# Contextualizing Genes:
# A Rejection of Strong Genetic Determinism in Favor of Nuanced Interactionism

By

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*Certificate of Approval*

This is to certify that the accompanying thesis by Lauren Hunter Wilson has been accepted in partial fulfillment of the requirements for graduation with Honors in Philosophy.

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# Abstract

Outdated notions of strong genetic determinism frame the current debate and cloud our understanding of the issues surrounding the use of genetic selection and genetic modification. I make the case that we ought to reject the paradigm of strong genetic determinism, embodied by the language of genetic disorder, in genetic selection and modification. In rejecting the "paradigm of strong genetic determinism", I argue that (1) it is mistaken about the interplay between molecular genes and environment in the creation of phenotypes and (2) when informing the ethics of genetic selection and modification, it ignores viable solutions to improve people's wellbeing, such as the alteration of cellular and social-environmental contexts. I propose that the outdated paradigm of strong genetic determinism and its accompanying language, be replaced by the paradigm of nuanced interactionism. I argue for the adoption of a nuanced interactionism based on difference making and specificity because this interactionism 1) holds environment and molecular genes as both loci for improving wellbeing 2) while also allowing for context dependent evaluation of difference-makers. Under nuanced interactionism, molecular genes will be considered only one of many causes that generate phenotypes and wellbeing. A nuanced interactionist-based ethic will in turn affect our understanding of issues in genetic selection and editing.

# Chapter 1 The Paradigm of Strong Genetic Determinism

Outdated notions of strong genetic determinism frame the current debate and cloud our understanding of the issues surrounding the use of genetic selection and genetic modification[1]. I make the case that we ought to reject the paradigm of strong genetic determinism, embodied by the language of genetic disorder, in genetic selection and modification. In rejecting the "paradigm of strong genetic determinism", I argue that 1) it is mistaken about the interplay between molecular genes[2] and environment in the creation of phenotypes and 2) when informing the ethics of genetic selection and modification, it ignores viable solutions to improve people's wellbeing, such as the alteration of cellular and social-environmental contexts. I will use examples to illustrate how the paradigm of strong genetic determinism has limited our understanding of the possible avenues by which negative effects on wellbeing can be alleviated. I propose that the outdated paradigm of strong genetic determinism, and its accompanying language, be replaced by the paradigm of nuanced interactionism. Nuanced interactionism allows for a more accurate understanding of the interplay between molecular genes and their contexts. Under nuanced interactionism, molecular genes will be considered only one of many causes that generate phenotypes and wellbeing. A nuanced interactionist-based ethic will

[1] Resnik & Vorhaus, *Genetic Modification and Genetic Determinism,* 2

[2] Within this paper, I will be using "Molecular genes" and "Molecular gene sequences" in place of alleles. Much of the bioethics literature uses the language of "molecular genes" or "molecular gene sequences" while scientific writers use the language of alleles. Alleles are variations in the sequence of a particular molecular gene. There are some sections such as II. iii where I use molecular genes and alleles interchangeably. I want to clarify that generally people do not have different molecular genes. However, the sequences of a given molecular gene can differ across individuals or within the same individual (heterozygous individuals).

in turn affect our understanding of issues in genetic selection and editing. The first section of this paper will give an overview of the paradigm of strong genetic determinism, the second section will provide an account of why genes ought to be examined within environmental contexts, the third section expands upon examining genes in contexts to include the social-environmental contexts, and finally, I advocate for the adoption of a nuanced interactionist-based ethic.

It is important to note that the conception of wellbeing that I am concerned with in this paper is based upon the authentic happiness theory of wellbeing. Authentic happiness theory of wellbeing's focus is "that one's happiness should reflect a response of ones, to a life that is one's own."[3] This is in direct contrast with the "nature-fulfillment"[4] understanding of wellbeing utilized by strong genetic determinism. "Nature-fulfillment" understanding of wellbeing involves "fulfillment of our natures as human beings" and thus, wellbeing is largely determined by an individual's ability to embody what it means to be human. To begin the discussion, one must first have a solid understanding of the term strong genetic determinism and the language of genetic disorder.

