The Genesis of Change:

On the Adequacy of the Genocentric Perspective to Describe the Baldwin Effect

By: Pam Tönissen

Student number: 4480910

Supervisor: prof. dr. Christoph H. Lüthy

Words: 17729

Date: 30-06-2019

Thesis for obtaining a “Master of arts” degree in philosophy at Radboud University Nijmegen

I hereby declare and assure that I, Pam Tönissen, have drafted this thesis independently, that no other sources and/or means other than those mentioned have been used and that the passages of which the text content or meaning originates in other works - including electronic media - have been identified and the sources clearly stated.

Place: Nijmegen – Date: 30-06-2019

Abstract

The aim of this thesis is to examine the tension between the different roles that the individual plays in the Baldwin Effect – introduced by James Mark Baldwin as 'organic selection' – and the Genocentric Perspective – described through the work of Richard Dawkins. In order to analyse this tension, the question whether the Genocentric Perspective is sufficient to describe the Baldwin Effect is considered. Firstly, Baldwin's original ideas are described, after which they are combined with two modern notions of 'genetic assimilation'. This combination provides a connection with the Genocentric Perspective, as the latter understands the Baldwin Effect as equivalent to one of these two forms of genetic assimilation. The legitimacy of this understanding is then judged, and the central question answered.

Table of Contents:

Introduction

As a young child, I played a lot of basketball. This caused me to develop a layer of callus on my fingertips, something that worried me considerably. I imagined that my own future children would be born with these same calluses and that, if they decided to play basketball themselves, their children were in turn to be born with even thicker calluses. In my mind's eye, I could already picture my great-great-grandchildren: how would they ever be able to dribble, draw, write or even eat with their grotesque paws full of calluses as thick as mattresses? Was I not obliged to keep the thickening of my calluses to a minimum, so that my progeny would not turn out severely disabled?

In retrospect, I can recognize the profoundly Lamarckian nature of this way of thinking. Just as the French biologist Jean-Baptiste Lamarck had done in the early 19th century, I had supposed that acquired characteristics – in my case calluses – could be directly inherited.[1] A further supposition that logically followed from this, is that the evolutionary development of a species could be steered through the accumulation of changes that occurred during the lifetimes of individuals. Largely thanks to Darwin's theory of evolution by natural selection, however, we now know that this is not how evolution works: a species evolves gradually through the differential survival and reproduction of individual organisms, and not because of the way individuals adapt to their surroundings during their lifetimes. Lamarckism quickly fell out of favour as Darwinism gained more and more ground.

However, the American psychologist James Mark Baldwin was dissatisfied with the idea that the theory of natural selection on congenital variations was enough to explain the progressive nature of a species' evolutionary development.[2] He observed that individual organisms are not just the passive

[1] *Biology Online Dictionary*, s.v. "Lamarckian Theory," accessed 12-06-2019, URL: https://www.biology-online.org/dictionary/Lamarckian_theory.
[2] James Mark Baldwin, *Mental Development in the Child and the Race: Methods and Processes* (New York: Kessinger Publishing, 1906), 193-194.

puppets of natural selection, but that they often have the ability to adapt to extreme environmental changes during their own lifetimes. This gave them an advantage in the struggle for survival. As a consequence, the advantageous individual adaptation could later be mimicked, amplified or confirmed by congenital variations occurring in subsequent generations.[3] An acquired characteristic could thus, over time, become hereditary after all.

At first glance, this idea may seem like a blunt return to Lamarckian inheritance of acquired characteristics. However, Baldwin did not suppose that the acquired characteristics *themselves* were directly passed down. Instead, he argued that it is possible for organisms to influence the workings of natural selection through their ability to modify their own forms and functions, and so direct the course of evolution to a certain extent. Baldwin thus claimed that acquired characteristics *did* deserve a place within Darwinian evolution, without falling back onto Lamarckian inheritance. His theorem is nowadays referred to as the 'Baldwin Effect', and has in recent years been employed within the context of computational cognitive science and bio-semiotics. It has also often been understood, notably by theorists like Daniel Dennett and Terence Deacon, as a mechanism that speeds up evolution and that can help to account for the emergence of human consciousness and language. At its conception, however, Baldwin's idea mainly signified speculation about the influence of learned behaviour on evolution.[4]

As Baldwin developed this modified version of the inheritance of acquired characteristics, Gregor Mendel's work on the laws of inheritance – which would later form the basis for modern genetics – was well underway. With the introduction of genetics into Darwinian evolutionary theory – a combination that resulted in the paradigm we now know as the 'Modern Synthesis' –, the once so

[3] Adam C. Scarfe, "James Mark Baldwin with Alfred North Whitehead on Organic Selectivity: the "Novel" Factor in Evolution," *Cosmos and History: The Journal of Natural and Social Philosophy* 5, no. 2 (2009): 52.
[4] Bruce H. Weber and David J. Depew, "Preface," in *Evolution and Learning: The Baldwin Effect Reconsidered*, eds. Bruce H. Weber and David J. Depew (Cambridge, Massachusetts: The MIT Press, 2007), ix-x.

mysterious mechanisms of heredity were illuminated; it became widely accepted that it is not a characteristic itself that is inherited, but the genes that encode for that characteristic.[5] Because acquired characteristics lack this genetic foundation, they cannot be directly passed down. The idea of Lamarckian inheritance of acquired characteristics was thus dealt the final blow.

The Modern Synthesis is not plainly incompatible with the Baldwin Effect. However, the considerable degree of causal influence that Baldwin allocates to organisms appears to be tricky, since the introduction of genetics into evolutionary biology caused biologists to question the central position of the individual within evolutionary theory. The uncovering of the underlying units of heredity meant that it was no longer obvious to see the individual organism as the undisputed target of natural selection. It now became a crucial question as to whether we should not think about evolution by natural selection in terms of the differential survival of genes instead of organisms, since it is actually always a genetic tendency that is passed down.

Around the turn of the 20th century, we can detect the first clear signs that the pendulum was swinging away from an organism-centred biology. In his book *Creative Evolution*, the French philosopher Henri Bergson depicts the evolution of life in the following manner:

> *[…] life is like a current passing from germ to germ through the medium of a developed organism.* It is as if the organism itself were only an excrescence, a bud caused to sprout by the former germ endeavouring to continue itself in a new germ. The essential thing is the continuous progress indefinitely pursued, an invisible progress, on which each visible organism rides during the short interval of time given it to live.[6]

[5] *Dictionary*, s.v. "Modern Synthesis," accessed 12-06-2019, URL: https://www.dictionary.com/browse/modern-synthesis.
[6] Henri Bergson, *Creative Evolution*, trans. Arthur Mitchell (New York: Random House Publishers, 1944), 32.

In this passage, the organism is not only discarded as the fundamental evolving entity, but reduced to a medium of which germs – which we can retrospectively identify as genes – can make use in their journey down life's current. During the following decades, this idea slowly grew into an explicitly gene-centred view of evolution, until it was prominently put on the map through the work of Richard Dawkins in the 1970s. In his account of the so-called 'Genocentric Perspective' on evolution, organisms are identified as the passive vehicles for genes. If we want to understand any given adaptive trait we may observe in an individual, Dawkins argues, we should ask ourselves how this trait's existence helps its underlying genes to survive and replicate. From the Genocentric Perspective, the selection of genes is thus the ultimate cause of evolutionary development, while the organism is merely a short-lived and inconsequent result of it.[7]

Here, a certain tension seems to emerge between the Genocentric Perspective and the Baldwin Effect: according to the latter, the organism is to be seen as an entity with actual causal power in directing the evolutionary development of its species, while the former results in an understanding of the organism as a mere result of selection at the genetic level. In short, the tension that emerges here concerns the role that the individual organism is to play in a species' evolution. In this thesis, I will analyse this tension, and examine whether it can be nullified by uniting the Baldwin Effect with the Genocentric Perspective. To find this out, I will consider whether Baldwin's ideas can, with hindsight, be integrated into the Genocentric Perspective on evolution, or whether this modern viewpoint

[7] It should be noted that Dawkins developed his version of the Genocentric Perspective at a time when considerably less was known about genetics, heredity, ontogenetic development and the relationships between them. Within our current time of extensive research into the compositions of genomes and especially into epigenetic factors, it can be doubted whether the somewhat simplistic ontological idea of the gene as the 'basic unit of selection' is still as plausible as it was in the 1970s. In this thesis, however, I will consider the refined and modified version of Dawkins' ideas, in which he has attempted to deal with some of the most prominent criticisms. The core of the Genocentric Perspective on evolution, however, has remained roughly unchanged and continues to draw heavy fire. Here, I will not discuss whether this is legitimate or not, nor will I consider if and how the most recent developments in epigenetics and related fields should be integrated into the Genocentric Perspective. Instead, I will regard Dawkins' latest version of the this perspective as a valuable and influential position, regardless of the objection that parts of it may nowadays be viewed as obsolete.

is conceptually insufficient to deal with Baldwin's old observations. The central question on which I shall focus, can thus be formulated as follows: *to what extent can the Genocentric Perspective on evolution be considered sufficient to describe the Baldwin Effect?*

In order to answer this question, the most important aspects of both the Baldwin Effect and the Genocentric Perspective will have to be clearly defined and thoroughly examined. In the first chapter, I will start this examination by returning to the time when the term 'Baldwin Effect' had not yet been coined. The process Baldwin originally described was called 'organic selection', and it only came to be known as the Baldwin Effect after the renowned palaeontologist George Gaylord Simpson named it thus in one of his articles, over half a century later.[8] Since Baldwin's theory of organic selection predates the integration of genetics into evolutionary biology, the first chapter will be exclusively written from a pre-genetic point of view. In the second chapter, we will enter a scientific discourse in which genetics has claimed a prominent place, and complement our theoretical framework with a more modern biological vocabulary. Two distinct concepts of 'genetic assimilation' will be introduced, both of which have at times been understood as equivalent to the Baldwin Effect. This equalization provides us with a way to introduce the Genocentric Perspective into our inquiry, as this perspective defines the Baldwin Effect as equivalent to one of the two forms of genetic assimilation. In the third chapter, we will consider why this interpretation of the Baldwin Effect naturally results from the Genocentric Perspective. In doing so, the most fundamental aspects of the Genocentric Perspective will be illuminated. In the fourth and final chapter, we will return to Baldwin's original theorem, and consider how the two forms of genetic assimilation that were distinguished relate to it. This will enable us to draw a conclusion regarding the extent to which the Genocentric interpretation of the Baldwin Effect suffices to give a comprehensive description of Baldwin's original theorem. After this

[8] David J. Depew, "Baldwin and His Many Effects," in *Evolution and Learning: The Baldwin Effect Reconsidered*, eds. Bruce H. Weber and David J. Depew (Cambridge, Massachusetts: The MIT Press, 2007), 3.

conclusion has been drawn, we will briefly consider how it should be interpreted in the discussion. Our inquiry will come to a close with the suggestion of two possible opportunities for further research.

The Birth of the Baldwin Effect: Organic Selection

Before we can begin our investigation into the Baldwin Effect and the Genocentric Perspective, it must be noted that both Darwin and Baldwin were unfamiliar with the notion of genes. Although Gregor Mendel's discovery of the laws of heredity in 1865 – which laid the basis for modern genetics – took place within the lifetimes of both Darwin (1809-1882) and Baldwin (1861-1934), neither of them ever read his work.[9] Mendel's findings only began to be noticed by other scholars within the field of evolutionary biology at the beginning of the 20th century.[10] To understand the context in which Darwin, Baldwin and their contemporaries worked, we thus have to go back to the period before genes first entered the scene, when naturalists had to make do with directly observable phenomena. It was in this context that Darwin wrote his immensely influential *On the Origin of Species*, a work which quickly became the foundation upon which countless naturalists built their own theories. Since Baldwin was one of these naturalists, it is with Darwin's theory that any comprehensive account of the Baldwin Effect must begin.

