case for conceptualising the genome as realising dispositional properties. Note that the process of transcription, whereby the causal process from gene to protein is initiated, is not a simple, binary one – it is a well regulated, gradient-based input and output function. Although an entire wealth of intra-cellular causal factors are important in the production of proteins, consider here a central type: 'transcription factors'. Transcription factors are a class of proteins that function to "control" the flow of transcription – that is, the *amount, rate* and *timing* at which DNA codons are "read" by RNA Polymerase to produce strands of mRNA. Transcription factors bind to certain sequences on the DNA strand (known

[8] Dispositions of this sort are generally referred to as "multi-track" dispositions – see Mumford (2004), Jacobs (2011), Martin (2008), Manley & Wasserman (2008), and Vetter (2013)

[9] Keeping in mind that these counterfactuals are meant to express "reliable and repeatable" connections, and are, as mentioned above, sensitive to context, etc.

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

as 'enhancer sequences', or 'cis-regulatory elements') that effect a gene's transcriptional output in three important ways: (1) by turning transcription on and off (2) by increasing or decreasing the rate of transcription and (3) by ensuring that transcription takes place only during certain stages of ontogenetic development. Transcription factors achieve this control over the flow of transcription by means of a variety of chemical reactions upon their binding with DNA sequences, most of which are quite complicated.

All of the precise biochemical details of *how* transcription factors do their work in regulating the process of transcription is not important here – what's important is that the complex group of causal factors that initiate that process do not do so in a simple "on/off" manner: there is instead a wide-range of specific and determinate values of that group which are associated with a complementary range of specific values of the production of proteins. In other words, for any particular gene, alterations in the determinate value of the complex of causal factors that are required for the initiation of the processes of transcription and translation causally co-vary with the determinate value of the production of a specific protein. If, as I have suggested, we conceptualise genes as realising dispositions whose stimulus conditions are the various causal factors needed to initiate the processes of transcription and translation, and whose manifestation states are completed proteins, we can recognise that phenomenon as the *dispositional* relation of 'causal influence': it being the case that subtle changes in the causally relevant complex of transcription factors are reliably associated with correspondingly fine-grained changes in the in production of proteins *just is* the relationship that holds between the 'stimulus' and 'manifestation' states of a dispositional property.

More to the purposes of this paper, and on a larger, genome-wide perspective, this fine-grained, dispositional relation of causal influence is exhibited in the phenomenon of 'phenotypic plasticity', where a *single* genome is associated with a *set* of interrelated, yet quantitatively distinct forms of a particular phenotypic trait - its so-called *reaction norm*. In light of this phenomenon, a particular phenotypic trait is viewed as "…the unique consequence of a particular genotype developing in a particular environment", and the reaction norm of that genome with respect to that trait is conceptualised as "…a list or graph of the correspondence between different possible environments and phenotypes that would result"[10].

Because the intra-cellular environment of a gene – the constituents of the cell membrane containing the nucleus-enclosed DNA – can be significantly altered by the extra-cellular environment of that gene – its surrounding cells and their constituents, and more to the point at hand, the external "environment" of the organism as a whole - the "environment" of an organism can play a crucial role in determining the "environment" of a gene – namely, the presence and absence of transcription factors within its host nucleus. This is important because a particular phenotypic trait is not produced simply by specific proteins *being present*, but by their being present *in a particular fashion* – at particular times and in particular places (as well as during certain period of development generally). Thus, changes in the timing of gene expression (heterochrony), and changes in the location of gene expression (heterotopy) - that is, changes in the "pattern of expression" of genes - results in the alteration of the particular form of a phenotypic trait[11].

In other words, if the presence or absence of the transcription factors associated with a particular set of genes is altered, the pattern of expression of those genes are altered and, as a consequence, the phenotypic trait whose formation depends upon the proteins produced by those genes is altered. In this way, the "environment" that an organism finds itself in, via influencing the presence, number and type of

[10] Lewontin (1982: 21-22)
[11] Schlichting and Smith (2002); Whitman & Agrawal (2009)

transcription factors present within an organism's cells, is causally correlated with changes in the specified development of particular phenotypic traits. Indeed, changes in even the most mundane of environmental conditions, for instance, in external temperature, can have a wide-ranging, fine-grained effect in everything from the size of the wings[12] to the number of bristles on the back[13] of developing *Drosophila* fruit flies.

We are now in a position to conceptualise the genome as realising dispositional properties in a larger, more complex sense – for, importantly, dispositional properties are not limited to having a "realisation base" of a *single* member, nor is it necessary that their manifestations be *simple*[14]. With that in mind, the philosophical picture that the phenomenon of phenotypic plasticity paints, on my view, is that of the genome possessing complex dispositional properties, realised by distinct sets of individual genes, whose stimuli are particular sets of transcription factors (in certain configurations, at certain times, etc.) and whose manifestations are the various quantitatively distinct forms of particular phenotypic traits, represented by their respective reaction norms[15]. Of course, unlike the dispositional properties just discussed, these dispositional properties are realised by a *complex* of individual genes in the genome, and their pathway from stimulus to manifestation is a wide, multi-stage causal gap – much wider than, for instance, the pathway from gene to protein. But this abstraction is admissible, I would again argue, because that pathway is *stable* – the causal process that begins with the influence of environmental conditions (first extra-, then intra-cellularly) and ends with the resultant specified production of a particular form of a phenotypic trait is reliably repeatable; that it is so is the reason we can construct informative lists and graphs *of* reaction norms. On my view then, the causal connection between the "environment" of the organism and a particular phenotypic trait is one of 'causal influence', and it is established via the existence of a particular set of genes' realisation of a dispositional property.