Genetic determinism "can be loosely defined as the view that genes (genotypes) cause traits (phenotypes)."[5] Resnik and Vorhaus identify three degrees of genetic determinism- strong, moderate, and weak[6]. In strong genetic determinism, genes "almost always" cause a given trait[7]. With moderate genetic determinism, genes "more often than not" cause a trait[8]. In contrast, the weak conception of genetic determinism holds that genes

[3] Haybron, *The Pursuit of Unhappiness,* 35
[4] Haybron, *the Pursuit of Unhappiness*, 35
[5] Resnik and Vorhaus, *Genetic Modification and Genetic Determinism*, 3
[6] Resnik and Vorhaus, *Genetic Modification and Genetic Determinism*, 3
[7] Resnik and Vorhaus, *Genetic Modification and Genetic Determinism*, 3
[8] Resnik and Vorhaus, *Genetic Modification and Genetic Determinism*, 3

"sometimes leads to the development" of a trait[9]. Currently very few, if any, philosophers of biology show out right support for a strong sense of genetic determinism. Yet the paradigm of strong genetic determinism is visibly present in both medical and bioethical discussions and practices, as embodied by the language of genetic disorder.

A genetic disorder as defined by the NIH is:

> A genetic disorder is a disease caused in whole or in part by a change in the DNA sequence away from the normal sequence. Genetic disorders[10] can be caused by a mutation in one gene (monogenic disorder), by mutations in multiple genes (multifactorial inheritance disorder), **by a combination of gene mutations and environmental factors,** or by damage to chromosomes (changes in the number or structure of entire chromosomes, the structures that carry genes).[11]
> [Emphasis added.]

A genetic disorder, by this definition (the first line of the NIH statement), is defined solely by

changes in the molecular DNA sequence. Even with the addition of the phrase "a combination of gene mutations and environmental factors" towards the end of the statement, does little to displace molecular genes as the main cause of genetic disease. By defining a genetic disease mainly in terms of a person's molecular sequence, the NIH perpetuates strong genetic determinism.[12] Moreover, the reference to the environment the NIH does include is vague and lacks even a brief discussion of what ought to be considered an environmental factor. Furthermore, when an environmental factor is mentioned, it is only in conjunction with gene mutations, not as a legitimate factor on its own. The NIH perpetuates the notion that for genetic disorders, the cause is almost

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[9] Resnik and Vorhaus, *Genetic Modification and Genetic Determinism*, 3

[10] Strohman, *Genetic Determinism as a Failing Paradigm in Biology and Medicine: Implications for Health and Wellness,* 196

[11] NIH, *"FAQ about Genetic Disorder*s"

[12] NIH, *"FAQ about Genetic Disorders"*

always due to mutations in molecular gene sequence. Finally, the section labeled "causes" on the NIH website repeatedly describes the sequence of a given molecular gene and its function as *the only cause* of the genetic disorder. Both the NIH definition of genetic disorder and the "Causes" section under many disorders espouse a strong notion of genetic determinism because they locate molecular genetic sequence almost always as the determinate of a phenotype, disease.[13] The NIH is not just espousing genetic determinism, but strong genetic determinism. It is clear that the paradigm of strong genetic determinism is at play in the discussion of genetic disorder, genetic selection, and gene editing through the adoption of the language of genetic disorder.

The NIH definition is one example of how scientists, researchers and physicians perpetuate a paradigm of strong genetic determinism; however, they are not alone. Some bioethicists who work on the ethics of genetic selection and genetic engineering perpetuate notions of strong genetic determinism in their discussions of how to improve wellbeing. While many bioethicists who work on genetic engineering cite a variety of factors that can influence wellbeing, their recommendations continue to focus on the alteration of molecular genes to improve wellbeing. This continued focus on the alteration of molecular genes to improve wellbeing neglects work done in the field of philosophy of biology to expose the ways the social-environmental context impacts gene expression and by extension, wellbeing.