In brief, Darwin observed the following: organisms within a given population all have certain defining characteristics, but in each population there exists variety among individuals. The individuals best adapted to their environment have a higher chance of surviving and will therefore produce more offspring than their less adapted peers. Generally, more offspring is produced in each new generation than can possibly survive. The offspring of well-adapted

[9] Richard Dawkins, *The Selfish Gene: 40th Anniversary Edition* (Oxford: Oxford Landmark Science, 2016), 43.
[10] Gregory Carey, *Human Genetics for the Social Sciences* (Thousand Oaks: Sage Publications, 2003), 148.

individuals will inherit their parents' adaptive characteristics, which increases the number of well-adapted individuals within the population over the subsequent generations. However, a particular individual is not identical to its offspring; due to the mixing of organisms' characteristic traits in reproduction, variety between individuals keeps re-emerging in each new generation. Because of this reoccurring variety, the species never reaches a point at which all individuals are equally well-adapted. Instead, better adapted individuals will continue to prosper, while less adapted individuals will continue to be penalized. Through this process, organisms of a given species get more and more adapted to their ever changing environments. As Darwin pointed out, it is as if a conscious breeder ceaselessly selected the best individuals for breeding, until the optimally adapted individual would emerge. Darwin's revolutionary insight was that the natural struggle for survival could fulfil the role of an unconscious, blind selector. Hence, he adequately coined this process 'natural selection'.[11]

Natural selection is as powerful a process as it is ruthless, especially when interpreted in the manner of many neo-Darwinian naturalists who came shortly after Darwin. These naturalists – already familiar with August Weismann's empirical evidence for the falsity of Lamarckian inheritance of acquired characteristics – supposed that evolutionary change was solely based on natural selection, and that natural selection in turn was solely based on inheritance of congenital characteristics.[12] This idea of 'the survival of the fittest' evokes a rather gruesome image: a pile of passive creatures that are completely at the mercy of the deadly scythe of natural selection, powerless to do anything but wait to either receive its devastating blow, or to feel it pass just over their heads.[13] Less metaphorically, this interpretation of Darwinism is consistent with the idea of natural selection as a purely negative force, causing species to gradually evolve adaptations by simply removing all maladaptive variants.

[11] Charles Darwin, *On the Origin of Species by Means of Natural Selection* (New York: D. Appleton and Company, 1859), 61.
[12] Depew, "Baldwin and His Many Effects," 8-9.
[13] Ibid., 13.

The – in his opinion – disproportional attention that naturalists payed to this element of Darwin's theory, prompted Baldwin to take action. In his paper "A New Factor in Evolution," published in 1896, he challenged those neo-Darwinians who thought this purely negative force was enough to account for the adaptive direction in which species tend to evolve.[14] He noted that natural selection is simply a description of what happens when organisms are not sufficiently well-adapted to survive within their given circumstances, and that it is therefore inadequate to clarify how the organisms that *do* survive develop their adaptations in the right place and at the right time.[15] In other words, while natural selection can describe which organisms die, it cannot define positively the qualifications which enable organisms to live.[16] To provide a more comprehensive account of a species' evolutionary development, Baldwin argued that the emphasis should not only be laid on natural selection, but also on the variation among individuals and especially on how this variation comes about. Variation, Baldwin stressed, is not simply a given, because organisms can – to a certain extent – adapt to their circumstances *during their lifetimes*, and so actively influence the range of variations that are available to natural selection. Emphasizing this positive side of Darwinism would enable naturalists to apply Darwin's theory not only to what organisms are, but also to *what they can do*.[17]

In order to construe this more inclusive way of employing Darwinism, Baldwin introduced the concept of 'organic selection'. He did not coin this term as a replacement of natural selection, but rather as complementary to it. Just as Darwin had done, Baldwin set out by observing the enormous amount of variety between individual organisms, but his attention was particularly drawn to the flexible nature of this variety; organisms were not just born with a static set of characteristics, but could acquire new characteristics if sufficiently pressured by

[14] Scarfe, "James Mark Baldwin," 51.
[15] James Mark Baldwin, "A New Factor in Evolution," *The American Naturalist* 30, no. 354 (1896): 3.
[16] Ibid., 11.
[17] Ibid., 12.

their environments. The present-day philosopher Adam Scarfe nicely captures the gist of this idea:

> […] especially in times of environmental duress, members of a species may manage to make accommodations to their dominant modes of behaviour, developing and selecting new habits or functions which allow them to adapt to their conditions and keep themselves temporarily alive.[18]

By producing new variations during their own developmental history, some organisms manage to delay the blow of natural selection's scythe, or even to avoid it altogether. Baldwin called these developmental variations *ontogenetic variations*, as opposed to *phylogenetic variations*. The latter refers to the raw material that is involved in the evolutionary development of a species from generation to generation.[19] In contrast to this, the raw material of organic selection consists of variations acquired during ontogenetic development. Here, it must be noted that the 'genetic' within the words 'ontogenetic' and 'phylogenetic' does not yet refer to our word 'gene', which was only coined in 1909.[20] The word 'genetic' as used in the 19th century is linked to the word 'genesis' and was commonly understood to mean 'pertaining to origins', although Darwin used it as 'resulting from common origin'.[21] In what is to follow, I will clearly mark the moment at which I will first use the word 'genetic' as linked to our contemporary word 'gene'.

There are three distinct forces which can influence an organism in the course of its life, each giving rise to a different kind of ontogenetic accommodation, namely 'mechanical', 'neurological' and 'intelligent'. The individual features in an increasingly active role in each of these types of

[18] Scarfe, "James Mark Baldwin," 51-52.
[19] Baldwin, "A New Factor," 1-3.
[20] Ananya Mandal, "Gene History," *News Medical Life Sciences*, last modified 22-04-2019, URL: https://www.news-medical.net/life-sciences/Gene-History.aspx.
[21] *Online Etymology Dictionary*, s.v. "Genetic," accessed 10-06-2019, URL: https://www.etymonline.com/search?q=genetic.

accommodation. Firstly, an individual may be caused to make accommodations due to physical, environmental influences, such as temperature changes, contact with other individuals, chemical agents, and growth hindrances. These influences are external to the individual, and since Baldwin considered them to be accidental – the individual 'happens' to be confronted with them –, he also called the consequent accommodations they induce 'accidental', or 'fortuitous'.[22] Although Baldwin does not provide concrete examples of any of his three proposed classes of accommodation, we can assume that words like 'fortuitous' and 'accidental' are in this context to be understood as opposed to 'directed'; an organism *inadvertently* and mechanically modifies its congenital form or functions, in reaction to contingent features of the environment. I would propose the following example of such an accommodation: suppose a certain plant thrives in a shady environment, because this allows it to open its flowers which would otherwise wither in the sun. Since plants of this kind normally grow in places where the sun cannot singe them, their flowers are typically opened all day. The large tree shading one particular plant now happens to fall down, allowing the sunrays to reach the flowers. The plant is thus induced to adapt to this new circumstance by opening its flowers at a later time of the day, when the sun is once again blocked by lower vegetation. This change in its normal function allows it to survive.

Of course, the plant itself does not deliberately initiate the change in its congenital function. Instead, an external trigger induces the fortuitous accommodation to occur; had the trigger not been there, the accommodation would not have been induced. We may indeed consider this an ontogenetic modification of the plant's normal characteristics, even if we presume that the capacity to accommodate is already present in the plant's congenital constitution; were the plant to reproduce, its offspring would not directly inherit the deviant timing of its parent, although they might in turn adapt to changes in the amount of sunlight they themselves receive. Only the potential to accommodate is thus inherited, not the accommodation itself. Baldwin used the term *physico-genetic* to

[22] Baldwin, "A New Factor," 2.

describe these ontogenetic accommodations, in reference to the physical forces which induce them.[23] Although the ability to make such mechanical accommodations does not elevate individuals from their passive role, it does enable them – up to a certain degree – to counteract the immediate workings of natural selection.

The second class of accommodations Baldwin distinguishes are not solely induced by external influences – although environmental duress does promote their occurrence –, but spring from spontaneous, neurologically induced activities the individual itself performs in carrying out its normal functions. These accommodations thus rely more upon the individual as an active agent that deals with changing circumstances. An example of this could supposedly be a lioness that instinctively hunts with her body held very low to the ground, to avoid being spotted by her prey on the open plains of the savannah. Should this lioness suddenly find herself in a dense forest, she may spontaneously take on a more upright hunting-posture in order to better track her prey. Since the chances of losing sight of the prey will increase in a wooded area, and the dense vegetation will likely prevent the prey from spotting the lioness, changing her normal hunting-posture would be advantageous to the lioness. This new hunting-posture may grow out into a habit if it continues to benefit the lioness, at which point a new accommodation will have been developed. Here too, we can speak of an ontogenetic accommodation, since the lioness' offspring will not be born with an instinct to adopt this new hunting-habit when they are only confronted with normal circumstances. Yet it differs from a physico-genetic accommodation, since the occurrence of the external environmental trigger is not sufficient – although it is necessary – to induce a specific accommodation. The lioness' own actions, which are constituted by neurological processes, are needed as a mediator between the external trigger and the accommodation that is initiated in reaction to this trigger. In reference to the 'selective property' of organisms' nervous systems – that is to say, the neurological selection of some movements, postures, actions and

[23] Baldwin, "A New Factor," 2.

consequential habits over others –, Baldwin called these modifications *neuro-genetic*.[24]

It is with the third class of ontogenetic accommodations that the individual graduates to *deliberate* agent, selecting consciously – to a greater or lesser extent – which modifications to make. Accommodations of this kind may be induced by various different influences, which can be put together under the word 'intelligent'. An organism may for instance be induced to accommodate by a simple process of trial and error, but we may also think of imitation, social learning and even of reasoning from means to ends as potential triggers. Take the case of a starling that discovers how to open the lid of a milk bottle. Via a process of trial and error, the starling will gradually modify this new skill and in time master it completely. Other starlings may imitate this new skill, which will therefore spread through the then living population. Yet the starlings' chicks will in due course have to acquire the skill anew, potentially via parental instruction or imitation. Accommodations of this type differ from physico-genetic ones, since the environmental change alone is insufficient to mechanically produce the accommodation. They differ from neuro-genetic ones in that they are not thoughtless, spontaneous actions and movements but *deliberations* that rely upon a highly developed capacity to recognize patterns, and remember previous experiences. After all, without the ability to recount the results of previous – spontaneous – actions, a process of trial and error could not arise, let alone the capacity to connect cause and effect. Baldwin called this last group of adaptations *psycho-genetic*.[25]

All in all, the gist of Baldwin's distinction between the three kinds of ontogenetic accommodation, is the following: physico-genetic accommodations are changes that *happen to the individual*. They are accidental in the sense that the individual *happens* to come into contact with some environmental trigger and inadvertently and mechanically adapts to them. Neuro-genetic accommodations

[24] Baldwin, "A New Factor," 2.
[25] Ibid., 2.

are changes that are *initiated by the individual* through its own spontaneous actions in the face of changing circumstances. They are dependent upon the individual's initiative, in the sense that without its actions, the change would not be initiated. Psycho-genetic accommodations are also changes that are *initiated by the individual*, but in this case through actions that are – to a greater or lesser extent – *consciously directed* towards coping with certain changing circumstances, and can potentially spread through the population socially. In that sense they not only depend upon the individual's initiative, but also on its *deliberations*.[26]

Taken together, these three types of ontogenetic accommodation form the raw material on which organic selection works. Once more, the analogy with natural selection is plain: both processes rely upon the initial production of variants, and just as some congenital characteristics are selected because they simply work better, some acquired characteristics are selected because they allow the organism that acquired them to survive.[27] The one important difference is the nature of the force driving the selection in each respective process, a difference to which Baldwin refers when he explains his choice of the term 'organic selection':

> [t]he use of the word "Organic" in the phrase was suggested from the fact that *the organism itself cooperates* [emphasis mine] in the formation of the adaptations which are effected, and also from the fact that, in the results, the organism is itself selected; since those organisms which do not secure the adaptations fall by the principle of natural selection. And the word

[26] Note that these classes should not be thought of as clear-cut categories. Instead, they should be considered as arbitrary classes, described as clearly outlined types of accommodations for the sake of convenience. The factual differences between them, however, are a matter of degree. We will revisit this point in the discussion.