2. *Developmental Systems Theory and the Causal Parity Thesis*

Having argued that the genome realises dispositional properties, both at a gene-protein level and, as a consequence, at a genome-phenotype level, I now want to argue that this conceptualisation can offer us a novel criterion of *causal primacy* – one that singles out the genome as "first among equals" with respect to phenotypic traits. But before that can be done, in order to properly set the stage, we need to examine why it is that proponents of Developmental Systems Theory (**DST**) endorse the *causal parity thesis*, as this will provide a useful, contrastive background against which my proposed criterion will be made more clear.

It is no overstatement to say that, at least in the field of the *philosophy of* biology, **DST** and the thesis of causal parity are now very much in vogue[16]. Generally, the thesis of causal parity is that there is no genuine, principled, ontological distinction between the causal contribution of genetic factors and those of non-genetic factors that establishes one type as causally primary with respect to the specified

---

[12] Powell, Davis, and Powell (2010)
[13] Gibson (1970)
[14] In fact, I think that even the seemingly most mundane of dispositions exhibit this level of complexity. Take the philosophers' favourite "simple" disposition – 'fragility': the realisation base of 'fragility' is undoubtedly (at least) a multi-faceted, complex chemical structure, and its manifestation – 'shattering' – is no "simple" event, but must be comprised of the aligning of various micro-events that represent decreasing degrees of structural integrity.
[15] Of course, transcription factors *alone* do not comprise the full 'stimulus' of these dispositional properties, as many other intra-cellular causal factors are required for the initiation of the process of transcription – a fact not ignored in the following discussion of the causal parity thesis. They have been singled-out here only because their central importance in the *regulation* of transcription, and thus, the genome's pattern of expression.
[16] For some prominent defences of the 'developmentalist' perspective, see Oyama (2000), Griffiths and Knight (1998), and Gray (1992).

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

production of proteins, and thus the formation of phenotypic traits. For some advocates of **DST**, the thesis of causal parity is a simple position founded in the symmetry of logical dependence between various causal factors and some effect: the point is made either that "[b]oth [genes and environment] are necessary for development, neither is sufficient"[17], or else that genes "…have parity with other molecules as severally necessary and jointly sufficient conditions [to produce traits]"[18]. However, the thesis isn't always so bare – in fact, as Stegmann (2012) makes clear, **DST** advocates endorse (often implicitly) a wide variety of different (and occasionally overlapping) senses of 'causal parity', some with more conceptual adornments than the others.

I want to focus here on the form of the thesis that I think has had the most influence on and is therefore the most common in the literature – the form Stegmann (*ibid.*: 913) calls *informational parity*, which is the claim that "[g]enes and non-genetic factor[s] are on a par insofar as non-genetic factors will carry information on any viable account of information according to which genes carry information". There's a good reason that this form of the causal parity thesis is the primary and most powerful one operative in the literature: the *primacy* of the genome has historically been understood in terms of its being the unique carrier of *biological information* about the phenotype of an organism. Naturally then, putting other more conceptual critiques aside, if the thesis of causal parity is going to be empirically plausible, it must be shown that "containing information" about the phenotype of an organism isn't the sole purview of the genome.

And, on the most viable form of information theory, utilising 'Shannon information' (sometimes called 'causal information'[19]) - that *can* be shown. Putting the mathematical structure aside, according to Shannon (1948), something x is a *source* of information if it can occupy a number of alternative states, and something else y "carries information" about x if its state is correlated with the state of x. So, a variable x "contains information" about another variable y *iff* the state of y causally co-varies with the state of x, in such a way that changes in the value of x are reliably associated with changes in the value of y (and vice versa). Thus, one can "gain information" about the state of y by attending to the state of x, and vice versa[20]. If the genome is labelled as the 'transmitter' of information, proteins as the 'receivers' of that information, and the various cellular machinery as the 'channel' through which that information is passed, the genome "contains information" about the production of proteins: the state of the DNA bases of the genome causally co-varies with the state of the proteins. The problem is however that these labels can be re-organised and re-applied to *any* of the causal factors in this set-up, and *whichever* way the labels are designated, the same "informational" correlation can be found. If we choose to treat *the genome* as 'the channel', we find that RNA Polymerase, tRNA and transcription factors contain information – i.e. have a reliable, counterfactual pattern of the correlation of states – about protein production, just as the genome does[21]. As Maynard-Smith (2000: 189) points out, while it will be the true that the genome "contains information" for example, about the morphology of a child, "…we can equally well say that a baby's environment carries information about its growth; if it is malnourished, it will be underweight".

