



# What's all the fuss about? The inheritance of acquired traits is compatible with the Central Dogma

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**Abstract** The Central Dogma of molecular biology, which holds that DNA makes protein and not the other way around, is as influential as it is controversial. Some believe the Dogma has outlived its usefulness, either because it fails to fully capture the ins-and-outs of protein synthesis (Griffiths and Stotz in *Genetics and philosophy* Cambridge introductions to philosophy and biology, Cambridge University Press, Cambridge, 2013; Stotz in *Hist Philos Life Sci* 28(4):533–548, 2006), because it turns on a confused notion of information (Sarkar in *Molecular models of life*, MIT Press, Cambridge, 2004), or because it problematically assumes the unidirectional flow of information from DNA to protein (Gottlieb, in: Oyama, Griffiths, Gray (eds), *Cycles of contingency: developmental systems and evolution*, MIT Press, Cambridge, 2001). This paper evaluates an underexplored defense of the Dogma, which relies on the assumption that the Dogma and the Inheritance of Acquired Traits, a principle which dates as far back as Jean Baptiste-Lamarck, are incompatible principles (Smith in *The theory of evolution*, Cambridge University Press, Cambridge, 1993; Judson in *The eighth day of creation*, Jonathan Cape, London, 1979; Dawkins in *The extended phenotype*, Oxford University Press, Oxford, 1970; Cobb in *PLoS Biol* 15(9):e2003243, 2017. <https://doi.org/10.1371/journal.pbio.2003243>; Wilkins in *BioEssays* 24(10):960–973, 2002. <https://doi.org/10.1002/bies.10167>; Graur *The fallacious commingling of two unrelated hypotheses: ‘the central dogma’ and ‘dna makes rna makes protein’*. *Judge Starling.*, 2018. <http://judgestarling.tumblr.com/post/177554581856/the-fallacious-commingling-of-two-unrelated>). By appealing to empirical evidence in molecular science, I argue that this apparent incompatibility is indeed merely apparent. I conclude by briefly demonstrating how these considerations bear on the topic of conceptual pluralism in the philosophy of science (Stencel

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and Proszewska in *Found Sci* 23(4):603–620, 2018. <https://doi.org/10.1007/s10699-017-9543-x>; Lu and Bourrat in *Br J Philos Sci* 69(3):775–800, 2018. <https://doi.org/10.1093/bjps/axx019>).

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## 1 Introduction

The Central Dogma of molecular biology, which says that DNA makes protein and not the other way around, is as influential as it is controversial. Some believe the Dogma has outlived its usefulness, either because it fails to fully capture the ins-and-outs of protein synthesis (Griffiths and Stotz 2013; Stotz 2006; Oyama 2000, 2009), or because it turns on a confused notion of information (Sarkar 2004), or because it problematically assumes the unidirectional flow of information from DNA to protein (Gottlieb 2001).

Despite the wide-spread criticism, the Central Dogma continues to be heralded as a fundamental principle in biology (Weber 2006; Rosenberg 2006; Graur 2018). One underexplored reason, in particular, is that the Central Dogma has been perceived as implying denial of Lamarckian inheritance, or something close to it (Smith 1993; Judson 1979; Dawkins 1970; Cobb 2017; Wilkins 2002; Graur 2018). Cobb (2017) for instance claims that “the Central Dogma supported the neo-Darwinian view that it was impossible for any character that was acquired during an organism’s life to affect its hereditary characters.” Similarly, Smith (1993) notes that “the greatest virtue of the central dogma is that it makes clear what a Lamarckist must do—he must disprove the dogma.” Ernst Mayr, in an interview with Wilkins (2002), claimed that the Central Dogma secured “the final nail in the coffin of the inheritance of acquired characteristics.” These quotes suggest that a great deal of the Dogma’s influence has been tied to its perceived incompatibility with the heritability of acquired traits; if we deny the latter, then the former must be true.<sup>1</sup>

My aim is to complicate the supposed connection between the Central Dogma and the inheritance of acquired traits.<sup>2</sup> By drawing from recent discoveries in molecular science, I evaluate whether the inheritance of acquired traits implies a rejection of the Central Dogma—and if so, of what interpretation.<sup>3</sup>

<sup>1</sup> On the flipside, if the Dogma is false, then the heritability of acquired traits must be true.