[27] Celia L. Moore, "Evolution, Development, and the Individual Acquisition of Traits: What We've Learned Since Baldwin," in *Evolution and Learning: The Baldwin Effect Reconsidered*, eds. Bruce H. Weber and David J. Depew (Cambridge, Massachusetts: The MIT Press, 2007), 115.

> "Selection" used in the phrase is appropriate for just the same two reasons.[28]

The concept of organic selection thus incorporates the organism as a force of selection in its own right, while at the same time it emphasizes that the effects of organic selection influence the mechanism of natural selection.[29] Baldwin goes on to highlight several of these effects as particularly important steps in the transition from individual adaptations to the evolution of a whole species.

In the first place, as has already become apparent in the above, the foremost effect of securing ontogenetic adaptations is that the individual is kept alive, even in the face of changing circumstances.[30] This first effect may seem trivial, but it does form the very foundation of the bridge between ontogeny and phylogeny: those phylogenetic traits that are open to mechanical, neurological or intelligent modification are kept in existence, because the modifying individual itself is kept in existence. These modifiable traits will consequently 'stay in the game' for a longer period, perhaps even long enough to be passed down to the next generation. In this manner, ontogenetic modification helps determine the phylogenetic variation that will stay available to natural selection during a species' evolutionary development.[31]

An indirect but crucial consequence of the spread of modifiable traits through a population, is that this population potentially gains breathing space to further evolve and secure these traits. This effect accompanies all ontogenetic modifications, but is especially strong when psycho-genetic modifications are

[28] Baldwin, "A New Factor," 13.
[29] As was stated at the outset of this chapter, Baldwin did not agree with some of his neo-Darwinian contemporaries, who held that natural selection was sufficient to account for Darwinian evolution. However, it must be noted that Darwin himself was quite open to the idea that changing conditions of life could induce modifications in individual organisms. For this, see: Scarfe, "James Mark Baldwin," 53. Nor did he dismiss the notion that intelligent actions could, after being handed down socially from generation to generation, become hereditary and turn into instincts. Baldwin's introduction of organic selection was not meant to criticize a faulty aspect of Darwin's original ideas, but merely as an appropriate supplement to a sound theory that he felt had been interpreted in too narrow a manner by many of his contemporaries.
[30] Baldwin, "A New Factor," 2.
[31] Ibid., 3.

involved, since these may be maintained in a population over a long period through imitation and social heredity. It is quite conceivable that a beneficial trait that initially has to be learned with each new generation gives this population enough time to develop – via the normal process of reproduction and natural selection – new phylogenetic, hereditary variants that confirm and coincide with the ontogenetic trait. A trait that starts out as an individual adaptation can thus over time lose its dependency upon learning processes and environmental triggers, and become hereditarily fixed.[32] These effects of organic selection embody a potential way around Weismann's idea that all inheritable characteristics are carried in the germ plasm and acquired characteristics cannot be inherited in any way.[33] Moreover, they adequately provide the positive side of Darwinism that Baldwin was looking for: instead of only stating which organisms have what it takes to survive from the get-go, a process had now been postulated that provides a more inclusive account of how the qualifications needed for survival might emerge and how initially individual modifications could mature into full-fledged Darwinian adaptations.

The combination of organic selection and its effects have now become commonly known as the Baldwin Effect, even though Baldwin was only one among several authors who, more or less simultaneously, described the same phenomena.[34] Modern accounts of the Baldwin Effect are, however, far from uniform.[35] Although most accounts take organic selection to be the first step in the Baldwin Effect, there is less agreement about the nature of the accommodations on which this selection works. Nor is there consensus as to what place the effects of organic selection should be assigned, or how they relate to each other; it may at times be quite difficult to define exactly where one of these effects stops and another begins. I would therefore argue that the effects of organic selection should

[32] Depew, "Baldwin and His Many Effects," 6-7.
[33] *Dictionary*, s.v. "Weismannism," accessed 11-06-2019, URL: https://www.dictionary.com/browse/weismannism.
[34] Depew, "Baldwin and His Many Effects," 3-4.
[35] To take stock of the many ways the Baldwin Effect has been understood by different scholars, see: Depew, "Baldwin and His Many Effects," 3-31.

be seen as successive steps on a continuous path that starts with the occurrence of flexibility in a certain trait, and eventually leads to the increased inflexibility – or fixation – of this trait. This path could equally well be described as connecting individual development to the development of a whole population or species.

The arbitrary points of departure and arrival of this path coincide with the two 'transitions' that Peter Godfrey-Smith has identified as the most fundamental elements in any proper account of the Baldwin Effect: the first revolves around *plasticity*, the other around *genetic fixation*.[36, 37, 38] It is at this point in our inquiry that I will begin to use the word 'genetic' as referring to our contemporary word 'gene'. Before Godfrey-Smith's two transitions can be further explained, it is therefore helpful to equip ourselves with a more modern vocabulary and to deal with the incorporation of genetics into evolutionary biology. To obtain a better understanding of both plasticity and genetic fixation – and thus of the transition from ontogenetic to hereditary characteristics –, the mechanisms that underly heredity must be uncovered. It is to modern genetics that we must therefore now turn.

Plasticity and Genetic Fixation: How the Baldwin Effect Relates to Genetic Assimilation

The concept of heredity is so thoroughly integrated within both Darwin's theory of evolution by natural selection and the Baldwin Effect, that it is dangerously

[36] Peter Godfrey-Smith, "Between Baldwin Skepticism and Baldwin Boosterism," in *Evolution and Learning: The Baldwin Effect Reconsidered*, eds. Bruce H. Weber and David J. Depew (Cambridge, Massachusetts: The MIT Press, 2007), 54.

[37] Because the term 'plasticity' will prove to be a multi-faced concept in what is to follow, I will at this point not explicate whether it is to be understood as something that emerges as a reaction to environmental change, or as the basis for the very possibility of accommodations. The difference between these two possible interpretations of the word 'plasticity' will become apparent in the next chapter.

[38] Godfrey-Smith does not use the term 'genetic fixation' to describe the second transition. Instead, he uses the term 'genetic assimilation'. This term, however, has been used by many authors to refer to a range of slightly diverging phenomena and will be employed for different ends in the next chapter. To minimize confusion, I have therefore chosen a different name for Godfrey-Smith's second transition. This difference, however, is merely syntactic and not semantic: my 'genetic fixation' and Godfrey-Smith's 'genetic assimilation' refer to the same transition.

easy to forget that neither Darwin nor Baldwin were equipped with most of the basic conceptual tools to understand how this process works. Although much about it is still unknown, biologists can nowadays hardly think of the word 'heredity' without immediately picturing a gene. Genes, however, do have an obvious disadvantage: we cannot directly observe them. What we see are only their effects on the bodies and behaviour of relatively large organisms. Without including the hereditary nature of these effects, the idea of natural selection would simply not have been viable. Heredity thus had to be postulated, even though its underlying laws and mechanisms remained unknown. In *On the Origin of Species*, Darwin himself recognized that the mystery surrounding heredity formed a formidable lacuna in evolutionary theory:

> [t]he laws governing inheritance are quite unknown; no one can say why the same peculiarity in different individuals of the same species, and in individuals of different species, is sometimes inherited and sometimes not so […].[39]

When one can exclusively draw from observable varieties within populations, it is indeed hard to imagine why certain characteristics are inherited from mother to daughter, while others may at times skip several generations or never reoccur at all. To discover any useful patterns in this apparently capricious set of occurrences, a distinction between observable characteristics and some kind of additional 'underlying heredity-property' seems necessary. This is exactly the distinction that surfaces in Mendel's work on the laws of heredity.

Mendel was one of the first evolutionary theorists who thought of heredity in terms of discrete inherited units that give rise to observable physical characteristics. In postulating these units, he laid the foundation for one of the most fundamental distinctions in contemporary evolutionary biology: the distinction between the – to him still quite mysterious – units storing hereditary

[39] Darwin, *On the Origin of Species*, 21-22.

information, and the actual bodily expression of this information. Today, these different categories are respectively referred to as 'genotype' and 'phenotype'. Through a series of experiments, Mendel uncovered a numerical lawfulness in the inheritance of observable traits. This led him to the supposition that 'hereditary factors', or what we now call 'genes', underlie these observable characteristics.[40] This supposition would later play a crucial role in enabling other authors to explain why Lamarckian inheritance of acquired characteristics cannot work: if an individual acquires a certain characteristic or skill during its lifetime with which it was not born – say it learns to respond to a command or it loses a toe –, this trait will not be passed down to the individual's offspring, due to the lack of the necessary hereditary factors that underlie it. In contemporary terms, it is not a phenotypic trait that is hereditary, but its genetic foundation. It follows that those genes that encode for beneficial phenotypic traits are favoured by natural selection, while those genes that do not, will perish.

To understand how some genes can be favoured by natural selection at the expense of others, it is important to realize that a gene, according to today's understanding, has two fundamental and intimately intertwined characteristics: firstly, it makes copies of itself during reproduction and secondly, it controls the production of proteins. By means of proteins – via an immensely complex process which biologists and geneticists have only just begun to uncover – a gene is able to control chemical reactions within cells and thus guide the manufacture and conservation of a body.[41] In other words, the second important thing a gene does, is influence an organism's phenotype. This second defining characteristic is crucial, for without it a gene would be invisible to natural selection. Thanks to technological advancements, we are now able to observe that the bits of DNA hidden away within cell cores are all inconveniently similar. Without taking into account their phenotypic effects, there simply is not enough variety for natural selection to work on. A gene is thus favoured or penalized by natural selection on

[40] Carey, *Human Genetics*, 148.
[41] Dawkins, *The Selfish Gene*, 28-29.

account of its effects, not its intrinsic properties, and is successful if it manages to bring about beneficial effects.[42] These effects are not limited to the physical body of the organism, but also extend to its most intricate behavioural patterns: whether it is eye-colour or an instinct to hunt at night, any congenital characteristic we may observe in an organism is indirectly caused by its genes.

Distinguishing between an individual's genotype and phenotype also enables one to explain the above-mentioned observation that congenital traits sometimes skip several generations. The genetic tendency to develop a certain trait may be present, but this does not have to result in any phenotypic expression of these genes. Recessive genes, as opposed to dominant ones, can lay dormant within the body of an organism without losing their ability to be passed on.[43] If a dominant and a recessive gene carry hereditary information concerning the same phenotypic characteristic, the dominant gene will be phenotypically expressed at the expense of the recessive one. Moreover, even for a dominant gene there is no guarantee that it will ever make it into the phenotype, because this largely depends on environmental factors as well. If, for instance, a person possesses a dominant gene that makes her skin particularly prone to developing a tan in the sun, this in no way means she will actually develop a tan in her lifetime: were she to spend her whole life indoors, her skin would obviously remain pale and the genes for tanning would stay dormant. This flexible nature of phenotypic expression is actually implied by the definition of the word 'phenotype', given in the Merriam-Webster dictionary: *"*the observable properties of an organism that are produced by the interaction of the genotype and the environment.*"*[44] This definition exclusively highlights the importance of the interaction with the environment, because it only mentions the organism's *whole* genotype. However, once we look at the potential expression of one particular gene – be it of the recessive or dominant kind –, it becomes clear that its interaction with the other genes within

[42] Dawkins, *The Selfish Gene*, 303-304.
[43] Ibid., 32.
[44] *Merriam Webster*, s.v. "Phenotype," accessed 07-03-2019, URL: https://www.merriam-webster.com/dictionary/phenotype.

the genotype is just as relevant. These two interactions combined explain why some traits can seemingly skip several generations. Note, however, that the terms and distinctions used in the previous sections do not directly derive from Mendel: he only described the regularities he observed in the inheritance of phenotypic characteristics, and deduced hypothetical hereditary units from these observations. This notwithstanding, his work *did* provide several basic insights into the workings of heredity, which in turn has elegantly complimented and reinforced the theory of evolution by natural selection.