Given that it's the case that *any* of the causal factors that are necessary for the production of proteins can be treated as 'channels' against which variation in some particular causal factor can causally

---

[17] Oyama (2000: S340)
[18] Schaffner (1998: 234)
[19] Sterelny & Griffiths (1999: 101)
[20] Typically the "correlation" between the two states/entities/systems is cashed out in terms of reduction of uncertainty, or the decrease of epistemic entropy, in the sense that knowing the value of the second variable allows one to know the value of the first (or vice versa).
[21] Cf. Oyama (1985); Griffiths and Gray (1994)

co-vary with protein production, we are seemingly forced to conclude that the designation of any *particular* causal factor as *the* signal is not based on any *ontological* distinction; in other words, as Wheeler (2007) makes clear, having states that causally co-vary with the state of protein production is *not* the ontologically *unique* causal purview of the genome. If this is the case, then the criterion of "containing information" about the phenotype of an organism *cannot* single-out the genome as "first among equals", and the thesis of causal parity looks well-founded - for we apparently must admit either that nearly *every* causal factor is causally primary with respect to phenotypic traits, or else, what amounts to the same thing, that *no* causal factor is. Are we then forced to secede and agree with the advocates of **DST** that genes are "just one interactant in a field of context-dependent difference makers"[22] and that therefore "…*practical,* not theoretical, considerations direct the singling out of genes as [primary] causes"[23]?

3. *Responding to the Thesis of Causal Parity*

As Weber (*forthcoming*) points out, arguments from the defenders of the causal primacy of the genome generally fall into one of two categories: 'information-theoretic' responses, and 'causal selection' responses. Information-theoretic responses provide an addendum to the concept of 'information' in an attempt to show that *only* the genome "contains information" about phenotypic traits: causal co-variance of states is still a requirement for the informational relation, but some other requirement must be met as well. Some have been *representational* theories, where "containing information" about an effect entails containing *semantic content* about that effect, content that is typically taken to be imbued by evolutionary selection contingencies - these are the so-called 'teleosemantic' accounts of information[24]. There have also been a variety of *non-representational* theories, where the informational *content* of the genome is taken to be either "instructional"[25], or "semiotic" (of some form)[26]. Causal selection responses, on the other hand, instead of offering a novel form of *content* with which to buffer the concept of 'information', focus solely on the informational link of the causal co-variance of states, requiring that the values of a causally primary factor must causally co-vary with the values of phenotypic traits *in the right way*: either that co-variance must be of a very particular type[27], or else it must meet some degree of fine-grained specificity[28].

As one would expect, philosophers have objected to the sufficiency of both of these types of responses to provide an adequate account of the causal primacy of the genome. I will not here delve into the to-and-fro of these arguments for causal primacy, and for a principled reason: at the heart of these two types of responses lies what I take to be a fundamental mistake in the search for a theory of causal primacy. Notice that in the main responses to the problem of causal parity, the attempt to restore primacy has consisted in either *adding* something to the relation of the causal co-variance of states that forms the foundation of information theory, or else in *adding* something to that relation itself. However, I want to suggest that while the fact that a causal factor "contains information" about an effect – that is, that its state-values correlatively co-vary with the values of the effect - *is* important, it's not what's *most* important, and therefore focusing on that relation *isn't* going to lead one to an adequate theory of causal primacy.

---

[22] Gray (1992: 179)
[23] Gannett (1999: 356; my emphasis)
[24] Sterelny, Smith & Dickison (1996); Maynard Smith (2000); Shea (2007)
[25] Stegmann (2005)
[26] Sarkar (2003); Godfrey-Smith (2000)
[27] Stegmann's (2012) 'external ordering' is a possible example of this.
[28] Woodward (2010); Waters (2007) adds the additional requirement that a causally primary factor must be an 'actual difference maker' that is also *causally specific* in Woodward's sense.

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

Within causal set-ups, being a causally contributing factor that is capable of having multiple values, ones which correlatively co-vary with the value of the effect, is undoubtedly an important role: it is, as far as I'm concerned, a clear marker of a causal relationship, and it is by studying this co-variance that we are able to exploit these factors in experimental and then practical circumstances – and this is clearly of central importance to the advancement and utility of scientific research. However, although this causal role is important, and thus so are the set of causal factors who play this role within any particular set-up, there is another *type* of causal role that I believe is *more* important – namely, the one played by the causal factor that is *responsible for the fact that those other factors' values co-vary with the value of the effect*. Accordingly, it is my claim that if we want to elect a candidate for 'causal primacy', we need to draw a distinction between (a) the contribution of causal factors whose values are functionally correlated with the value of the effect and (b) the contribution of causal factors which are responsible for the existence of that functional value-correlation. I will refer to this as the distinction between (a) causal *relevance* and (b) causal *responsibility*, and my claim is that whatever is causally *responsible* in this sense ought to be considered causally *primary*.

In what follows, I show that understanding the division of causal labour between *responsible* and *relevant* factors with respect to some effect brings out an important point: the fact that a particular causal factor's value correlatively co-varies with the value of some effect is not *enough* to merit it the title of causal primacy. For as we will see, any *pro tanto* claim to that title that causally *relevant* factors might possess is one inherited from the existence and nature of causally *responsible* ones; this is because, in short, relevant factors causally depend upon responsible ones for their own relevancy. However, to get clearer on what *responsibility* in this sense amounts to, and why we ought to consider it amounting to causal *primacy*, I will offer an explication by display, by showing why a particular class of properties deserve the title of causal responsibility.