<sup>2</sup> There is a historical question about the extent to which the Inheritance of Acquired Traits is deeming of the term “Lamarckian.” As some commentators have noted, the Inheritance of Acquired Traits was advanced long before Jean Baptiste Lamarck ever propounded the principle (Burkhardt 2013). Given that my aim is to investigate the connection between the Central Dogma and the Inheritance of Acquired Traits, these issues will be side-stepped in this paper.

<sup>3</sup> It should be noted that many have endorsed the heritability of acquired traits, in some form or other (Gissis 2011; Jablonka and Lamb 2014; Oyama 2003; Wilschut et al. 2016). For more on the heritability of acquired traits, from a contemporary philosophical perspective, see Koonin (2019), Woolley et al. (2019), Wideman et al. (2019), and Jablonka (2019).

Before outlining the structure of this paper, one major point is in order. It should be noted that, in this paper, I set aside cases of cultural inheritance. There is no question, of course, that the Central Dogma is entirely consistent with the existence of culturally inherited traits (Godfrey-Smith 2009). The question at stake, rather, is whether the Central Dogma rules out the inheritance of acquired *phenotypic* traits.

This paper is organized as follows. In Sect. 2, I note that while the Central Dogma is often treated as a unified thesis, its interpretations in the philosophical and biological literature abound. I therefore restrict my focus to three major interpretations, before delving into a contemporary interpretation of the Inheritance of Acquired Traits. Then, I revisit an underexplored defense of the Central Dogma grounded in the incompatibility between the Dogma and the Inheritance of Acquired Traits. After briefly exploring the rationale for this supposed tension, I analyze whether this incompatibility is justified. To do this, I consider each interpretation of the Dogma and evaluate whether they are *truly* incompatible with instances of the Inheritance of Acquired Traits. By appealing to empirical evidence in molecular science, it is shown that the two theses are compatible and that, contrary to Mayr's assertion, the Dogma *is not* "the final nail in the coffin of the inheritance of acquired characteristics." I conclude by briefly demonstrating how these considerations bear on the topic of conceptual pluralism in the philosophy of science (Stencel and Proszewska 2018; Lu and Bourrat 2018).

## 2 The Central Dogma and the Inheritance of Acquired Characteristics

Since its original formulation by Francis Crick in 1958, much has been said about the Central Dogma of molecular biology along with its connection to the Inheritance of Acquired Traits. The problem, however, is that many interpretations of the Dogma *and* the heritability of acquired traits have been advanced in the literature (as we will see), making it difficult to pin down precisely how the two principles relate to each other. The goal in this section, therefore, is to make explicit precisely how these concepts are to be understood in this paper, beginning with the Central Dogma.

### 2.1 Some Central Dogmas of Molecular Biology

As some commentators have pointed out, the Central Dogma admits of many interpretations (Camacho 2019). Classically, perhaps, the Central Dogma may be construed as saying that DNA makes protein (Watson 1965) and not the other way around (Crick 1958, 1970), but other interpretations have digressed from this classic reading (Crick 1958, 1970; Rosenberg 2006; Weber 2006; Sarkar 2004; Watson 1965; Sustar 2007; Griffiths and Stotz 2013; Stotz 2006). Indeed, there are three main variants of the Central Dogma that appear in the literature: the causal interpretation, the informational interpretation and the negative interpretation. (These variants can be further subdivided, but considering these further subdivisions won't matter here, since their details are not pertinent to the arguments that follow.)

This paper considers each of these three variants, all of which have figured recently in the philosophical and scientific literature (Graur 2018; Sarkar 2004; Rosenberg 2006; Weber 2006; Griffiths 2017; Stotz 2006; Sustar 2007). Stotz (2006) and Griffiths and Stotz (2013), for example, have repeatedly criticized an interpretation advanced by Rosenberg (2006) and Weber (2006), according to which DNA is the most significant causal factor involved in protein synthesis.<sup>4</sup> In contrast, Graur (2018) argues that what the Dogma is *really* trying to say is that proteins cannot affect amino acids sequences or nucleotide sequences (Crick 1958, 1970; Graur 2018).<sup>5</sup> Finally, Sarkar (2004) considers a notion of the Dogma that draws on the concept of information, according to which DNA carries information about protein. Since all of these interpretations will be considered in this paper, it is worth fleshing them out in a bit more detail.