Now that our vocabulary has been enriched, we can take a closer look at the aforementioned two transitions that Godfrey-Smith has identified as fundamental in describing the Baldwin Effect. The first transition takes place when certain individuals are confronted with environmental change and start to make phenotypic accommodations as a consequence. Crudely stated, this is a transition from a static to a plastic condition, which takes place at the level of the individual organism. It is thus referred to as *plasticity*. The second transition takes place when a new phenotypic trait becomes fixed within the genotypes of individuals in later generations and thus loses its dependency upon environmental inducement or learning processes. This is referred to as *genetic fixation*, and can be thought of as a reverse transition from a plastic to a static condition, with the difference that this transition takes place over multiple generations and not at the level of a single individual.[45]

As was already mentioned, both of Godfrey-Smith's transitions are included in the notion of the Baldwin Effect. There are, however, at least two other processes that encompass a similar set of transitions, and which have for this reason occasionally been equated with the Baldwin Effect. To make matters even more confusing, these two alternative processes have both been referred to as 'genetic assimilation', which gravely obscures the fact that they differ from one another. The upshot of this is that the term 'Baldwin Effect' can nowadays refer to at least three theorems: to Baldwin's original theory of organic selection and its

[45] Godfrey-Smith, "Between Baldwin Skepticism," 55.

effects, and additionally to either one of two distinct understandings of genetic assimilation.[46] To disembroil this tangle of terms, I will first introduce both conceptions of genetic assimilation. These conceptions will then be linked to Godfrey-Smith's two transitions, which will uncover that they both entail somewhat different understandings of these transitions. This is what makes both types of genetic assimilation relevant to our current inquiry: identifying the Baldwin Effect with the first type, will elucidate several crucial aspects of Baldwin's ideas, but it will leave others quite underexposed. Diving into the second type, however, will clarify these other aspects, which will supplement the clarifications derived from our consideration of the first type. The combination of the two types of genetic assimilation will thus provide us with a comprehensive description of the Baldwin Effect in modern terms, which will in due course enable us to compare Baldwin's original ideas with the modern Genocentric Perspective. Since genetic assimilation was originally introduced by Conrad Hal Waddington in 1953, his conception of it will be first in line.[47]

According to Waddington, genetic assimilation entails the loss of an acquired trait's dependency on environmental triggers. He advanced the view that there exists variety in an organism's genotype, which renders some individuals more prone than others to express this variety in their phenotypes under the right set of conditions. When an organism is exposed to the right environmental 'trigger' during its development, a trait that was at first only present potentially can become present actually. Natural selection can then favour the previously unexpressed genetic variety, which will eventually result in the spread of this variety through the population.

To test his hypothesis, Waddington exposed a strain of fruit flies to severe heat shock. Under normal circumstances, these flies develop cross-veins in their

[46] Stephen M. Downes, "Baldwin Effects and the Expansion of the Explanatory Repertoire in Evolutionary Biology," in *Evolution and Learning: The Baldwin Effect Reconsidered*, eds. Bruce H. Weber and David J. Depew (Cambridge, Massachusetts: The MIT Press, 2007), 40.
[47] J. Masel, "Genetic Assimilation Can Occur in the Absence of Selection for the Assimilating Phenotype, Suggesting a Role for the Canalization Heuristic," *Journal of Evolutionary Biology* 17, no. 5 (2004): 1106.

wings, connecting the larger veins to one another. When exposed to heat shock, however, some 40% of the flies developed a cross-veinless phenotype. Waddington then selected for cross-veinless phenotypes under heat shock conditions, with the result that the percentage of flies that developed a cross-veinless phenotype under heat shock rose with each generation. After several generations, cross-veinless phenotypes started to appear without the occurrence of heat shock; the phenotypic accommodation – which we would call an ontogenetic accommodation if we projected Baldwin's terms onto Waddington's observations – had lost its dependency upon its environmental trigger and had become wholly integrated within the set of congenital characteristics of the relevant strain of flies.[48]

Waddington explained the emergence of cross-veinless phenotypes under normal circumstances in the following manner: he figured that the hidden genetic basis for the development of a cross-veinless phenotype had to consist of a multitude of genes, all contributing to the occurrence of this phenotypic effect.[49] Moreover, in order to be phenotypically expressed, a certain frequency of these contributing genes had to be present. Under normal circumstances, this frequency did not reach the threshold for expression to occur. During heat shock, however, the threshold for expression was lowered. As a result, a lower frequency of genes now sufficed to cause the phenotypic accommodation. Subsequent selection of those flies that developed cross-veinless phenotypes under heat shock conditions then increased the frequency of cross-veinless genes in the population. This continued for several generations, until the frequency was high enough to breach the higher threshold for the development of a cross-veinless phenotype in individuals that were only exposed to normal conditions. Genetic assimilation had

[48] Masel, "Genetic Assimilation," 1106.
[49] Brian K. Hall, "Baldwin and Beyond: Organic Selection and Genetic Assimilation," in *Evolution and Learning: The Baldwin Effect Reconsidered*, eds. Bruce H. Weber and David J. Depew (Cambridge, Massachusetts: The MIT Press, 2007), 146.

taken place.[50] With reference to its originator, I will call this theorem *Waddington's Genetic Assimilation*.

The second conception of genetic assimilation is quite different: on this view, genetic assimilation takes place as a result of fortuitous genetic mutations that confirm purely phenotypic traits that originally had to be learned or otherwise triggered.[51] Because these traits, as long as they are sufficiently beneficial, can be maintained within the population through social learning or imitation, the population is potentially provided with enough time to 'hit upon the right mutation'. Genetic assimilation of a particular trait would in this case occur when new genes are produced that encode for said trait.[52] In other words, a so-called 'genocopy' of a phenotypic trait has to be produced. Selection then favours this genocopy, which spreads through the population until the once purely phenotypic trait no longer has to be environmentally induced or learned. Genetic assimilation will then have taken place. I will call this, for want of a more catchy term, *Mutation-based Genetic Assimilation*.[53]

Plasticity and genetic fixation are present in both forms of genetic assimilation, but they do differ from each other in the two particular cases. For starters, the forms of plasticity involved in Waddington's Genetic Assimilation and Mutation-based Genetic Assimilation are not equivalent. As Godfrey-Smith himself notes: "the kinds of plasticity involved in the early rounds of Waddington's experiments have only a very superficial analogy with the mechanisms enabling adaptive plasticity of the kind seen in learning."[54] Waddington's experiments uncovered an already present genetic tendency as the

[50] Masel, "Genetic Assimilation," 1106.
[51] Note that, although authors such as D. C. Dennett, G Hinton and S. Nowlan have appealed to this alternative understanding of genetic assimilation it in their work, it does not originate from Waddington's own writings.
[52] Downes, "Baldwin Effects," 41.
[53] It should be noted that the boundary between these two kinds of genetic assimilation may not always be clear, mainly due to the fact that many traits are developed under the direction of many genes. This caveat will be discussed further in the discussion, along with the observation that Baldwin's categories may blend into each other. For now, however, all these somewhat arbitrary categories can help us to work towards a substantive conclusion.
[54] Godfrey-Smith, "Between Baldwin Skepticism," 59.

origin of the acquired traits that were being genetically fixed in later stages.[55] The plasticity that forms the basis of Mutation-based Genetic Assimilation does not amount to the uncovering of such a genetic tendency, for it includes a wide range of accommodations that are not genetically based. The social inheritance of traits and the emergence of new habits through the performance of contingent actions by individuals presuppose a plasticity of a completely different order.

Because both conceptions of genetic assimilation start out from a different kind of plasticity, the way the subsequent genetic fixation takes place also differs in the two cases. When the genes that encode for a certain trait are already present in dormant form, as is the case with Waddington's Genetic Assimilation, natural selection can favour these genes once they have been brought out into the phenotype. The continuation of this process then increases the frequency of these genes, until genetic fixation has taken place. In the case of Mutation-based Genetic Assimilation, however, genetic fixation rather depends upon a genetic basis 'catches up' in later generations with the already established accommodations, in the form of a genocopy. Only once this genocopy has been produced does natural selection get a chance to favour the trait. Considering this difference in the second transition, it might be argued that the term 'genetic fixation' is much more applicable to the second phase of Waddington's Genetic Assimilation than to the second phase of Mutation-based Genetic Assimilation. After all, in the former case, the traits are truly being genetically 'fixed' in the sense that their basis will surely result into phenotypic expression, the possible absence of environmental inducement notwithstanding. In the case of Mutation-based Genetic Assimilation, genetic *acquirement* might actually be a better word, since the genetic basis only first originates during the second transition. Not only the trait, but also the genes needed for its fixation are quite literally acquired when the right mutation occurs. Nevertheless, I will maintain the established terminology in order to avoid needless complications.

[55] Downes, "Baldwin Effects," 40.

All in all, when Godfrey-Smith's two transitions involved in the Baldwin Effect are combined with the two conceptions of genetic assimilation, the following scheme can be composed:

	Waddington's Genetic Assimilation	**Mutation-based Genetic Assimilation**
Transition 1: Plasticity *Takes place within an individual organism's lifetime (ontogeny)*	Environmental trigger causes an already present genetic variety to be expressed in the individual's phenotype	Individual makes phenotypic accommodation that is not genetically based, which can be passed on via non-genetic routes like imitation or social learning
Transition 2: Genetic fixation *Takes place over the span of multiple generations (phylogeny)*	Natural selection favours genes that enable individuals to make the phenotypic accommodation when the environmental trigger is present, causing the frequency of these genes to rise within the population until the threshold for phenotypic expression under normal conditions is breached. The trigger is then no longer needed.	The phenotypic accommodation is kept in existence via non-genetic routes, providing time for the occurrence of fortuitous mutations within the population that lead to the same phenotypic accommodation without the need for environmental trigger or learning. This 'genocopy' is then favoured by natural selection and spreads through the population.

Hitherto I have only analyzed the Baldwin Effect – and processes closely resembling it –, whilst avoiding any mention of my research question's second prominent component: the Genocentric Perspective. The distinctions made in the

scheme above, however, provide us with a conceptual toolbox sufficient to connect the Baldwin Effect with the Genocentric Perspective. As has been shown before, both distinct forms of genetic assimilation have been used interchangeably with the Baldwin Effect. One author who does so quite explicitly is Richard Dawkins.[56] He understands the Baldwin Effect as equivalent to Waddington's Genetic Assimilation and even refers to it as the 'Baldwin/Waddington Effect'.[57] This is hardly surprising, as Dawkins is one of the most eminent representatives of the Genocentric Perspective on evolution, and it makes perfect sense in this perspective to understand the Baldwin Effect as Waddington's Genetic Assimilation.[58]

Before assessing whether the Baldwin Effect, as originally conceived by Baldwin, can be equated with either form of genetic assimilation without any loss of meaning to the concepts involved, I will take a side-track into the Genocentric world of Dawkins and explain why it is perfectly consistent within this world to understand the Baldwin Effect as Waddington's Genetic Assimilation. After all, if we can understand why this conception of the Baldwin Effect logically results from the Genocentric Perspective, we will eventually be able to judge the latter's suitability to represent the former in its entirety.

[56] Downes, "Baldwin Effects," 40.
[57] Richard Dawkins, *The Extended Phenotype: The Long Reach of the Gene* (Oxford: Oxford Landmark Science, 2016), 440.
[58] I have chosen the work of Richard Dawkins as the source on which to base my discussion of the Genocentric Perspective. I realize that Dawkins is neither the first, nor the only advocate of this view on evolution and that his account of it is largely built upon the work of authors like Henri Bergson (whose work was already identified as one of the first steps towards a gene-centered biology in the introduction), John Maynard Smith, George C. Williams, and W.D. Hamilton. However, I believe it is safe to say that Dawkins' work is most frequently associated with the Genocentric Perspective, which at least partly justifies my narrow treatment of the literature on this subject. Additionally, providing a full overview of the development of the Genocentric Perspective with the contributions of all its most prominent advocates, would be vastly beyond the scope of this thesis.