To begin with, Julian Savulescu argues for the use of the principle "Procreative Beneficence" which states "couples should select the child, of the possible children they

[13] Supporting evidence for this claim provided: "Changes in the *F8* gene are responsible for hemophilia A, while mutations in the *F9* gene cause hemophilia B. (NIH, Hemophilia, Causes), "Mutations in the *HBB* gene cause sickle cell disease" (NIH, Sickle cell anemia, causes)

could have, who is expected to have the best life."[14] Savulescu uses this principle to guide human reproduction, which has implications for genetic selection and gene editing. In his argument, Savulescu states, "we should allow for the selection of non-disease genes" noting that gene or genes for asthma have a negative effect on wellbeing[15]. Kean Birch argues, "such terminology implies that the environment, whether natural or social, is inconsequential to human life."[16] Savulescu places genes as the strongest determinate of traits without considering how the social- environmental context interacts with molecular genes to give rise to complex traits. It is a further stretch to say that molecular genes have a negative (or positive) effect on one's wellbeing. This is especially evident in one of the key recommendations Savulescu makes. Savulescu states that parents ought to use information from genetic testing to inform their reproductive decisions. While Savulescu may be providing a practical recommendation, it is still problematic. It may be the case that parents may have far more control over their child's molecular genes than their child's larger social environmental context. However, this aspect of his argument is still an example of strong genetic determinism since he does not consider altering the micro or macro biological environment in which the genes operate. Moreover, Savulescu places their potential child's genetic information as the main consideration with little discussion of the way parents can take steps to alter their social-environmental context to offset the potential challenges of their child's genetics. Savulescu argues that one critical aspect of procreative beneficence is that parents "have a moral obligation to test for genetic contribution to non-disease states… to use this information in reproductive

[14] Savulescu, *Procreative Beneficence: Why We Should Select the Best Children*, 415
[15]Savulescu, *Procreative Beneficence: Why We Should Select the Best Children*, 415
[16]Birch. *Beneficence, Determinism and Justice*, 18

discussion making."[17] His argument hinges on a strong sense of genetic determinism that does not accurately reflect the force the social-environmental context can have in shaping a child's wellbeing. Even authors who have different views about the types of genes that warrant modification or genetic selection, such as Dena Davis and Laura Purdy, share the same outdated assumption that the conditions are almost always caused by molecular genes.

Dena Davis' argument is founded on the idea that certain molecular genes greatly limit a child's future. Resnik and Vorhaus highlight her reliance on strong genetic determinism for "the open future also assumes a strong form of genetic determinism …in arguing that genetic modification narrows the range of life choices available to the individual."[18] Davis' insistence that genetic modification can obstruct a child's open future fails "in the absence of a strong causal link between genotypes and phenotypes" for "genetic modification might not close off any options or the child."[19] When Laura Purdy discusses the discovery of the cause of Huntington's disease, she uses the phrase "the defective gene itself" and "gene for the disease."[20] She pinpoints a single molecular gene as the sole cause of Huntington's disease and the misery that accompanies the phenotype. Purdy's very argument hinges on the assumption of strong genetic determinism because certain gene's sequences almost always lead to a given detrimental phenotype and therefore, should be avoided even if there is even a small likelihood of passing on a given genotype. In order to show why the paradigm of strong genetic

[17] Julian, *Procreative Beneficence: Why We Should Select the Best Children*, 414
[18] Resnik & Vorhaus *Genetic Modification and Genetic Determinism*, 6
[19] Resnik & vorhaus *Genetic Modification and Genetic Determinism*, 6
[20] Purdy, *Genetics and Reproductive Risk*, 43

determinism ought to be abandoned, it is critical to understand how genes are seen to contain causal information as informed by philosophy of biology.

# Chapter 2 Genes in Contexts

Phenotypes with a negative effect on wellbeing that have been passed from one generation to the next have long been the avenue by which physicians and ethicists understood genetic disorders. Instead of looking at molecular gene sequences, scientists would track the heredity of certain phenotypes (physical traits) such as hemophilia. These hereditary phenotypes were understood as classical genes. This particular focus (classical genetics), which took the classical gene as its object, helped scientists trace heritable aliments before traits were connected with a physical particle (molecular genes). The connection between classical gene (phenotype) and a physical heritable particle occurred with the discovery of the structure of DNA. This discovery led to the understanding that some[21] classical genes (phenotypes) are caused by particular regions within DNA that serve as a template for the production of proteins that in turn create phenotype. We can call these regions 'molecular genes', which are said to be the cause of classical genes. In the case of Huntington's disease, the classical gene is the physical neuro degeneration (phenotype) and its heritability through generations. Specifically, parents with Huntington's disease have a 50% chance of having a child with Huntington's disease each pregnancy. In contrast, the molecular gene is the presence of a trinucleotide