Let's begin by offering a sketch of the *Gene-centric Dogma*, which roughly holds that genes make the biggest difference to protein synthesis. This positive interpretation supports a kind of gene-centrism, and has been advanced by the likes of Rosenberg (2006), Weber (2006) and Waters (2007):

Genes and DNA can be a specific difference maker in the sense that many specific difference in the sequence of nucleotides in DNA result in specific differences in RNA molecules. This is not the case with many other actual difference makers, such as polymerases, which are more like on/off switches. (Rosenberg, p. 561)

This interpretation holds that DNA is the most significant cause contributing to protein synthesis, because DNA constitutes a causally-specific, actual, difference-maker (or SAD, which is short for Specific, Actual, Difference-Maker). Let us call this interpretation the *Genecentric Dogma*. To better characterize this interpretation, let us first consider what it means for some factor to count as a *difference-maker* by appealing to James Woodward's interventionist account of causal explanation.<sup>6</sup> According to Woodward (2005), *X causes Y* if a change in the Y-value, from say  $y^1$  to  $y^2$ , is prompted by manipulation of the X-value, from say  $x^1$  to  $x^2$ . Now, to determine whether such a cause constitutes an *actual* difference-maker, we must identify *actual* effects as well as the *actual* causes that brought them about. To further elaborate the notion of an *actual difference-maker*, consider the following four conditions exposed by Waters (2007). For Waters, some causal factor, X, counts as an *actual difference maker* iff...

- i. X causes Y.
- ii. The value of Y actually varies among individuals in a given population.p.
- iii. The relationship X causes Y is invariant over at least parts of the space(s) of values

<sup>4</sup> I should note that this reading of the Dogma bears a strong resemblance to an interpretation attributed to Watson (1965), which holds that "DNA makes RNA makes protein."

<sup>5</sup> This interpretation, too, has been criticized in the literature: see Camacho (2019).

<sup>6</sup> For more on interventionism, see Woodward (2005), von Wright (2004), Pearl (2000) and Lewis (2000).

that other variables actually take in  $p$ . (In other words, it is invariant with respect to a portion of the combinations of values the variables actually take in  $p$ )  
 iv. Actual variation in the value of  $X$  partially accounts for the actual variation of  $Y$  values in population  $p$  (via the relationship  $X$  causes  $Y$ ). (Waters, p. 567)

Now, let's sketch what it means for an actual difference-maker to be *causally specific*. An actual difference maker is *causally specific* if manipulating the causal variable makes for specific differences in the effect variable. Now that we have a grip on what it means for something to count as a *causally-specific, actual, difference-maker* (or SAD for short), let's discuss how one might appeal to this notion to support the *Gene-centric Dogma*.

First, DNA *makes a difference* to protein synthesis, since the manipulation of nucleotides (i.e. A, C, T and G) in DNA sequences makes a difference to the codon that is expressed in the amino acid sequence. Next, DNA *actually* makes a difference to the *actual* effect in question, since *actual* differences in amino acid sequences are accounted for, in part, by *actual* changes to nucleotides in the DNA template. In other words, the actual differences in codons like Methionine (i.e. ATG) are accounted for by differences in nucleotide sequences in DNA, namely, the sequences containing nucleotides Adenine (A), Thymine (T), and Guanine (G). Lastly, DNA is *casually-specific* since very specific manipulations to the DNA template result in very specific changes to the codon expressed in the amino acid sequence. Again, manipulating nucleobases in DNA makes for very specific differences to the codon expressed in the amino acid, and this is made evident by the fact that a single nucleotide change in nucleobases that could constitute Methionine (ATG), could give rise to a completely different codon in the peptide sequence, namely, Isoleucine (i.e. ATT, ATC, ATA). On these grounds, we might say that DNA is a SAD and, therefore, the most significant cause involved in protein synthesis, unlike transcription factors like RNA Polymerase.<sup>7</sup>

The next interpretation—hereafter called the *Informational Dogma*—makes appeal to the notion of information (Maynard Smith 2000; Sarkar 2004; Godfrey-Smith 2000; Sustar 2007). Maynard Smith (2000), who draws from Dretske (1981), cashes out the account as follows. Suppose the occurrence of rain (R) is correlated with a particular type of cloud (C). In this case, we might say that C carries information about R. Similarly, the *Informational Dogma* says that a particular codon in a protein chain like Leucine is correlated with a particular gene sequence, which