The Genocentric Perspective: What the Baldwin Effect Looks Like Through the Eyes of Genes

There exists a considerable amount of literature devoted to analysing the Baldwin Effect, but Dawkins' work can hardly be counted among this. In his book *The Extended Phenotype*, he only mentions the Baldwin Effect in passing and adds his definition of it in the glossary. Brief as his reference to the Baldwin Effect may be, however, the way he names and defines it is quite telling within the context of our current inquiry:

> **Baldwin/Waddington Effect** […] A largely hypothetical evolutionary process (also called *genetic assimilation*) whereby natural selection can create an illusion of the inheritance of acquired characteristics. Selection in favour of a genetic tendency to acquire a characteristic in response to environmental stimuli leads to the evolution of increased sensitivity to the same environmental stimuli, and eventual emancipation from the need for them. [I have previously suggested] that we might breed a race of spontaneously milk-producing male mammals by treating successive generations of males with female hormones and selecting for increased sensitivity to female hormones. The role of the hormones, or other environmental treatment, is to bring out into the open genetic variation which would otherwise lie dormant.[59]

It is rather obvious how to correctly place this Baldwin/Waddington Effect within the scheme drawn above, and not only because of its revealing double name. In the first place, it can be noted that both of Godfrey-Smith's transitions are readily recognizable: the acquirement of a characteristic in response to environmental stimuli corresponds to an individual showing plasticity in the face of duress, while the eventual emancipation from these stimuli can be reformulated in terms of

[59] Dawkins, *The Extended Phenotype*, 440.

genetic fixation over the course of multiple generations. Secondly, the *types* of plasticity and of fixation Dawkins employs both fully belong in the column of Waddington's Genetic Assimilation: an already present genetic tendency is explicitly identified as the basis for the acquired characteristic and the eventual emancipation from environmental stimuli could just as well have been formulated in terms of emancipation from environmental triggers due to an increase in gene frequencies. Dawkins' lactation example, in which he mentions a 'dormant genetic variation' that is brought out into the open, confirms this once more. He thus not only understands genetic assimilation in the original Waddingtonian sense, but he also defines the Baldwin Effect as equivalent to it. From this it follows that to Dawkins, the Baldwin Effect is a process in which the incentive for phenotypic accommodations lies with the individual's genes, and is not to be found in the individual's actions or deliberations. To fully grasp the implications of this interpretation, we need to familiarize ourselves with its genocentric foundation. In other words, we must try and fathom what it means to look at evolution from the perspective of genes.

How adopting the Genocentric Perspective can influence one's conception of Darwinian evolution becomes evident when we compare Dawkins' definition of natural selection with a more conventional one. The definition listed in the Merriam-Webster dictionary, for instance, reads as follows:

> [natural selection]: a natural process that results in the survival and reproductive success of individuals or groups best adjusted to their environment and that leads to the perpetuation of genetic qualities best suited to that particular environment.[60]

Note that in this definition the survival of individuals is mentioned first, and that the reproductive success of those individuals best adjusted to their environment is

[60] *Merriam Webster*, s.v. "Natural Selection," accessed 16-05-2019, URL: https://www.merriam-webster.com/dictionary/natural%20selection.

said to *lead* to the survival and spread of adaptive genetic qualities. This implies that an individual's adaptations are, first and foremost, to be seen as beneficial to the individual, and that the perpetuation of the underlying genes is merely a result of the fact that the individual benefits from the adaptations. The legacy of an organism-centred biology shines through here: the discovery of genes has taught us that an individual's congenital characteristics are strictly passed down via genetic means, but it is still quite intuitive to see these units of heredity as entities that gracefully carry encoded information across the generations, like microscopic vehicles for the blueprints of our bodies. Formulated differently, the definition above seems to concord with the idea that genes are a means to an end.[61] Dawkins' definition, however, implies something altogether different:

> [n]atural selection is the process by which replicators change in frequency in the population relative to their alleles.[62]

In Dawkins' definition, there is no explicit mention of the individual at all, nor is there any indication that the individual organism should be seen as the entity for whose benefit adaptations arise. Instead, he characterizes natural selection as the differential survival and consequent frequency changes of so-called *replicators*. The term 'replicator' refers to any entity of which copies are made during reproduction. Over the course of multiple generations, some replicators will increase in number at the expense of alternative replicators that are not copied.[63] According to Dawkins, only an entity suited to fulfil the role of replicator should be identified as the proper *unit of selection*, the unit on which natural selection works. It is with the help of this notion that he challenges the centrality of the individual, for it enables him to ask whether an individual organism can be

[61] Dawkins, *The Selfish Gene*, 306-307.
[62] Dawkins, *The Extended Phenotype*, 135.
[63] Ibid., 82-83.

assigned the role of replicator: does natural selection actually work on individuals, or should the role of replicator be assigned to some other entity?[64]

Dawkins' answer to this question was already foreshadowed in the brief discussion of the two fundamental characteristics of genes, back in chapter 2: a gene is an entity that is copied during reproduction and that has the ability to influence the phenotype of the body in which it sits. To see why the first of these two characteristics makes the gene a good candidate to take on the role of replicator, it must be clarified what Dawkins means by 'individual genes' that replicate themselves. He does not use the word 'gene' to refer to a well-outlined entity that can never be segmented – which in fact can happen due a phenomenon called 'crossing over' that occurs during sexual reproduction, or due to mutations. Instead, he provides a so-called 'fading-out definition', which characterizes the gene as:

> […] a genetic unit that is small enough to last for a large number of generations and to be distributed around in the form of many copies. […] The more likely a length of chromosome is to be split by crossing-over, or altered by mutations of various kinds, the less it qualifies to be called a gene in the sense in which I am using the term.[65]

Genes can thus be said to function as replicators in so far as they are preserved long enough to qualify as such. This way of defining the gene enables us to dismiss many other candidates for the role of replicator. For example, entire genomes do not have the potentially long futures that genes do. During sexual reproduction, individuals exchange genetic information and the genes that make up the genome of the new individual are reshuffled into a novel and unique combination. Hence, although it may not always be clear whether a certain length of DNA qualifies as

[64] Dawkins, *The Extended Phenotype*, 82.
[65] Dawkins, *The Selfish Gene*, 41.

a replicator, we can at least assert with certainty that complete genomes are not long-lasting enough to qualify.

Or can we? The phenomenon of asexual reproduction, during which an organism's entire genome is replicated, forms an exception to this rule.[66] For instance, asexual stick-insects reproduce without exchanging genetic information with other individuals, which indeed renders their genomes fit to bear the name of replicator. This, however, most certainly does not mean that the individual stick-insects *themselves* are replicators! The body of a stick-insect is not formed as a replica of its parent's body – identical as it may be to it –, but the genome that directs the building process of this body is moulded as a replica of the parent's genome. This is why, if we were to remove a leg from the parent stick-insect, its offspring would obviously not be born with a missing leg.[67] Whole organisms are thus unsuited for the role of replicator – including those organisms whose entire genomes can be called replicators – and should therefore not be seen as the unit of selection.[68]

The potentially long-lasting nature of the gene is not enough to render it suitable to serve as the unit of selection. Its second ability – influencing the phenotype – is just as important, for genes are favoured or penalized because their effects are, respectively, beneficial or disadvantageous. But beneficial or disadvantageous to whom? For whose benefit do genes exert their adaptive phenotypic effects?[69] In Dawkins view, unsurprisingly, there is only one possible answer: the gene is the entity for whose benefit the adaptations that emerge through natural selection exist.[70] He clarifies this by stressing that the gene is the only entity whose *utility function* can explain the existence of every possible adaptation in nature.

---

[66] *Biology Dictionary*, s.v. "Asexual Reproduction," accessed 26-06-2019, URL: https://biologydictionary.net/asexual-reproduction/.
[67] Dawkins, *The Selfish Gene*, 366-367.
[68] Ibid., 43-45.
[69] Ibid., 124-125.
[70] Ibid., 14.

The term 'utility function' – originally used in economics – means 'that which is maximized'.[71] Dawkins uses this term in the context of evolutionary biology as follows: when we observe a certain trait, we might ask ourselves why it is there. Why does the flying squirrel have membranes between its wrists and ankles? Why does the owl have claws sophisticated enough to grasp the flying squirrel in mid-air? According to Dawkins, all questions of this type can be reformulated in terms of utility function: what commodity is being maximized by the existence of this trait?[72] On the face of it, the squirrel's membranes and the owl's claws might be supposed to serve opposite ends and therefore to maximize opposite commodities – respectively, squirrel survival chances and the number of squirrel deaths. Yet despite the opposite nature of the ends that are being pursued, the underlying genes of both these traits increase their own chances of being passed down in causing the individuals in which they sit to pursue these ends. Consequently, a gene that succeeds in increasing its own chances of being passed down, will in all likelihood increase in frequency within the population. Phenotypic effects should thus always be explained with reference to the quantitative maximization of the genes which cause them, a conclusion about which Dawkins leaves no doubt:

> [t]he great universal Utility Function, the quantity that is being diligently maximized in every cranny of the living world is, in every case, the survival of the DNA responsible for the feature you are trying to explain.[73]

The idea that successful genes exert phenotypic effects that facilitate their own quantitative maximization thus substantiates the statement that the gene is the entity for whose benefit adaptations exist. This means that whether a particular gene's influence is beneficial to the individual organism in which it happens to sit

[71] Richard Dawkins, *River out of Eden: A Darwinian View of Life* (London: Weidenfeld & Nicolson, 1995), 103.
[72] Ibid., 105-106.
[73] Ibid., 120.

or not, is of no consequence. Any benefits to the organism – phenotypic effects that increase its chances of survival and reproduction, which can lead to the evolution of an adaptative trait – are to be understood as the additional outcomes of the fact that these effects are beneficial to the proliferation of underlying genes, not as the cause of the genes' proliferation. Simply put, it is the quantitative maximization of genes that causes the evolution of adaptive traits, and not the evolution of adaptive traits that causes certain genes to be maximized in quantity. As counterintuitive as it may appear, the central question in the Genocentric Perspective on evolution should thus *not* be how a gene's phenotypic effect benefits the organism on which it is exerted. Instead, we should ask ourselves: *how does the phenotypic effect the gene exerts on an individual organism benefit the gene's own replication and consequent quantitative maximization?*

At this point, a complication seems to arise: most traits are not developed under the direction of a single gene. Instead, they are *polygenic*, which means they are controlled by multiple genes.[74] Now if most traits are polygenic, and their *raison d'être* is the proliferation of their underlying genes, it seems that we should ask how the existence of the trait benefits *all* of these genes as a cooperating group. Yet we have seen that genes get reshuffled during sexual reproduction, which makes it possible that cooperating genes will get separated. This separation might leave single genes incapable of producing the effects that are beneficial to their own proliferation. Would it therefore not be more accurate to assert that cooperating groups of genes are favoured or penalized by natural selection, instead of single genes?

Well, not necessarily. Any single gene that makes but a modest contribution to the development of an adaptive trait still increases its own chances of replication through this effect. Suppose thirty different genes work together to develop a certain trait. If and when this trait increases the individual's chances of reproduction, all thirty of the individual genes involved will get to cash in.

[74] *Biology Dictionary*, s.v. "Polygenic Traits," accessed 17-06-2019, URL: https://biologydictionary.net/polygenic-traits/.