[21] Classical genetics focused on the origin of heritable phenotypes. Origin could be molecular genes or other causes such as development environment and intervention.

sequence (CAG) repeated 10-30[22] times in the HTT gene on 4p16.3.[23] The discovery of the molecular gene allowed scientists to trace classical genes to the molecular gene. By doing this, scientists were able to designate segments of DNA as the cause of genetic disorders.[24]

As stated in the introduction, many molecular genes have been labeled as "bad genes" or "disease genes" because they are seen as almost always causing specific phenotypes that are seen to have a negative effect on wellbeing. This understanding is informed by strong genetic determinism, which in turn produces the language of genetic disorder. However, the term "genetic disorder" and the label of "bad gene" which follows do not make sense when applied to the molecular gene. The creation of phenotypes requires molecular genes to interact with their surrounding social-environmental context. It is this interplay with the environment that is not considered when the term "bad" is applied to a single molecular gene sequence. The term "bad" or "disordered" cannot be applied to a molecular gene's sequence in isolation; this designation of good or bad can only be understood within a given context.

[22] NIH, HTT Gene
[23] NIH, HTT Gene
[24] In addition, the discovery of the molecular gene categorized certain diseases such as Down Syndrome as genetic disorders, which before the discovery of DNA had not been understood in terms of classical genes. Until recently, novel changes in DNA sequence, like Down Syndrome, were not viewed as classical genes because they were not passed down. They were not hereditary people with these novel genetic sequences may not have lived long enough to reproduce.

## Section 2.1 Genes in Micro-Context

To challenge the idea that genes can be inherently good or bad outside of a specific molecular context, I will explore the occurrence of suppressor mutations[25]. Previous experimental studies in cancer research have explored the use of suppressor mutations to restore p53[26], a gene which works to suppress tumor development. Suppressor mutations occur after the first mutation in a gene (in this case p53). In this example, p53 mutates[27] in such a way as to produce a differently structured protein that no longer interacts at all, or as well, with proteins it had previously interacted with. Therefore, p53* can no longer suppress tumor formation. The mutation results in a variation of the molecular gene that is seen as negative because the gene p53*'s protein cannot conduct the necessary interactions with other proteins in the cell in order to function in its given molecular-context. A suppressor mutation occurs when a gene or genes B (B represents any genes or proteins the protein of p53 would have interacted with) mutate in such a way that B* can now interact with p53* in a way that restores p53* function in this specific context. Within this example, the mutation that caused a genetic difference in gene p53 was negative without corresponding mutations in genes B because p53* could not interact with proteins B it had previously interacted with, which were needed for cell functioning. This same gene, mutant p53*, when placed in a new context with mutant B* is able to perform the necessary interactions for cell wellbeing.

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[25] Brachmann et al. "Genetic selection of intragenic suppressor mutations that reverse the effect of common p53 cancer mutations"

[26] P53 acts as a transcription factor that induces other genes to produce proteins needed to carry out G1 arrest or apoptosis

[27] Mutated p53 and B will be noted as p53* and B*

p53* is able to suppress tumor formation. In fact, wild type[28] gene p53 may have a negative effect within the context of mutant gene B* system because it may not be able to suppress the formation of tumors.

This is a specific example of how gene's contribution to a specific phenotype is heavily influenced by what other genes and gene products are present. More generally, genes cause problematic phenotypes when they are either not expressed, over expressed, or expressed at the wrong times. A gene's expression is entirely dependent on regulatory elements such as proteins, RNA, hormones and other molecular particles present at a molecular-level. Furthermore, recessive changes in molecular gene sequence (recessive mutations[29]) are commonly thought to be bad. However, recessive mutations only become problematic when there is not redundancy. Thus, it is not just the presence of other genes at other loci and other regulatory elements, but also the presence and sequence of the corresponding allele of the same gene on its homologous chromosome. The role of different regulatory elements and other gene products a given gene interacts with determines whether a particular genetic variation is positive or negative. Therefore, the term good or bad cannot be applied to a single molecular gene sequence; rather it can only be applied to a molecular gene sequence in a given context.

[28]Wild type refers to a molecular gene sequence (allele or alleles) which results in normal species typical functioning or most common allele of the gene found in wild populations; not a mutant strain

[29] In this case, I am refereeing to non-silent mutations. Silent mutations change the molecular gene sequence but do not change the corresponding amino acid during translation.