<sup>7</sup> The *Gene-centric Dogma* has received attention from commentators like Rosenberg (2006), who contests the apparent incompatibility between the concept of epigenetic inheritance and the *Gene-centric Dogma*. To be sure, the concept of epigenetic inheritance, as Rosenberg understands it, applies to cases of phenotypic inheritance that does not involve changes in gene sequences; so it is different from the inheritance of acquired traits as I define it in this paper. In what follows, I focus on this interpretation because I want to extend Rosenberg's treatment, by demonstrating that the *Gene-centric Dogma* is compatible with the inheritance of acquired traits.

in this case may be either CUU, CUC, CUA or CUG. Given this, DNA alone carries information about protein, because gene segments like those above alone carry information about the resulting codon in peptide sequences.<sup>8</sup>

At this point, however, it may be objected that the Central Dogma, as Francis Crick envisioned it in 1958, bears little resemblance to the *Gene-centric Dogma* or the *Informational Dogma*. In fact, one might point out that the two Dogmas above bear a closer resemblance to the Sequence Hypothesis, a *positive statement* which, for Crick, says that “the transfer of information from nucleic acid to nucleic acid, or from nucleic acid to protein” is possible (Crick 1970). By contrast, one might think that the Central Dogma *really* holds that the “transfer from protein to protein, or from protein to nucleic acid is impossible.” For Crick (1970), the Central Dogma...

is not the same, as is commonly assumed, as the sequence hypothesis... In particular the sequence hypothesis was a positive statement, saying that the (overall) transfer nucleic acid-protein did exist, whereas the central dogma was a negative statement, saying that transfers from protein did not exist. (p. 562)

The Central Dogma, for Crick, then is a *negative statement*, which holds that the transfer of information from protein to protein and from protein to DNA is not possible. Information is here understood as “the *precise* determination of sequence, either in bases of the nucleic acid or of amino acid residues in the protein” (Crick 1958). In other words, for Crick, the Dogma says that proteins cannot alter nucleic acids (in RNA or DNA) or amino acids in proteins. This interpretation—hereafter referred to as *Crick’s Dogma*—for instance holds that proteins such as RNA polymerase cannot determine nucleic acid sequences, which are made up of nucleobases Adenine (A), Cytosine (C), Thymine (T) and Guanine (G) in DNA. What’s more, *Crick’s Dogma* holds that proteins cannot alter amino acid sequences, which are made up of codons, in other proteins. This interpretation, again, constitutes a negative thesis, as it merely states what proteins cannot do, and may therefore be immune to many criticisms that befall positive interpretations of the Central Dogma.<sup>9</sup>

Now that we have a better understanding of how interpretations of the Central Dogma are to be understood in the context of this paper, it would help to get clear on what the heritability of acquired traits stipulates. Doing so will help us understand why the Dogma and so-called Lamarckian inheritance are seen as by many as incompatible theses.

<sup>8</sup> Maynard Smith (2000) makes an additional point about intentionality, but—as we will soon see in the analysis that follows—it will not be necessary to engage in this further for the purposes of this paper.

<sup>9</sup> Many commentators have pointed out that the Central Dogma and the Sequence Hypothesis have been confused with each other (Camacho 2019; Graur 2018). Given this, I want to avoid this confusion by evaluating Crick’s original formulation of the principle, in addition to the *Gene-centric Dogma* and the *Informational Dogma* sketched above.

## 2.2 The Inheritance of Acquired Traits

Like the Central Dogma, The Inheritance of Acquired Traits—which generally says that organisms acquire heritable traits—admits of numerous interpretations and is therefore subject to many misunderstandings (Loison 2011; Loison and Herring 2017; Burkhardt 2013; Bowler 1979, 1988, 1992).<sup>10</sup> Since the aim of this paper is to better grasp the connection between the Central Dogma and the Inheritance of Acquired Traits, we must arrive at a precise interpretation of the latter before evaluating whether the two concepts are incompatible.