Moreover, it is likely that these gene complexes, once formed, will be found in a large number of individuals in subsequent generations. After all, a single gene that works best if it is present in combination with the other genes with which it encodes for a trait will be replicated more often if it finds itself in that combination. If the genes that encode for a particular adaptive trait lose some of their collaborators – and if this prevents the trait to be efficiently developed – all of the genes involved will have a smaller chance of replication. The collaborating genes to which this does not happen *do* maintain their high replication chances through their joint effects. Hence, groups of collaborating genes will tend to survive, even though there is no intrinsic property that causes them to be favoured *as a group* in their journey down the generations. They simply tend to stick together because this is what is best for the numerical maximization of each and every *single* gene.[75]

What it means to look at evolution from the Genocentric Perspective, is thus to adopt a fundamentally different way of explaining phenotypic traits, due to the replacement of the individual as the unit of selection by the gene. Still, if the individual organism does not feature as the unit of selection, what role does it play in the drama of evolution? This is where Dawkins turns the abovementioned picture – that of genes as the vehicles for hereditary information – completely upside-down. According to him, it is the organism that quite literally plays the role of vehicle, not the gene. An organism, Dawkins argues, is nothing more than a complicated survival-machine, built by its genes to increase their own numbers in the genepool.[76] Such a survival-machine can be regarded as a temporal shell, programmed to preserve and propagate the genes that ride inside it.[77] Each time the genes replicate their way into the next generation, these temporal vehicles are cast aside and replaced. This means that any dents, gadgets or new tricks these vehicles may pick up in the course of their lifetimes will be lost once their passengers have built themselves a fresh carriage:

[75] Dawkins, *The Selfish Gene*, 110-111.
[76] Ibid. 24-25.
[77] Dawkins, *The Extended Phenotype*, 452.

> [g]enes do indirectly control the manufacture of bodies, and the influence is strictly one way: acquired characteristics are not inherited. No matter how much knowledge and wisdom you acquire during your life, not one jot will be passed on to your children by genetic means. […]. *A body is the genes' way of preserving the genes unaltered* [italics mine].[78]

In this last sentence, Dawkins' reverse way of conceptualizing the status of both individuals and genes becomes especially evident. The individual vehicle gets assigned the role of means to an end, while the gene is firmly established as the entity around which evolutionary development revolves.

It can now be grasped why a conception of the Baldwin Effect as Waddington's Genetic Assimilation naturally follows from adopting the Genocentric Perspective. We have seen that this perspective corresponds to the idea that the existence of every trait can be explained by disclosing how this trait benefits the replication of its underlying genes. Now if we apply this explaining-strategy to a trait that has started out as an ontogenetic accommodation, it is clear what kind of plasticity and subsequent genetic fixation need to be presupposed: the ontogenetic accommodation needs to be understood as having an initially unexpressed genetic foundation, otherwise the fundamental genocentric question could not be asked. After all, without postulating an underlying genetic foundation, there would not be anything to increase in quantity as a consequence of the phenotypic expression of this foundation. The Genocentric Perspective thus gives rise to an interpretation of the Baldwin Effect as a process in which the incentive for ontogenetic accommodation lies with replicators, not vehicles. With this conceptual framework in the back of our heads, we are now in a position to assess to what extent the Genocentric Perspective is adequate to describe the Baldwin Effect. In order to do so, we must return to the phenomena that Baldwin originally described, and consider how they relate to the concepts involved in the

[78] Dawkins, *The Selfish Gene*, 29.

two forms of genetic assimilation that were distinguished. This will enable us to judge whether understanding the Baldwin effect as Waddington's Genetic Assimilation actually suffices to incorporate all the intricate components of Baldwin's original theorem.

Back to Baldwin-basics

Recall that the Baldwin Effect has been described as the combination of organic selection within three types of ontogenetic accommodations on the one hand, and the effects of this selection on the evolutionary development of the relevant species on the other hand. We are now able to see that it is not as simple as connecting these respective phases to Godfrey-Smith's plasticity and genetic fixation, since both these latter terms may refer to different concepts. I will argue that, in order to represent all the accommodations identified by Baldwin, we need both the kind of plasticity involved in Waddington's Genetic Assimilation, and the kind that is involved in Mutation-based Genetic Assimilation. Additionally, since these different kinds of plasticity give rise to different forms of genetic fixation, it follows that we can exclude neither of the two forms of the second transition. What this means for the adequacy of the Genocentric Perspective to describe the full Baldwin Effect will become clear as the argument unfolds.

Let us begin by pairing the distinct kinds of plasticity to their corresponding types of accommodation. For starters, a clear analogy can be drawn between Baldwin's physico-genetic accommodations and the type of plasticity on which Waddington's Genetic Assimilation is based. With physico-genetic accommodations, an organism simply has the potential to make an accommodation and if the right environmental trigger is present, the accommodation will inadvertently and mechanically be made. This mechanism is explicable once the existence of an underlying genetic basis is postulated, and can therefore be paralleled to the conclusions Waddington drew from his experiments on fruit flies: the flies that developed a cross-veinless phenotype when exposed to

heat shock did so because they possessed the right underlying genetic variety. Had the heat shock not been there, however, not even these flies would have developed a cross-veinless phenotype. Moreover, the fact that both authors refer to the same kind of trigger to induce the changes they describe, further accentuates the analogy between them. Baldwin explicitly mentions temperature changes when he lists the environmental influences that could potentially cause physico-genetic accommodations and Waddington uses exactly this kind of influence in his experiments. Unsurprisingly, considering that Dawkins understands the Baldwin Effect as Waddington's Genetic Assimilation, a similar analogy can be drawn between Dawkins' and Baldwin's environmental triggers: chemical agents are listed among Baldwin's potential causes for physico-genetic accommodations as well, and Dawkins uses hormones as the triggers for accommodation in his lactation-example.[79] It seems plausible to suppose that the hormones Dawkins mentions could with hindsight be grouped under the heading of Baldwin's 'chemical agents'. All these considerations substantiate my claim that the plasticity of Waddington's Genetic Assimilation can account for the class of physico-genetic accommodations.

If Baldwin's phase of organic selection only encompassed physico-genetic accommodations, the type of plasticity invoked by both Waddington and Dawkins would suffice to give a comprehensive description of it. However, to also account for Baldwin's neuro- and psycho-genetic accommodations, we seem to require the plasticity involved in Mutation-based Genetic Assimilation. As mentioned, this plasticity includes accommodations that are purely phenotypic and can be maintained in a population through non-genetic processes like learning and imitation. Here it must be stressed that when an accommodation is said to be 'purely phenotypic' this does *not* mean that it has no ties of any kind to the genotype. Rather, what is meant is that such phenotypic accommodations cannot be mapped directly onto a specific genetic basis, and that their occurrence must thus not be reduced to the mechanical expression of underlying genes. It is

[79] Baldwin, "A New Factor," 2.

probably safe to assume that when an individual acquires a new skill by imitating various muscular movements, it harbours genes that enable it to adequately imitate these movements. However, to suppose that this individual already possessed the genes that encode for this specific skill *itself* – which would be necessary to facilitate its direct inheritance – would be one step too far. It would be like supposing that, when a starling learns to open the lid of a milk bottle, the presence of this bottle and a dormant tendency to open bottles must automatically and inadvertently lead to the expression of this exact skill, without the need for neurological or psychological mediation. This is a power that even a convinced advocate of the Genocentric Perspective like Dawkins would not grant the gene. After all, if genes could control the ontogenetic development and behaviour of their vehicles in such a direct manner, why would they need to build such complicated nervous systems and brains at all? For these reasons, only postulating a plasticity based on a pre-existing genetic tendency is not enough to represent all of Baldwin's classes of accommodation; the plasticity on which Mutation-based Genetic Assimilation relies is needed as well.

Does this mean that the second type of plasticity is inclusive enough to represent all three of Baldwin's classes of accommodation? Well, not likely; although the plasticity concept of Mutation-based Genetic Assimilation can adequately represent both neuro-genetic and psycho-genetic accommodations, it cannot be matched with the passive and unmediated nature of physico-genetic accommodations. To do this, one would have to suppose that accommodations that are inadvertently and mechanically made when the right environmental trigger is present, somehow still depend upon neurological or psychological mediation. This would come dangerously close to the idea that if a male mammal is injected with female hormones, whether he will develop the ability to lactate will be determined by his own subsequent actions and deliberations. It would imply that a fruit fly with the right genetic variant to develop a cross-veinless phenotype might, through its own actions, prevent this development when it is exposed to heat-shock – and, for that matter, that humans could determine whether

their skin tans when it is exposed to sunrays, or not. Giving such a degree of control to the individual does not comply with the plausible idea that genes sometimes do determine how we accommodate, without needing any assistance from our actions and deliberations. Only postulating plasticity in the sense of phenotypic accommodations that cannot be mapped onto a genetic basis, is therefore not enough to represent all of Baldwin's classes of accommodation either.

These considerations concerning plasticity have profound implications for the way genetic fixation should be seen. Of course, Baldwin himself did not distinguish between different kinds of fixation; he only provided a rough sketch of the process through which ontogenetic accommodations could become hereditary over time, but whether this happened because pre-existing genes increased in frequency, or a genetic basis was newly produced in later generations cannot be deduced from his pre-genetic considerations. In other words, Baldwin gives us no positive reason to identify the effects of organic selection with one specific form of genetic fixation. This notwithstanding, what *can* be deduced from Baldwin's findings is the following: because we would have to make do with too narrow a concept of plasticity if we equated the Baldwin Effect with Waddington's Genetic Assimilation, the genetic fixation that belongs to this type of assimilation would also be insufficient. After all, once accommodations are included that do not initially rely upon a pre-existing genetic basis, the emergence of genocopies needs to be postulated in order for these accommodations to become genetically fixed. The second transition in the Baldwin Effect thus cannot simply be identified with the second transition involved in Waddington's Genetic Assimilation. From this, it inevitably follows that, if one equates the Baldwin Effect with Waddington's Genetic Assimilation, one can only do justice to part of the phenomena Baldwin described.

When applied in reverse, the same logic dismisses the identification of the Baldwin Effect with Mutation-based Genetic Assimilation; the only plasticity one would then have at one's disposal could not possibly result in genetic fixation of

the kind supposed by Waddington. There would be no genetic basis to be brought out into the phenotype, and so no possibility of a trait becoming genetically fixed through an increase in quantity of the relevant genes. What follows, is that the need to include both types of plasticity results in the impossibility to commit to only one form of genetic fixation. The upshot of all of the above is that the Baldwin effect cannot be understood as the mere equivalent of Waddington's Genetic Assimilation, or of Mutation-based Genetic Assimilation.

At this point, it may seem as if the question central to this thesis can be answered without further ado: the Genocentric Perspective can be considered sufficient to describe the Baldwin Effect to the extent that it implies the identification of this effect with Waddington's Genetic Assimilation, but this identification *itself* will not do because the plasticity and fixation concepts it entails are not inclusive enough. Ergo, the Genocentric Perspective is insufficient to describe the Baldwin Effect in its entirety. However, it would be somewhat premature – not to mention uncharitable towards the Genocentric Perspective – to draw this conclusion already here. The fact that the Genocentric Perspective results in an understanding of the Baldwin Effect as based upon the plasticity of Waddington's Genetic Assimilation, does not mean that this perspective leaves no room for the plasticity associated with Mutation-based Genetic Assimilation. In fact, Dawkins himself would fully agree with the statement that neurological and psychological processes can, and often do, mediate between genes and an individual's behaviour and ontogenetic development. He stresses that the influence genes exert upon organisms can be quite indirect – especially when the organism in question possesses a nervous system that includes a highly developed brain.[80]

Genes contain information to steer the process of building a brain during embryotic development, but their influence is finite. They cannot possibly encode the appropriate neurological or psychological reaction to every possible situation, nor can they directly intervene; they can only provide a set of guidelines for the

[80] Dawkins, *The Selfish Gene*, 63-64.

brain to act upon, but the actions themselves are, to a greater or lesser extent, initiated by the brain itself.[81] As soon as a brain has been developed, it becomes the executor that has to deal with a highly contingent world by making predictions about that world. It is thus in the best interest of genes to provide their vehicles with a brain that is capable of doing this as accurately as possible. One way for them to achieve this, is to equip this brain with the capacity to learn new habits and skills. Dawkins proposes that genes may 'program' a brain to actively and formatively interact with its environment, for instance by inducing it to repeat actions with positive results and to avoid actions that have in the past proved detrimental.[82] In other words, Dawkins recognizes that genes may provide their vehicles with an organ capable of quite sophisticated plasticity – plasticity in the sense on which Mutation-based Genetic Assimilation relies. However, to be able to speak of the Baldwin Effect, the characteristics that spring from this plasticity must become hereditary through some form of genetic fixation. This is where the fact that the Genocentric Perspective acknowledges both forms of plasticity begins to truly complicate matters: in order for a purely phenotypic accommodation to become genetically fixed, a genocopy must emerge in the population. This means that the acquisition of the accommodation in question *precedes* the acquisition of the genetic basis necessary to make the accommodation hereditary. It will come as no surprise that this is problematic for the Genocentric Perspective, but to really grasp why this is so, several notions introduced in the previous chapter need to be revisited.