As the reader will no doubt have already guessed, given that I have claimed that it is dispositional properties whose existence establishes that there is a relation of 'causal influence' between two particular states (§1.1), I will contend that the realm of the *dispositional* is the realm of the *responsible*. In order to argue for this, I want to put forward two main indicators that point us toward which causal factor in a particular set-up is *causally responsible* for some effect – the test of 'counterfactual dependency' and of 'specificity determination'. Passing these tests is an at least minimally sufficient requirement for *responsibility*. Dispositional properties, as I will show, satisfy this requirement: for while it is true that the existence of a set of causal factors that compose a stimulus condition are *required for* a particular dispositional property's manifestation to come about, and that their particular values are *relevant to* precisely *how* that manifestation comes about, that group of causal factors, in and of themselves, are not *responsible* for the fact that they are *required* and *relevant* in those senses. *That* they *are*, I argue, is a fact established by the existence and nature of that dispositional property – and this is the mark of *responsibility*.

Consider the first indicator of causal responsibility, the test of 'counterfactual dependency'. Here we look for the factor which, were it to be absent, the functional value-correlation between a collection of causal factors and the effect would also be absent. Consider an (admittedly simplified, "toy") example causal event involving the manifestation of a dispositional property – the lighting of a struck match. Here, we could conceptualise the 'stimulus' of the match's disposition of 'flammability' as a collection of casual factors[29]: 'striking', 'oxygen levels', 'match-box surface roughness", etc. – all of these factors must be in

[29] Although most philosophers would undoubtedly classify some of the following 'stimulus' factors as "background conditions", I ignore that distinction here, given the preceding discussion of causal parity, showing that *any* causal factor can be "held constant" while manipulations on the others are correlated with changes in the effect. For other

place in order for the disposition to manifest 'ignition', and the varying of any of their values will have a fine-grained effect on how that manifestation comes about, from a minute amount of flame to a venerable bonfire atop the match-tip; e.g. less striking is correlated with slower flame development, more oxygen is correlated with a larger flame, etc. But is it the case that, were the match to *lack* the dispositional property of 'flammability', it would be the case that fine-grained changes in the values of the other causal factors – the 'striking', the 'oxygen', etc. – would be correlated with fine-grained changes in the value of the effect, 'ignition'? Clearly not. If the match were not 'flammable', altering the Oxygen levels, the exact stroke of the match-tip on the box, etc. would have absolutely *no* effect on *whether* and *how* the match becomes enflamed. This suggests that the *establishing* of the well-ordered, functional relationship between the various values of those factors with the various possible values of the effect is *dependent upon* the existence of the dispositional property – and this is precisely what is required for that property to be considered causally *responsible* for that effect.

A further indication that a particular causal factor is causally *responsible* for some effect is that it establishes not only *that there is* a functional value-correlation between other causal factors and the effect, but also *the precise character* of that functional relationship – that is, if it is the case that those other causal factors somehow *rely upon* that causal factor to be causally connected to the effect *in the specific fashion that they are*. Consider the effect of Salt on two different organisms – slugs and humans. We know that Salt can have significant effects on the functioning of both of these organisms. But consider the fact that *the exact same measurement* of Salt that might provide nourishment/replenishment to a human's body would have the devastating effect of dehydrating and subsequently killing the body of a slug. Indeed, the relation of the values of the same causal factor – Salt – to its effect has an entirely distinct character when in the context of a slug, rather than a human: *too* much Salt for a slug, is not *too* much Salt for a human, and the "safe" value-range of Salt differs in each. The fact that the same exact causal factor can be related to an entirely different effect in an entirely different way in two different organisms suggests, I think, that this factor can neither establish the existence of *nor establish the specific character of* its particular relationship of causal co-variance between themselves and some effect; and the same goes, *mutatis mutandis*, for the other non-dispositional factors. The specificity of that relationship, I claim, is a consequence of the particular character of a dispositional property, and in this particular case, the difference between slugs and humans is their possession of distinct dispositional properties, both having 'salt' as stimulus conditions.

What these two tests indicate is that while all of the other types of causal factors are *causally relevant* – their presence and values are important in determining whether and in what respect some effect will occur – it is dispositional properties which are *causally responsible* – they establish that there is a well-ordered, functional relationship between the various values of those factors and the values of that effect; without dispositional properties, not only would that functional relationship not exist, but it wouldn't exist in just the particular fashion that it does. And it is in just this respect that these properties deserve the title of causal primacy – for the correlative value co-variance of *relevant* factors with an effect that most analyses of causal primacy place primary importance upon is, as we have seen, itself *dependent upon* and *subject to* those properties, *via* their role as causally *responsible*. Thus, I claim that dispositional properties, *qua* causally responsible for their effects, ought to be considered as causally *primary* with respect to those effects as well.

---

arguments against the distinction between 'causes' and 'conditions' with respect to dispositional properties, see Mumford & Anjum (2011) and Hauska (2009).

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

3.1 *The Causal Responsibility of the Genome*

Armed with the conclusion that dispositional properties play an ontologically unique role within causal set-ups, one that sets them apart as "first among equals", and given that the genome realises these properties, in virtue of the fact that particular complexes of genes bear the relation of 'causal influence' to particular phenotypic traits (§1), a straightforward solution to the problem of causal parity in the genome presents itself. It's time to see what theoretical work the model of dispositional properties presented in the previous sections can do in a biological context.