We should begin by issuing a distinction between phenotypic traits acquired through genetic transmission from traits acquired through cultural learning. Beginning with the latter, let us imagine a hypothetical scenario in which a child is born into a community of farmers.<sup>11</sup> Suppose also that the child's parents hold beliefs and values reflective of the farming community, and that the child not only comes to embrace the same beliefs and values about farming through the direct influence of her parents and community, but acts in accordance with these beliefs and values. We might say that the behavioral traits are passed on from parents to offspring. Let us further imagine that, once the child becomes an adult and bears children, these cultural beliefs and values are then passed onto their offspring in much the same way the child initially acquired these skills. It is in this sense, then, that the child inherited traits through learning. So, we might formulate an interpretation of the Inheritance of Acquired Traits that embraces heritable traits acquired genetically, such as the color of one's eyes, and heritable traits acquired culturally, as in the example above. We'll call this the *Broad Interpretation* of the Inheritance of Acquired Traits.

However, if the interpretation of the Inheritance of Acquired Traits were to embrace traits acquired through cultural learning as in the *Broad Interpretation* above, it would become immediately obvious that there is no tight-knit connection between the Inheritance of Acquired Traits and the Central Dogma. There is nothing about *Crick's Dogma*, the *Informational Dogma*, or the *Genecentric Dogma* that renders the *Broad Interpretation*—one that accounts for the cultural transmission of cultural traits—problematic: the Dogma's offered here simply do not speak to whether children inherit (some of) their beliefs and values through cultural learning. Importantly, this holds for many different forms of cultural learning (Boyd and Richerson 2005; Sterelny 2012; Heyes 2012).

Given the apparent compatibility between the Central Dogma and *Broad Interpretation* of the Inheritance of Acquired Traits, we should offer up an alternative interpretation that excludes instances of cultural learning. When commentators assert that the Central Dogma entails the denial of the Inheritance of Acquired Traits, I take it they have a narrower interpretation of the latter in mind (Smith 1993; Judson 1979; Dawkins 1970; Cobb 2017; Wilkins 2002; Graur 2018), a definition that concerns non-learned traits with a genetic basis only. We might therefore cash out the heritability of acquired traits as saying that *environmental factors can affect*

<sup>10</sup> As stated previously, many have endorsed the heritability of acquired characters (Gissis 2011; Jablonka and Lamb 2014; Oyama 2003; Wilschut et al. 2016; Koonin 2019; Woolley et al. 2019; Wideman et al. 2019; Jablonka 2019).

<sup>11</sup> This example is taken from Boyd and Richerson (2005).

*an organism's DNA in such a way that these genetic changes result in phenotypic traits that are passed onto the organism's offspring.*

Such an interpretation is plausible, as it would align with defenses of the existence of soft-inheritance, or ...

“the belief in a gradual change of the genetic material itself, either by use or disuse, or by some internal progressive tendencies, or through the direct effect of the environment” (Gissis 2011; Lamb 2011).

The notion of soft-inheritance, coined by Mayr (1982), appeals to the idea that a host of factors can have an impact on an organism's genetic material.<sup>12</sup> Given that soft inheritance is widely seen as a way of spelling out Lamarckian views of evolution (for more on this, see Burkhardt 2013), the congruence of the above view of the heritability of acquired traits with the notion of soft inheritance is thus a reasonable starting point for interpretations of the heritability of acquired traits.<sup>13</sup>

At this point, however, it may be argued that this view of the heritability of acquired traits is problematic, as it includes particular cases of mutagenesis. Consider, for example, cases in which genetic mutations occur in zygotic cells, of which there are many (Wang et al. 2017; Wossidlo et al. 2011). Despite the fact that the zygotic DNA differs from parent DNA, mutations in this example occur within the organism and may give rise to the heritability of phenotypic traits in the resulting offspring. However, while satisfying the above definition, we may not want to consider empirical examples like these instances of the IAT.

It is therefore useful to restrict the above definition of the heritability of acquired traits further, so that it excludes cases of mutagenesis. Specifically, we might offer up the following definition:

**Inheritance of Acquired Traits (IAT):** Environmental factors may affect a particular organism's DNA in such a way that these genetic changes result in phenotypic traits in the particular organism affected, and these phenotypic traits are then passed onto the organism's offspring.

The interpretation above (hereafter, the IAT) will be the definition employed in the rest of this paper.