Recall that it is essential to the Genocentric Perspective that the existence of an adaptive trait is explained by answering the question as to how this trait benefits the quantitative maximalization of its underlying genes. We have seen that when this type of explanation is applied to traits that are acquired during ontogeny, it automatically implies a plasticity in which a genetic tendency is already present. However, since the Genocentric Perspective also embraces the

[81] Dawkins, *The Selfish Gene*, 66-71.
[82] Ibid., 73.

acquisition of traits without a pre-existing genetic basis, it becomes quite unclear how to consistently answer the fundamental genocentric question *once such a trait has become genetically fixed*. Indeed, it is doubtful whether the Genocentric Perspective can make sense of this kind of plasticity leading to genetic fixation at all.

To elucidate this doubt, consider the following hypothetical scenario: within a certain population of birds, there exists a congenital instinct to use a particular type of rock to smash oysters. Although this trait is now hereditary, let us assume that it has started out as a purely phenotypic accommodation made by an individual organism several generations back. As we have seen, a trait that has begun as a purely phenotypic accommodation could only become hereditary by means of a genocopy. Prior to the emergence of this genocopy, however, the trait has been passed down from generation to generation through imitation and parental instruction. If we wanted to explain the existence of a trait with such a history in terms of the utility function of genes, we would be presented with a challenge. In the first place, the existence of such a trait implies that during the potentially long period *prior* to genetic fixation, it would make no sense to ask how this trait benefits the quantitative maximalization of its underlying genes. That is, we could explain the trait in terms of utility function, but the maximized commodity would not yet be the genes encoding for this trait. What would be maximized thanks to this trait, would rather be commodities at the level of the individual, such as pleasure, oyster-consumption and ultimately survival chances. Since at this point we are still talking about a trait acquired during individual ontogeny – and have thus not yet left the sphere of plasticity – this is not a problem for the Genocentric Perspective in and of itself. However, this first step does have a more problematic result: *after* genetic fixation has taken place, it becomes possible to explain the existence of the trait in terms of the utility function of its underlying genes, but this explanation will then not account for the ‘pre-genetic fixation period’ in the trait’s history. This makes for a rather mysterious transition from ontogeny into phylogeny: during ontogeny there is no point in asking how a

trait benefits its genes, because the trait is not yet hereditary. Once the trait enters the realm of phylogeny, we *can* ask how its existence benefits its genetic foundation, and we will be able to answer this question in terms of genetic utility function. Yet how the Genocentric Perspective accounts for the transition between ontogeny and phylogeny – at least, when neuro- and psycho-genetic accommodations are concerned – remains quite unclear. It seems that the only way to do this would be to suppose that genocopies can emerge following and confirming purely phenotypic accommodations, which would be inconsistent with the most fundamental components of the Genocentric Perspective.

One could object that the commodity that is being maximized prior to the genetic fixation of a purely phenotypic accommodation actually *is* gene-survival, namely the survival of the genes that enable the organism to imitate, learn through trial and error or social instruction. Because these genes are favoured by natural selection due to their effects on the individual, these effects will spread through the population. However, this would only account for the spread of plasticity – in particular the abilities to imitate and learn – through the population. The Baldwin Effect, on the other hand, is a process in which the imitated or learned habits and skills *themselves* become hereditary over time. Solely referring to the benefits to the genes that make the acquirement of habits and skills possible, will therefore not suffice.

Admittedly, this has been quite a journey, but we are now finally equipped with enough information to answer the question central to this thesis: *to what extent can the Genocentric Perspective on evolution be considered sufficient to describe the Baldwin Effect?* The premature answer to this question stated that the Genocentric Perspective can be considered sufficient to describe the Baldwin Effect to the extent that it implies the identification of this effect with Waddington's Genetic Assimilation. However, this identification itself was deemed inapt. A precise and refined substantiation for this claim can now be given. From all the considerations above, we can deduce that any comprehensive description of the Baldwin Effect must meet the following two criteria:

1) It must be a description in which all of Baldwin's types of ontogenetic accommodation – physico-, neuro-, and psycho-genetic accommodations – are included.

2) It must be a description which allows for all of Baldwin's types of ontogenetic accommodation to be genetically fixed over the span of multiple generations.

We have seen that the Genetic Perspective has no problem meeting the first criterion. After all, this perspective logically leads to an understanding of the Baldwin Effect as Waddington's Genetic Assimilation – whose plasticity concept accounts for physico-genetic accommodations – and in addition to this, it fully recognizes the plasticity concept on which Mutation-based Genetic Assimilation relies – a concept that accounts for neuro- and psycho-genetic accommodations. However, the Genocentric Perspective *does* have a problem meeting the second criterion; to account for the genetic fixation of neuro- and psycho-genetic accommodations, the genetic fixation involved in Mutation-based Genetic Assimilation is needed. This type of fixation, which takes place through the emergence of genocopies, has been shown to be incompatible with the idea that the existence of each and every hereditary trait can be comprehensively explained with reference to the quantitative maximization of its underlying genes. Ergo, the Genocentric Perspective can be considered sufficient to describe the Baldwin Effect *in part*, but not in its entirety.

Conclusion

Summary

This thesis has sprouted from a desire to understand the tension that seems to exist between the different roles that the individual plays in, respectively, the Baldwin

Effect and the Genocentric Perspective on evolution. In order to analyse – and potentially nullify – this tension, it was asked whether the Genocentric Perspective could be considered sufficient to describe the Baldwin Effect. The inquiry that has enabled us to answer this question, can be summarized as follows:

We started our inquiry by familiarizing ourselves with Baldwin's original formulation of the theorem that would later be named after him. It was shown to consist of two components: organic selection among three types of ontogenetic accommodations – physico-, neuro-, and psycho-genetic –, and the subsequent effects of this selection upon the evolutionary development of the relevant species. These two respective components were linked to Godfrey-Smith's notions of plasticity and genetic fixation. This linkage enabled us to insert two related processes into the argument that harbour notions of plasticity and of genetic fixation as well, and are both occasionally identified with the Baldwin Effect: Waddington's Genetic Assimilation and Mutation-based Genetic Assimilation.

These forms of genetic assimilation, however, were demonstrated to adhere to different ideas of plasticity and consequent genetic fixation. In the case of Waddington's Genetic Assimilation, a pre-existing genetic variety is supposed to be brought out into the phenotype by an environmental trigger. Natural selection could then favour these genes, causing them to increase in frequency within the population until phenotypic expression could occur without the need for environmental inducement. In the case of Mutation-based Genetic Assimilation, it is presumed that individuals initially make purely phenotypic accommodations, which are then kept in existence through non-genetic routes of inheritance like imitation or other types of learning. This could provide enough time for a genocopy to emerge within the population, which could then be favoured by natural selection.

The analysis of both these concepts of genetic assimilation provided us with a way to link the Baldwin Effect to the Genetic Perspective on evolution, as taking this perspective precisely results in an understanding of the Baldwin Effect as Waddington's Genetic Assimilation. After all, the Genocentric Perspective

adheres to the idea that the existence of every adaptive trait can be explained by disclosing how this trait benefits the replication of the genes that underly it – in other words, the idea that all traits can be explained in terms of a genetic utility function. Applied to a trait that started out as an ontogenetic accommodation, this explanation-strategy will naturally lead to the presupposition of a dormant genetic foundation that is not newly acquired, but is merely caused to be expressed. Without such a genetic foundation, there would be nothing to be increased through the process of natural selection.

After this detour into the world of the Genocentric Perspective, we returned to Baldwin's original account of organic selection on physico-, neuro-, and psycho-genetic accommodations, and combined these with the different plasticity and genetic fixation concepts involved in Waddington's Genetic Assimilation and Mutation-based Genetic Assimilation. As it turned out, physico-genetic accommodations can be seen as analogous to the plasticity of Waddington's Genetic Assimilation, while neuro- and psycho-genetic accommodations can be seen as closely linked to the plasticity of Mutation-based Genetic Assimilation. To represent all of Baldwin's types of accommodation, both forms of plasticity thus need to be appealed to. It was shown that because of this, we cannot exclude either one of the two forms of genetic fixation that spring from the respective plasticity concepts.

All these considerations allowed us to assess the adequacy of the Genocentric Perspective to describe the Baldwin Effect. Although taking the Genocentric Perspective was shown to lead to the identification of the Baldwin Effect with Waddington's Genetic Assimilation – and therefore to the adherence of the plasticity and fixation concepts that are associated with this type of assimilation –, it was noted that the Genocentric Perspective also recognizes the plasticity involved in Mutation-based Genetic Assimilation. The Genocentric Perspective was thus deemed adequate to account for all of Baldwin's types of accommodation.

However, it was established that any comprehensive account of the Baldwin Effect must also allow for all of Baldwin's accommodations to be genetically fixed over the span of multiple generations. This is where the Genocentric Perspective was demonstrated to fall short, as neuro- and physico-genetic accommodations rely upon the emergence of genocopies to be genetically fixed. This would mean that the acquisition of the accommodation in question would precede the acquisition of the genetic basis necessary for the direct inheritance of the accommodation. The question central to the Genocentric Perspective was shown to be insufficient to handle accommodations with such origin stories. On the basis of this argumentation, the question '*to what extent can the Genocentric Perspective on evolution be considered sufficient to describe the Baldwin Effect?*', was answered: the Genocentric Perspective has turned out to be sufficient to describe the Baldwin Effect in part, but can be considered insufficient to describe it in full.

Discussion

Before we take a look at what this conclusion implies, an important caveat already eluded to in multiple footnotes has to be considered. Throughout this investigation, I have made use of fairly outlined categories and well-defined concepts – both borrowed and original. This has made it possible to compare and contrast different theorems, which has in turn enabled me to draw the above conclusion. However, the categories I have employed may in some cases be oversimplified and arbitrary versions of the phenomena they are meant to represent in reality. For example, it is likely that many ontogenetic accommodations harbour elements associated with both neuro- and psycho-genetic accommodations, or even include elements belonging to all three of Baldwin's accommodation-categories. One could even argue – as Baldwin himself has remarked in his original paper on organic selection, although he seemed to consider purely physico-genetic accommodations as the exception to

this rule – that most ontogenetic accommodations involve some contribution of the organism's nervous system, although this in itself does not tell us at which point in the process of acquiring an accommodation this contribution is made.[83]

To name another example, the distinction between Waddington's Genetic Assimilation and Mutation-based Genetic Assimilation has been explained without reference to the fact that, in all probability, there exists a grey area between the two. In some cases, asking whether the acquisition of the phenotypic accommodation has preceded the acquisition of the necessary genetic foundation, may be like asking which came first, the chicken or the egg. There may not always be a fact of the matter about the order of these occurrences, which has to do with the fact that a large number of genes are involved in the development of most characteristics. When a mutation causes a small change in an already existing characteristic, this change may make the organism in which it occurs more likely to make further changes to the characteristic in question. These further changes may in turn be confirmed by sequential mutations, which brings us full circle. It seems rather likely that such a course of events can take place, since so many traits are polygenic. Accommodations that occur in this gradual way, are not easily placed under the heading of either Waddington's Genetic Assimilation or Mutation-based Genetic Assimilation, but seem more at home somewhere in-between.