Consider again the serious stumbling block for those who have attempted to defend the causal primacy of the genome - the phenomenon of phenotypic plasticity. That phenomenon shows that a particular genome is capable of producing a wide variety of quantitatively distinct values of a particular phenotypic trait in accordance with its exposure to a wide range of environmental conditions: with the genome as a constant "background condition/channel", we find that the values of environmental factors causally co-vary with the values of a particular phenotypic trait, and thus "contain information" about that trait – a causal role which was once assumed to be played uniquely by the genome. However, with the above discussion in mind, I contend that the relation of "containing information" is the *wrong* place to focus if we hope to find 'causal primacy' in the genome, and for a simple reason: although the contribution of causal factors whose values are functionally correlated with the value of the effect *is* important, the contribution of the causal factor which is responsible for the *existence and specification* of that functional value correlation is *more* so. According to that distinction, these environmental causal factors are designated as causally *relevant* to the production of a particular phenotypic trait, as it is their it is their values which appear to determine which specific determinate value of a particular phenotypic determinable occurs - but which factor is causally *responsible* for that effect? In other words, although the values of each of those causal factors are/could be functionally correlated with value of the effect, which among them are *responsible* for the very *existence* of that functional value-correlation? If, as I have argued, *responsibility* is the realm of the *dispositional*, then, as the genome realises dispositional properties, it too must be able to be shown to be causally *responsible*. And, by making use of the two aforementioned indicators for causal responsibility, I think this *can* be shown.

Consider the first indicator – the test of 'counterfactual dependency': is it the case that, were the genome to be *absent*, it would be the case that fine-grained changes in the RNA Polymerase and the amount/type/etc. of transcription factors would be correlated with fine-grained changes in the production of proteins, and hence of phenotypic traits? Clearly not. If a particular complex of genes were absent, no gathering of, or active co-operation of a complex of RNA Polymerase and transcription factors would have *any* effect on – let alone bear any fine-grained, 'causal influence' relationship to – the production of a particular phenotypic trait. Of course, this should not surprise us, as the template-based causal pathway from those causal factors to the production of proteins *requires* the mediating causal structure of the genome. That being the case, without the existence of the genome, no amount of "manipulations" on the complex of RNA Polymerase and transcription factors will have absolutely *any* effect on *whether* and *how* its associated proteins are produced. This suggests that the *establishing* of the well-ordered, functional relationship between the various values of RNA Polymerase and transcription factors with the various possible values of the particular reaction norm of a phenotypic trait is *dependent upon* the existence of the particular genome in question (or, more specifically, upon the particular complex of genes within that genome) – and, as we have seen, this is precisely what is required for the genome to be considered causally *responsible* for that trait.

That said, it has to be granted that, with respect to this first indicator, this 'responsibility' is deserved *because* the genome functions as an integral cog in the larger complex of the template-based

transcription/translation machinery at work within the cellular architecture, and further, that it *wouldn't* be 'responsible' *independently* of its place therein - after all, many other, extra-genetic factors of that system must be in play in order for the genome to function in this way. Given that the fact that the genome passes this "test" of counterfactual dependency *itself* counterfactually depends upon it being a proper part of a larger system, ought we to conclude that it is the system itself, rather than a mere part of it, that deserves the appellation of responsibility? I think not. For note that, *whenever* we're looking for a causally responsible factor, we're looking *within* a system - an 'informational' system, to be specific. It is therefore likely (if not necessary) that *any* factor within that system isn't going to function in the same way, if at all, outside of the context of that system – but this certainly doesn't entail that there aren't distinct and discernible causal roles played by individual components of that system[30], or that those roles aren't therefore the roles *of* those components. So while it's true that the genome couldn't play the role it does outside of the entire complex of extra-genetic transcription/translation cellular machinery, it is nevertheless true not only that *it* is the casual factor that *does* play that role, and that role – of being the functional mediator of the template-based causal pathway from particular causal factors to the production of proteins (and thus, of phenotypic traits) – is not only one at the very "causal centre" of that system, but is importantly *the* one we're interested in with respect to 'causal responsibility'.

Consider next then the indicator of 'specificity determination': is it the case that the nature of the *other*, *extra-genetic* factors determines the specific character of the relationship of co-variance between themselves and the values of the reaction norm of a particular phenotypic trait? Here we must be careful, and heed the distinction between *relevance* and *responsibility*. As we have seen, it's certainly true that the presence or absence of particular transcription factors is associated with the exact rate, amount, and timing of protein production, and thus, these factors are in "causal control" of picking-out a specific outcome from a particular reaction norm of a phenotypic trait. But although this causal control *does* amount to *specifying* which value is "chosen" from the possible values of a trait's complete reaction norm, it *doesn't* amount to *specifying* the particular relationship of causal co-variance between some set of causal factors and the values that make-up that reaction norm. In other words, while intra-cellular causal factors, like transcription factors, play an important role of determining *which* value of that reaction norm comes about, they do not, in and of themselves, outside of the mediation of a particular complex of genes within a genome, determine *which* values of those transcription factors are associated with *which* value of that reaction norm – and that latter role is precisely what is at issue when we perform the test of 'specificity determination'.