To illustrate the IAT, let us imagine a scenario in which a set of organisms ( $o^1 - o^n$ ) in a population (P) live in an environment (E) with various environmental factors ( $e^1 - e^n$ ). These environmental factors can range from nutritional resources to particulate matter in the environment. Let's suppose that a subset of the organisms above ( $o^5 - o^{10}$ ) are exposed to a range of particulate matter ( $e^5 - e^{10}$ ), causing changes to the organism's genetic material ( $g^1$ ), in a way that gives rise to a phenotype that is heritable. So, the organisms ( $o^5 - o^{10}$ ) now possess a phenotype (P) that

<sup>12</sup> For more on the idea of Soft Inheritance, see Gissis (2011) and Mayr and Provine (1998). For further discussion, see Dickins and Rahman (2012) Mesoudi et al. (2013) and Gissis (2011).

<sup>13</sup> Notice, however, that the interpretation of the heritability of acquired traits above is more restrictive than this statement of soft inheritance, in that it only makes appeal to environmental factors and not “internal progressive tendencies”—such as enzymes which help with the transcription of DNA.



can be passed on to their offspring ( $f^1 - f^n$ ). The offspring ( $f^1 - f^n$ ) now possess the phenotype (P). This illustration counts as an instance of the IAT, because environmental factors ( $e^5 - e^{10}$ ) affected organisms ( $o^5 - o^{10}$ ) DNA ( $g^1$ ) in such a way that these genetic changes resulted in phenotypic traits (P) that are passed onto the organism's offspring ( $f^1 - f^n$ ).

Now, it turns out that realistic examples of the IAT exist. One key such example may be found in the phenomenon of RNA interference (or RNAi). RNAi occurs when strands of RNA are introduced into an organism thereby inhibiting the expression of specific genes. Vastenhouw et al. (2006) demonstrated that such effects result in the heritable transmission of traits. Specifically, Vastenhouw et al. injected double-stranded RNA into the *ceh-13* gene in nematodes. The *ceh-13* gene was silenced in the organism and this silencing persisted indefinitely. This particular case of RNAi counts as an instance of the IAT since external factors are in fact affecting an organism's DNA in a way that such effects are inherited by an organism's offspring.

### 3 The Central Dogma and “Lamarckian” Inheritance

Given that we now have specific interpretations of the Central Dogma and the Inheritance of Acquired Traits out on the table, we are now in a better position to evaluate whether the truth of the Dogma implies the falsity of the IAT. Better yet, we can now evaluate whether the two principles are *really* incompatible.

Before doing this, however, it is worth noting that many commentators take it that the Central Dogma and the Inheritance of Acquired Traits are incompatible theses (Smith 1993; Judson 1979; Dawkins 1970; Cobb 2017; Wilkins 2002). Dawkins (1970) famously asserts that to treat an organism as a replicator “is tantamount to a violation of the ‘central dogma’ of the non-inheritance of acquired characteristics.” (p. 97) Sahotra Sarkar notes that the Central Dogma might be “an explication, at the molecular level of the well-known biological fact that acquired characteristics cannot be inherited.” (p. 205) Judson (1979) claims that the Dogma is “the restatement—radical, absolute—of the reason why characteristics acquired by an organism in its life but not from its genes cannot be inherited.” Finally, in an interview with Wilkins (2002), Ernst Mayr argues that the Dogma constitutes “the final nail in the coffin of the inheritance of acquired characteristics.” Given that for many of these commentators the Central Dogma marks conclusive evidence against the inheritance of acquired characters, the proponent of the inheritance of acquired characters faces a challenge; as Smith (1966) puts it, the burden is on the “Lamarckist” to “disprove the dogma.” (p. 66)

Given that Maynard Smith's challenge presupposes an incompatibility between the Central Dogma and the IAT, and it's worth taking a moment to wrap our minds around why this may be the case. As evidenced above, commentators take it that the Central Dogma implies a process that contradicts the processes involved in the IAT. Specifically, the Central Dogma implies token molecular processes where an organism's traits are the result of DNA making protein. According to the IAT, in order for an organism to acquire heritable traits, the token molecular processes described by

the Central Dogma would have to work backwards (as Maynard Smith points out). That is, DNA would have to be manipulated in a way that results in the heritable transmission of traits from parent to offspring. Since such a process *could not* work backwards—as laid out in the narrow interpretations of the Central Dogma—the falsity of the IAT is the flipside of the truth of the Central Dogma. As Cobb (2017) puts it, though there is much excitement about DNA methylation and epigenetic inheritance in the biological sciences, “there is no evidence in any organism that the information in a DNA sequence can be rewritten from information in a protein.” In other words, the Dogma and the IAT are incompatible if the token molecular processes described by the latter principle are ruled out by the processes described by the former principle—or vice versa.