In short, there are grey areas galore. Yet without projecting relatively crude boundaries onto gradual phenomena, it would become extremely difficult to make comparisons between them. Phrased differently, doing justice to the fluidity between all processes and concepts that are employed in our current investigation would get us stuck in a whirlpool of grey areas before we would even get a chance to formulate the research question – let alone answer it in any substantial way. I would argue, however, that this caveat in itself does not nullify the conclusion stated above. Even if no arbitrary categories had been postulated, only looking at evolution from the Genocentric Perspective would still result in an incomplete

[83] Baldwin, "A New Factor," 9.

account of the Baldwin Effect, simply because the many grey-areas – in which the extent to which the individual's initiative induces the relevant accommodation remains unclear – would not be sufficiently recognized. In short, doing justice to the probable fluidity of the processes involved in the Baldwin Effect, does not lead to a more Genocentric account of these processes; if anything, it reinforces their elusiveness, which makes it less plausible that they can be captured through the sole use of the Genocentric Perspective.

Now what does our conclusion imply for the tension concerning the role the individual organism should be assigned in evolutionary theory? Here, it must in the first place be remarked that we should not interpret this conclusion as an attempt to refute the validity of the Genocentric Perspective; seeing genes as the units on which natural selection ultimately works, provides us with immense explanatory power. However, I suggest that the explanations that spring from the Genocentric Perspective should be understood as only *one type* of explanation useful to grasp the intricacy of evolutionary processes. A legitimate answer to the question 'why is this trait there?' can in most cases be given in terms a genetic utility function, but this does not mean that an equally relevant answer of a different style cannot be given at the same time. For example, in order to explain why a certain kind of bird has the instinct to freeze when they detect a lurking predator, we can ascertain that the genes which underly this characteristic propagate themselves by grace of their effects on these birds. However, we could also refer to the way these birds have interacted with their environment – which includes interactions with other individuals from whom behaviour can be copied or who can act as instructors in social learning – as a supplementary answer. Although these answers differ greatly, they would both be sensible and informative.

From the Genocentric Perspective, to ask about the purpose of a certain trait means to ask how this trait helps to quantitively maximize its underlying genes. I would speculate that this approach is directed forwards – 'what is being attained through the existence of this trait?'. If, however, we ask about the causal

history of a trait – 'through which interactions between an individual and its environment has it come about?' – we take an approach that is directed backwards. With the latter approach, we obtain some elbowroom for the individual: the individual's survival may not be the purpose of its adaptive traits, but the way this individual has kept itself alive *can* feature as part of the causal history behind the development of an adaptive trait. These approaches, I argue, are by no means mutually exclusive, but should be seen as two different sides of the same coin – a Genocentric side and a non-Genocentric flipside, if you will.

Moreover, I have sought to show that, when we consider traits that have started out as ontogenetic accommodations, only looking at the Genocentric side of this coin will in some cases lead to an impoverished explanation as to why the trait is there. Note that this does not mean that in such cases, considering the Genocentric side of the coin is completely superfluous. However, *only* looking at this side would lead to an explanatory story that harbours cavities. The Genocentric Perspective is thus not rendered illegitimate by the conclusion drawn above, but what this conclusion *does* imply is that the tension between the different roles that the individual is assigned in the Baldwin Effect and the Genocentric Perspective, is not a phantom-tension: the individual might only feature as a vehicle on the Genocentric side of the coin, but on the flipside, its interactions with the environment – and the way these shape the evolutionary development of its species – are accentuated. We could hypothesize that, on the non-Genocentric flipside of our coin, the individual features in the role of 'interactor'.

Further Research

With the hypothesis mentioned above, a possibility for further research arises: the idea of assigning the role of 'interactor' to the individual, rather than that of a mere vehicle, was put forward by David Hull in his book *Science as a Process: An Evolutionary Account of the Social and Conceptual Development of Science*. He

defined this notion as "an entity that interacts as a cohesive whole with its environment in such a way that this interaction causes replication to be differential."[84] At first glance, it seems plausible to identify this role for the individual with the role it appears to play in the Baldwin Effect. After all, the causal power the interactor has over the differential replication of genes – and thus over the evolutionary development of a species or population – is explicitly mentioned. It would be interesting to investigate what it would mean to insert this notion of 'interactor' into the debate around the Baldwin Effect, especially with regard to the differences between the Baldwin Effect as Waddington's Genetic Assimilation and as Mutation-based Genetic Assimilation. In the latter case, it would in all probability make sense to talk about interactors and replicators, as this assimilation concept entails accommodations that are initiated by the individual and are later confirmed by its replicators. This would give the individual the causal power it would need to pass as an interactor in Hull's sense. Yet in the former case, it is not at all clear whether the individual can be assigned the role of interactor, as with this assimilation concept the initiative to make accommodations is exclusively attributed to the replicators. However, exactly how the notion of interactor fits within the conceptual framework that was edified above, is a question that would require further investigation to be answered.

A second opportunity for further research emerges when we dive back into the world of the Genocentric Perspective, and would consider another one of Dawkins' contributions to evolutionary theory: the notion of 'memes'. Dawkins defines this notion as:

> [a] unit of cultural inheritance, hypothesized as analogous to the particulate gene, and as naturally selected by virtue of its 'phenotypic' consequences on its own survival and replication in the cultural environment.[85]

[84] David Hull, *Science as a Process: An Evolutionary Account of the Social and Conceptual Development of Science* (Chicago: University of Chicago Press, 1988), 408.
[85] Dawkins, *The Extended Phenotype*, 290.

A meme can be most anything we normally associate with culture: a musical tune, an idea, a theory, a catch-phrase, or a fun fact.[86] It propagates itself by replicating its way into as many brains – the meme's own personal vehicle – as possible. The addition of memes into the Genocentric Perspective allows Dawkins to account for the huge class of – mostly human – characteristics that have no genetic foundation.[87] We have seen that, when neuro- and psycho-genetic accommodations are at issue, the Genocentric Perspective cannot give a satisfactory account of the transition between ontogeny and phylogeny. The question now becomes: could memes potentially fill the explanatory gap that has been shown to emerge here? For instance, could we say that neuro- and especially psycho-genetic accommodations are comparable to memes? If so, could we speculate further that the transition from ontogeny to phylogeny that has proved to be so problematic for the Genocentric Perspective can be rephrased in terms of a transition between memetic and genetic inheritance? What would this paraphrase of the problem imply for its potential solutions? Although this is all highly problematic – partly due to the fact that genes and memes are two completely separate types of replicator that may thus act independently of, or even against, each other – I would say that this set of questions deserves an investigation of its own. Yet this is not the time nor place to go down that road.

All in all, when we look back at the road we *did* go down, the following dichotomous image appears in our rear-view mirror: on the one hand, we see a road on which replicators, the ultimate evolutionary executors, are indifferently hitching a ride in their intricate vehicles. On the other hand, we see how these same vehicles can at times manifest themselves as assertive individuals, equipped with more or less highly developed self-driving functions. Which of these two images is closer to reality? Well, this is probably a lot like asking which way of

[86] Dawkins, *The Selfish Gene*, 249.
[87] Although Dawkins discusses memes and mostly within a context of human cultures, he does not claim that memes are exclusively to be found within this context. Remarkably, he even introduces the concept of memes by means of the example of a species of bird called the 'saddleback'. These birds pass on parts of songs from father to son via non-genetic, cultural routes. For this example, see: Dawkins, *The Selfish Gene*, 245-246.

looking at a Necker cube is the correct one. Our consideration of the Baldwin Effect, with all its complexity and elusiveness, strongly induces us to abstain from reducing the individual to either a mere vehicle, or the entity behind the wheel. Both roles may blend into and complement each other. Yet exactly how, why and when they do, may continue to elude us for a long time to come.

Bibliography

Baldwin, James Mark. “A New Factor in Evolution.” *The American Naturalist* 30, no. 354 (1896): 441-451.

Baldwin, James Mark. *Mental Development in the Child and The Race: Methods and Processes*. New York: Kessinger Publishing, 1906.

Bergson, Henri. *Creative Evolution*. Translated by Arthur Mitchell. New York: Random House Publishers, 1944.

Biology Online Dictionary. S.v. “Lamarckian Theory.” Accessed 12-06-2019, URL: https://www.biology-online.org/dictionary/Lamarckian_theory.

Biology Dictionary. S.v. “Asexual Reproduction.” Accessed 26-06-2019, URL: https://biologydictionary.net/asexual-reproduction/.

Biology Dictionary. S.v. “Polygenic Traits.” Accessed 17-06-2019, URL: https://biologydictionary.net/polygenic-traits/.

Carey, Gregory. *Human Genetics for the Social Sciences*. Thousand Oaks: Sage Publications, 2003.

Darwin, Charles. *On the Origin of Species by Means of Natural Selection*. New York: D. Appleton and Company, 1859.

Dawkins, Richard. *The Extended Phenotype: The Long Reach of the Gene*. Oxford: Oxford Landmark Science, 2016.

Dawkins, Richard. *River out of Eden: A Darwinian View of Life*. London: Weidenfeld & Nicolson, 1995.

Dawkins, Richard. *The Selfish Gene: 40th Anniversary Edition*. Oxford: Oxford Landmark Science, 2016.

Depew, David J. "Baldwin and His Many Effects." In *Evolution and Learning: The Baldwin Effect Reconsidered*, edited by Bruce H. Weber and David J. Depew, 3-31. Cambridge, Massachusetts: The MIT Press, 2007.

Dictionary. S.v. "Modern Synthesis." Accessed 12-06-2019, URL: https://www.dictionary.com/browse/modern-synthesis.

Dictionary. S.v. "Weismannism." Accessed 11-06-2019, URL: https://www.dictionary.com/browse/weismannism.

Downes, Stephen M. "Baldwin Effects and the Expansion of the Explanatory Repertoire in Evolutionary Biology." In *Evolution and Learning: The Baldwin Effect Reconsidered*, edited by Bruce H. Weber and David J. Depew, 34-51. Cambridge, Massachusetts: The MIT Press, 2007.

Godfrey-Smith, Peter. "Between Baldwin Skepticism and Baldwin Boosterism." In *Evolution and Learning: The Baldwin Effect Reconsidered*, edited by Bruce H. Weber and David J. Depew, 53-67. Cambridge, Massachusetts: The MIT Press, 2007.

Hall, Brian K. "Baldwin and Beyond: Organic Selection and Genetic Assimilation." In *Evolution and Learning: The Baldwin Effect Reconsidered*, edited by Bruce H. Weber and David J. Depew, 141-167. Cambridge, Massachusetts: The MIT Press, 2007.

Hull, David. *Science as a Process: An Evolutionary Account of the Social and Conceptual Development of Science*. Chicago: University of Chicago Press, 1988.

Mandal, Ananya. "Gene History." *News Medical Life Sciences*. Last modified 22-04-2019. URL: https://www.news-medical.net/life-sciences/Gene-History.aspx.

Masel, J. "Genetic Assimilation Can Occur in the Absence of Selection for the Assimilating Phenotype, Suggesting a Role for the Canalization Heuristic." *Journal of Evolutionary Biology* 17, no. 5 (2004): 1106-1111.

Merriam Webster. S.v. "Natural Selection." Accessed 16-05-2019, URL: https://www.merriam-webster.com/dictionary/natural%20selection.

Merriam Webster. S.v. "Phenotype." Accessed 07-03-2019, URL: https://www.merriam-webster.com/dictionary/phenotype.

Moore, Celia L. "Evolution, Development, and the Individual Acquisition of Traits: What We've Learned Since Baldwin." In *Evolution and Learning: The Baldwin Effect Reconsidered*, edited by Bruce H. Weber and David J. Depew, 115-139. Cambridge, Massachusetts: The MIT Press, 2007.

Online Etymology Dictionary. S.v. "Genetic." Accessed 10-06-2019, URL: https://www.etymonline.com/search?q=genetic.

Scarfe, Adam C. "James Mark Baldwin with Alfred North Whitehead on Organic Selectivity: the "Novel" Factor in Evolution." *Cosmos and History: The Journal of Natural and Social Philosophy* 5, no. 2 (2009): 40-107.

Weber, Bruce H. and David J. Depew. “Preface.” In *Evolution and Learning: The Baldwin Effect Reconsidered*, edited by Bruce H. Weber and David J. Depew, ix-x. Cambridge, Massachusetts: The MIT Press, 2007.