Indeed, just as in our previous example of the relation of 'salt' to slugs and humans, it's clear that no complex of transcription factors, in and of themselves, are *responsible* for the fact that they are related to a particular phenotypic trait *in the specific fashion that they are*, for one and the same transcription factor can function as an 'enhancer' of the rate of transcription in the context of one gene, and as a 'repressor' in the context of another[31]. This suggests that the *particular character* of any specific functional relationship of value co-variance between some set of transcription factors and the production of a particular phenotypic trait is established by the character of a particular *complex of genes within a particular genome*. And all of this suggests that it is the genome, *not* "environmental" factors (transcription factors, etc.), that passes the test for 'specificity determination', and thus, the test for establishing causal *responsibility*.

---

[30] If the fact that a causal factor couldn't play its purported causal role outside its specific, proper context disqualified it from playing that role, which causal factor (especially in the biological realm) could be said to perform *any* causal role whatsoever?

[31] Hollenhorst, et. al. (2009)

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

So it is my contention that while all of causal factors that make-up the intra-cellular "environment" of a genome – RNA Polymerase, a complex of transcription factors, etc. – are *causally relevant*, in that their presence and values are important in determining whether and in what respect the production of protein occurs, it is the *genome* which is *causally responsible*, as particular complexes of genes establish that there is a well-ordered functional relationship between the various values of those factors and the values of protein production, and hence the values of the reaction norm of a particular phenotypic trait. Without the genome, not only would that functional relationship not exist, but it wouldn't exist in just the particular fashion that it does. Accordingly, because the genome is absolutely foundational to bringing about/determining the character of phenotypic traits via its role as causally *responsible* for those traits, I suggest that it is also *causally primary* with respect to those traits.

4. *Parity and Pragmatism Revisited*

If, as I have argued, the genome is to be crowned with causal primacy with respect to the phenotypic traits, then it must be so as a matter of *ontology*, and not by mere conventional *fiat*, lest the charge of parity and pragmatism remain. In other words, if the genome truly deserves the honour of causal primacy, it had better not be the case that "environmental" causal factors can just as equally play the role of 'causal responsibility' with respect to phenotypic traits.

I have claimed that the causal factor that is *responsible* for some effect is the one that establishes the mediating functional relationship of co-variance between that effect and a set of other causal factors. But is the role of 'causal responsibility' one that *any* of the causal factors in a causal set-up could play? Couldn't we simply choose to "hold constant" *any* causal factor, and by varying the rest of them, still obtain the same functional relationship among those factors and the effect? Consider again the phenomenon of informational parity: when the genome is placed in the 'signal' position – the position according to which it "contains information" about a specific trait – the environment functions as the static, "background" channel, and manipulations in the value of the genome (for instance, *via* substituting nucleotide bases) correlatively co-vary with changes in the value of the phenotypic trait (that is, which particular value of its reaction norm comes about). In this case, the pragmatist will ask, isn't the environment playing the role of 'causal responsibility', in that *it* is the causal factor which is establishing the existence of a functional value-correlation between values of the genome and values of the phenotypic trait? Given informational parity then, isn't it the case that 'responsibility' is just as ubiquitous as 'information', and hence, that the designation of any *particular* causal factor as *primary* is more a reflection of *choice* than it is *ontology*?

In order for the pragmatist to be able to show that, in light of 'informational parity', there also exists a parity of *responsibility* among genetic and non-genetic factors, she must be able to *show* that non-genetic factors can *perform* the role of responsibility in a robust fashion. Showing *that* isn't trivial, and accordingly, there are two important points to keep in mind here. Firstly, the mere conceptual shuffling of positions within a relational equation does not necessarily represent an *ontologically* unique state of affairs: if the *causal* relationship among the variables is unchanged in the novel expression of the relation, then so are the *roles* played by those variables. Secondly, there is more to the role of being causally responsible for an effect than simply being the causal factor that is "held constant" while the others' values correlatively co-vary with the value of that effect: the role of responsibility is a *dynamic* one, and so isn't played by a causal factor in virtue of it being in the conceptual position of background condition/channel with respect to some effect.

With that those two caveats in mind, what has to be said is that it's certainly true that, given 'informational parity', it's the case both that the values of the genome and of the (extra- and intra-cellular)

This is a pre-print version of *The Dispositional Genome: Primus Inter Pares, Biology and Philosophy (2015).* The final publication is available at Springer *via* http://dx.doi.org/10.1007/s10539-014-9454-y

environment are capable of causally co-varying with value of a particular phenotypic trait. For the purposes of making a judgment regarding the 'parity of responsibility' however, the pertinent question is, with respect to each case, *why* changes in the former are causally correlated with changes in the latter. Take the problem case for the defender of the causal primacy of the genome via its role as causally *responsible* for a phenotypic trait – the case of the genome "containing information" about a phenotypic trait in virtue of changes in its values correlatively co-varying with changes in the phenotypic trait, where environmental factors appear to be playing the casually *responsible* role[32]. But are environmental factors *really* playing the role of causal responsibility here? In order to find out, we must ask, in these cases, *why* – causally - do we obtain differing values of the phenotypic trait when we alter the value of the genome? I contend that we do so because *altering the genome amounts to altering the functional value-correlative relationship between the "stable" set of environmental factors and the phenotypic trait*. If this analysis is correct, it shows that, contrary to the pragmatist's supposition, the genome is *still* playing the role of responsibility.