The point above can also be cashed out as follows. One might argue that defenders of the IAT (or soft-inheritance) are forced to deny the Dogma, on pain of empirical inconsistency.<sup>14</sup> This is, again, because the token molecular processes involved in the heritability of acquired traits rules out the occurrence of the molecular processes involved in the Dogma.

However, this is a misunderstanding. To see why, let’s assume that the IAT is true, which is relatively easy to do since, as noted earlier, empirical evidence suggests it *is* true! For concreteness, return to the case of RNA interference (the argument will be generalized momentarily). Does commitment to the view that cases of RNAi are cases of IAT imply that *Crick’s Dogma*, the *Informational Dogma*, or the *Gene-centric Dogma* are false? A closer look suggests that this is not the case.

Consider the first version of the Central Dogma, namely, *Crick’s Dogma*. Recall that *Crick’s Dogma* states that the transfer of information—in the specific sense of sequence determination—from DNA to RNA to protein is possible, and the transfer of information from protein to DNA and from protein to protein is impossible. However, the phenomenon of RNAi is entirely consistent with this claim. The fact that RNAi persists indefinitely in specific organisms does not yet imply that *Crick’s Dogma* is false. This is because *Crick’s Dogma* only prohibits information transfer from proteins to amino acids and from proteins to nucleotides, and RNAi occurs when RNA interferes with DNA, which is permissible according to *Crick’s Dogma*. Given this, the truth of the Central Dogma—if understood in terms of *Crick’s Dogma*—does not imply the falsity of the IAT. In order for the case of RNAi to imply the falsity of *Crick’s Dogma*, the effects of RNAi would have to occur in such a way that proteins do in fact alter amino acids in a protein sequence.

Let us now consider the *Informational Dogma*, which holds that DNA alone carries information about protein. Recall that, on this interpretation, in much the same way certain clouds signal rainfall, DNA signals and carries information about codons translated into peptide sequences. Like the *Gene-centric Dogma*, the *Informational Dogma* is entirely compatible with cases of RNAi, because the molecular processes stipulated by the *Informational Dogma* are not at odds with the ones in cases of RNAi: the *Informational Dogma* only prohibits that biomolecules other

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<sup>14</sup> For more on proponents of the IAT (or soft-inheritance generally), see Koonin (2019), Jablonka (2019), Jablonka and Lamb (2014), and Lamb (2011).

than DNA carry information about protein. Given this, the *Informational Dogma* is compatible with the IAT: the fact that the manipulation of DNA in nematodes can result in phenotypic traits that persist indefinitely for generations does not yet imply that the *Informational Dogma* is false, because it does not deny that DNA carries information about proteins.

Finally, exactly the same can be said of the *Gene-centric Dogma* and the IAT. Recall that the *Gene-centric Dogma* states that DNA is the most significant causal factor involved in protein synthesis, because DNA is a causally-specific, actual difference-maker (or, SAD) with respect to protein synthesis. Again, though, the case of RNAi is entirely consistent with the fact that DNA makes an actual, causally-specific difference to codons expressed in polypeptide sequences. In other words, that segments of DNA make a causally specific difference to protein synthesis has no bearing on the fact that, for certain organisms, heritable changes in phenotypes result from environmental factors. The *Gene-centric Dogma* only denies that factors other than DNA segments can make *the same* causally-specific differences to proteins that DNA makes. The *Gene-centric Dogma*, therefore, does not yet imply the falsity of the Inheritance of Acquired Traits. Indeed, this point follows for all relevant forms of the IAT. Whether or not acquired genetic changes can be inherited has no bearing on whether or not genes are the most significant causal determinants of protein synthesis (in a specific sense of “significant”). These are completely independent claims.

In this way, it becomes clear that one of the key examples of the IAT—RNA interference—is not intrinsically at odds with the Central Dogma. Something similar also holds for other forms of the IAT. For example, CRISPR technology has afforded molecular scientists a means for editing genes and with it the phenotypic traits of an organism (for a brief summary on CRISPR, see Ledford 2016.): there are cases where CRISPR alterations result in the heritability of acquired traits (for more on this, see Howells et al. 2018). Briefly, CRISPR technology works by using enzymes to alter DNA sequences, which in turn result in the generation of heritable phenotypic traits. CRISPR technology satisfies the IAT, but not in a way that implies the falsity of the *Gene-centric Dogma*, the *Informational Dogma* or *Crick's Dogma*, and thus not in a way that denies (a) the causal primacy of genetic over non-genetic factors, (b) that genes alone carry information about codons in peptide sequences, or c.) that proteins can alter other proteins or nucleobases in DNA or RNA.

Generalizing these points, this thus shows that there is no tight-knit connection between the heritability of (non-culturally) acquired traits and the Dogma. While it is possible that future instances of IAT do turn out to violate the Central Dogma—e.g. it is possible that we will discover instances where alterations to certain proteins somehow end up affecting nucleotide sequences—this is a contingent (and so far unactualized) possibility. The key point to note here is just that there is no *principled* incompatibility between the IAT and the Dogma. We can accept the truth of both the IAT and the Dogma: these two types of processes *need not* be in conflict with each other.

In this way, it becomes clear that Ernst Mayr's claim that the Central Dogma is “the final nail in the coffin of the inheritance of acquired characteristics”

(Wilkins 2002) is false. No intrinsic incompatibility between the Dogma (if sketched as the *Crick's Dogma*, *Gene-centric Dogma*, or *Informational Dogma*) and the inheritance of acquired characters (if sketched as the IAT) exists.

#### 4 A Brief Note on The Central Dogma and Conceptual Pluralism

Some commentators have advocated for conceptual pluralism in the philosophy of biology (Stencel and Proszewska 2018; Lu and Bourrat 2018). Stencel and Proszewska (2018), for example, note that certain concepts—which appear to be at odds with each other—play different roles in science in that they “respond differently to ongoing discoveries.” In the case of organisms, for example, Stencel and Proszewska (2018) distinguish between the *Developmental Concept* of organism and the *Cooperation-Conflict Concept* of organism. The former holds, roughly, that organisms are “dynamic entities which undergo a process of development from simple to more complex beings,” while the latter holds, roughly, that organisms are functional systems “built of elements that cooperate to sustain... stability.” (p. 606, p. 610) According to Stencel and Proszewska (2018), the *Developmental Concept* serves a particular scientific aim, in that it aims to capture a host of developmental processes occurring within the organism; the concept, however, is not particularly well-suited for the comparative study of distinct organisms, unlike the *Cooperation-Conflict Concept*. This doesn't mean, however, that the *Developmental Concept* should be rejected wholesale, and that the *Cooperation-Conflict Concept* should replace it. Rather, the two concepts serve two distinct scientific aims within biology. The *Developmental Concept* of organisms has the scientific aim of capturing the ins-and-outs of an organisms' development. The *Cooperation-Conflict Concept* of organisms has the scientific aim of allowing for the comparative study of distinct organisms.

The current discussion about the Central Dogma's compatibility with the IAT bears on the topic of conceptual pluralism, in that it shows that the acceptance of the IAT is independent of the debate about the plausibility of the Central Dogma, *however the latter is understood*. That is, if one adopts conceptual pluralism about the Central Dogma, and thus sees the *Gene-centric Dogma*, the *Informational Dogma* and *Crick's Dogma* as speaking to different questions, then the IAT is perfectly compatible with all of these interpretations. Unlike the example with the concepts of organism sketched above—where the comparative study of organisms is made possible by the Cooperation-Conflict Concept *as opposed to* the Developmental Concept—the discovery that DNA can be manipulated in ways that result in heritable traits does not pose a problem for *any* interpretations of the Central Dogma. So, it is not just that some forms of the Central Dogma are consistent with the IAT—rather, at least in principle, *all* of them are.

## 5 Conclusion

At the outset of this paper, it was noted that Smith (1993) once issued a challenge for those in favor of Lamarckian inheritance; for Maynard Smith, because the “process whereby information is passed from DNA to protein is now fairly well understood,” the Lamarckist “must disprove” the Central Dogma. As we saw, this challenge presupposes an incompatibility between the Central Dogma and the heritability of acquired traits. In critically evaluating this assumption, we saw not only that (a narrow interpretation of) the heritability of acquired traits is true, but also that the Central Dogma is not necessarily incompatible with Lamarckian inheritance. These considerations suggest that Maynard Smith’s challenge, and the assumption on which it rests, is unfounded.

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