When we have a stable set of environmental conditions, **E**, and we observe that varying the value of the genome, **G**, results in varying values of the phenotypic trait, **P**, what we are observing, I claim, is a change in the relation of **E** to **P**, brought about by the change in the value of **G**. For although there is a correlation between the various values of **G** ($\mathbf{G}_1$, $\mathbf{G}_2$, $\mathbf{G}_3$,…) with various values (of the reaction norm) of **P** ($\mathbf{P}_1$, $\mathbf{P}_2$, $\mathbf{P}_3$,…), what is happening, *causally*, when we obtain a unique value of **P** is that when the value of **G** changes, so too does the functional relationship between the "stable" value of **E** with the value of **P**. So although it's true that, for instance, with respect to a "stable" background condition $\mathbf{E}_1$, a change from $\mathbf{G}_1$ to $\mathbf{G}_3$ is correlated with a change from $\mathbf{P}_2$ to $\mathbf{P}_4$, the reason why a change in the value of **P** occurs upon a change in the value of **G** is that the existence of $\mathbf{G}_1$ brings it about that $\mathbf{E}_1$ is causally correlated with $\mathbf{P}_2$, and the existence of $\mathbf{G}_2$ brings it about that $\mathbf{E}_1$ is causally correlated with $\mathbf{P}_4$. In other words, changes in the value of **G** are causally correlated with changes in the value of **P** *because changing **G** amounts to changing the way the value of **E** relates to the value of **P***.

If the above understanding is correct, then an important point follows: although by treating it as the 'channel' of an informational link it may at first appear that the "environment" can also be a factor that is causally responsible for a particular phenotypic trait, when we examine the *causal* relations at play, we find that, even in this case, the genome is *still* playing the characteristic role of causal responsibility, establishing the existence and the particular character of a functional relationship between the values of the environmental factors and the trait – for changes in that particular complex of genes are responsible for changes in that functional value-correlation (even if that change in value-correlation amounts to changing the way in which a single, "stable" value of the former relates to the values of the latter).

The natural response from the proponent of **DST** will no doubt be that the same thing can be said, *mutatis mutandis*, for the "environment" - she will say that if we simply switch **E** for **G** in the above example, *via* parity of reasoning, the same argument can be ran, with the conclusion that it is the "environment" that is causally responsible in the required sense. But is that really the case? Consider the final assertion of that example, with the appropriate amendments : "changes in the value of **E** are causally correlated with changes in the value of **P** *because* changing **E** amounts to changing the way the value of **G** relates to the value of **P**". In order for this to be true – that is, if the 'because' is appropriately *causal* here, as it must be -, it would have to be the case that environmental factors *establish* a functional value-correlative relation between a particular complex of genes within a specific genotype and a corresponding

[32] It's worth noting that although the following argument addresses only the central case of informational parity with respect to the environment and the genome, I think it can be applied, *mutatis mutandis*, to any other non-genetic causal factor one proposes to play the role of causal responsibility.

phenotypic trait[33]. Note however, that if *that* claim is true, then so must *this* one be: "changes in the value of **G** are correlated with changes in the value of **P** because of the existence of a functional value-correlative relationship between **G** and **P**, *established by* ***E***". That claim strikes me, as I suspect it will many, as simply implausible: it isn't the causal purview of the "environment" to act as the architect and facilitator of the functional value-correlation between extra-environmental factors (read: the genome) and phenotypic traits. And importantly, this is precisely what 'responsibility' requires: the fact that when **E** is a "background"/channel condition, there exists a correlative relation between **G**-values and **P**-values is, although true, *not enough* to merit **E** the title of causally *responsible* – it must be somehow shown that the existence of **E** establishes, and that the nature of **E** specifies, that relation. That said, the onus is decidedly upon the defender of causal parity to show just that, and in a fashion that mirrors the plausibility and theoretical fit of the account given in §3. This is a burden I think defenders of the thesis of causal parity will find rather heavy.

In the absence of any such account, and given the above arguments, I contend that though it *is* true that the genome "contains information" about phenotypic traits in just the same way that the environment does, and accordingly that either causal factor can be "held constant" and treated as a background condition/channel, it *isn't* true that, when treated thusly, these two causal factors *operate in the same fashion*, or that they *play the same causal roles* with respect to the specified production of phenotypic traits; in other words, their symmetry with respect to functioning as 'informational channels' isn't one that is reflected in the ontology of the causal structure of their relationship to those traits. Contrary to the rebuttal of the pragmatist then, I claim that there is no 'parity of responsibility' among genetic and non-genetic factors and furthermore, that lack of parity is not a matter of mere heuristic convention, but is instead a consequence of ontology.

Summing Up

If there is a stable, reliable and repeatable causal pathway from extra- and intra-cellular "cues" to phenotypic traits such that the former bears an "informational link" to the latter *via* a relation of 'causal influence', then the genome realises dispositional properties that are "directed toward" those traits. I have argued that the unique role of dispositional properties of being the causal factor that establishes the existence of a functional relationship of causal, correlative value co-variance between a former state and that "directed toward" state singles it out as "first among equals" among its causal peers with respect to that state. Thus, contrary to the causal parity thesis endorsed by the proponents of **DST**, I claim that the genome, on account of its ontology, and not as mere artefact of any conceptual schematic, occupies a privileged, *primary* position with respect to the formation of phenotypic traits.

[33] Cf. §3

Bibliography

Armstrong, D. (2004). *Truth and Truthmakers.* Cambridge: Cambridge University Press.

Gannett, L. (1999). What's in a Cause?: The Pragmatic Dimensions of Genetic Explanations. *Biology and Philosophy*, 349 - 373.

Gibson, J. (1970). Effects of Temperature on Selection for Scutellar Bristles. *Nature*, 591 - 607.

Godfrey-Smith, P. (2000). On the Theoretical Role of "Genetic Coding". *Philosophy of Science*, 26-44.

Gray, R. (1992). Death of the Gene: Developmental Systems Strike Back. In P. Griffiths (Ed.), *Trees of Life: Essays in the Philosophy of Biology* (pp. 165 - 209). Kluwer Academic Publishers.

Griffiths, P., & Gray, R. (1994). Developmental Systems and Evolutionary Explanation. *Journal of Philosophy*, 277 - 304.

Griffiths, P., & Knight, R. (1998). What Is the Developmentalist Challenge? *Philosophy of Science*, 253-258.

Hauska, J. (2009). Dispositions Unmasked. *Theoria*, 304-335.

Hollenhorst, P., Chandler, K., Poulsen, R., Johnson, W., Speck, N., & Graves, B. (2009). DNA Specificity Determinants Associate with Distinct Transcription Factor Functions. *PLoS Genetics, 5*(12), 1 - 12.

Jacobs, J. (2011). Powerful Qualities, Not Pure Powers. *The Monist*, 81-102.

Lewis, D. (2000). Causation as Influence. *The Journal of Philosophy*, 182-197.

Lewis, D. (2001). *Counterfactuals.* Oxford: Blackwell Publishers Inc.

Lewontin, R. (1982). *Human Diversity.* New York: Scientific American Books.

Manley, D., & Wasserman, R. (2008). On Linking Dispositions and Conditionals. *Mind*, 59-84.

Martin, C. (2008). *The Mind in Nature.* Oxford: Oxford University Press.

Maynard Smith, J. (2000). The Concept of Informatio in Biology. *Philosophy of Science*, 177-194.

Mumford, S. (2004). *Laws in Nature.* London: Routledge.

Mumford, S., & Anjum, R. (2011). *Getting Causes From Powers.* Oxford: Oxford University Press.

Oyama, S. (1985). *The Ontogeny of Information.* Cambridge: Cambridge University Press.

Oyama, S. (2000). Causal Democracy and Causal Contributions in Developmental Systems Theory. *Philosophy of Science (Proceedings)* (pp. S332-S347). Chicago: University of Chicago Press.

Powell, A. M., Davis, M., & Powell, J. R. (2010). Phenotypic Plasticity Across 50 MY of Evolution: Drosophila Wing Size and Temperature. *Journal of Insect Physiology*, 380-382.

Sarkar, S. (2003). Genes Encode Information for Phenotypic Traits. In C. Hitchcock (Ed.), *Contemporary Debates in Philosophy of Scienc* (pp. 259 - 272). London: Blackwell.

Schaffner, K. (1998). Genes, Behavior, and Developmental Emergentism: One Process, Indivisible? *Philosophy of Science*, 209 - 252.

Schlichting, C., & Smith, H. (2002). Phenotypic Plasticity: Linking Molecular Mechanisms with Evolutionary Outcomes. *Evolutionary Ecology*, 189-211.

Shannon, C. (1948). A Mathematical Theory of Communication. *Bell Systems Technical Journal*, 379-423, 623-656.

Shea, N. (2007). Representation in the Genome and in Inheritance Systems. *Biology and Philosophy*, 313-331.

Stalnaker, R. (1968). A Theory of Conditionals. *Studies in Logical Theory, American Philosophical Quarterly Monograph Series*, pp. 98-112.

Stegmann, U. (2005). Genetic Information as Intstructional Content. *Philosophy of Science*, 425 - 443.

Stegmann, U. (2012). Causal Control and Genetic Causation. *Nous*, 1 - 18.

Stegmann, U. E. (2012). Varieties of Parity. *Biology & Philosophy*, 903-918.

Sterelny, K., & Griffiths, P. (1999). *Sex and Death: An Introduction to Philosophy of Biology.* Chicago: University of Chicago Press.

Sterelny, K., Smith, K., & Dickison, M. (1996). The Extended Replicator. *Biology and Philosophy*, 377 - 403.

Vetter, B. (2013). Multi-Track Dispositions. *The Philosophical Quarterly*, 330-352.

Waters, C. K. (2007). Causes That Make a Difference. *Journal of Philosophy*, 551 - 579.

Weber, M. (forthcoming). Causal Selection vs Causal Parity in Biology: Relevant Counterfactuals and Biologically Normal Interventions. *Minnesota Studies in Philosophy of Science*.

Wheeler, M. (2007). Traits, Genes, and Coding. In M. Matthen, & C. Stephens (Eds.), *Philosophy of Biology* (pp. 369 - 402). Elsevier.

Whitman, D. W., & Agrawal, A. A. (2009). What is Phenotypic Plasiticty and Why Is It Important? In T. N. Ananthakrishna, & D. W. Whitman (Eds.), *Phenotypic Plasticity of Insects: Mechanisms and Consequences* (pp. 1-63). Enfield: Science Publishers.

Woodward, J. (2010). Causation in Biology: Stability, Specificity, and the Choice of Levels of Explanation. *Biology and Philosophy*, 287-318.