## Section 2.2 Genes in Their Cellular and Organismal-Context

The breadth of the context one should consider when trying to understand a gene as positive or negative expands when context is not just understood on the molecular level, but also at the organismal-level. Mutations in polycomb repressive complexes can illustrate the importance of how a gene's position within an organismal unit is critical to understanding how a gene's designation as positive or negative is dependent on organismal-context. Polycomb repressive complexes are responsible for maintaining heterochromatin structure of certain regions of DNA[30]. If the polycomb repressive complex fails to maintain heterochromatin structure within some cells (thus within its microenvironment), this can lead to expression of genes that, within current cells and tissues, are not generally transcribed. The transcription of these genes, with standard sequence and protein structure, can lead to homeobox-like[31] mutations. Since the polycomb repressive complex failed to shut these genes down within heterochromatin, they were expressed in unintended cell types within an organism. Expression of these genes within cells in the standard tissue is beneficial. It is only when these genes are expressed in the improper context (in this case the wrong cells or tissue within an organism) that leads to these genes being detrimental. A common example of miss expressed homeobox genes, similar to failed polycomb repressive complex, can lead to

[30] Heterochromatin is a structure made up of DNA, histones and other proteins which form a tightly enclosed structure which prevents the expression of DNA. Genes within the heterochromatin structure cannot be transcribed and therefore cannot make protein. Polycomb repressive complexes are responsible for signaling another cell machinery that a gene should be enclosed in heterochromatin; essential telling the cell that this gene should not be transcribed.

[31] Homeobox genes produce transcription factors that help establish gene cascade pathways that lay out an organism's body plan. Errors in these genes can lead to mistakes in body plans such as legs growing from a fly's head.

the expression of eyes on the legs and wings of flies. Similar processes dictate heterochromatin structure in humans, that when working properly, lead to the correct body plan for human and when fail, lead to catastrophic alteration in body plan incompatible with life. While the mutation of a polycomb repressive complex is an extreme example, it illustrates that a gene's cellular context (in euchromatin or heterochromatin) matters, as well as where gene expression occurs in the organism. Genes, which when expressed in their typical cell types, lead to smooth cell operation; can also cause severe dysfunction when they are expressed in a different cell type. This example further advances my argument that it is not genes themselves that cause negative effects on wellbeing, but rather genes in cells in organisms that cause negative effect on wellbeing. This negative or positive nature of genes can only be understood within a given context.

Thus far, I have established that this context includes other genes or allele within the genome, the specific cells or tissues these genes are expressed in, and the tissues' configuration within the body of the organism as a whole. Next, I will argue that a gene's context also includes the biological environment in which the organism exists. This context dependent understanding must extend past the molecular-context to the macro-environmental-context.

## Section 2.3 Genes in Macro-Context

One example of how macro-context affects a gene's designation as good or bad can be seen in differential mechanisms for high altitude adaption in humans. Two populations used to study human high-altitude adaption are Tibetan and Andean native populations[32]. For the purpose of this paper, I will focus on how genetic differences between high elevation adaption differ between Tibetan's dwelling at high elevations and other human populations which have historically dwelled at low elevations. Research[33] locates genetic differences within the gene EPAS1 and EGLN1 gene, both hypoxia related genes, as a leading cause of success for Tibetan adaptation to high altitudes. Most humans dwelling at low altitude conditions adapt to high altitudes through a process of acclimatization. The authors describe how "acclimatization to low oxygen involves an increase in blood hemoglobin levels."[34] This increase in hemoglobin levels leads to increased blood viscosity. However, the way Tibetans are able to survive at high altitudes is not the same process that results in increased blood viscosity[35]. Tibetan individuals who have a different allele[36] of EPAS1 gene experience lower levels of hemoglobin at high altitudes than other populations. It is thought that this different copy of EPAS1 molecular gene is what allows Tibetans to thrive at high altitudes.

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[32] Beall et al. *Andean, Tibetan, and Ethiopian Patterns of Adaptation to High-Altitude Hypoxia.*
[33] Peng et al. *Genetic Variations in Tibetan Populations and High-Altitude Adaptation at the Himalayas*
[34] Peng et al. *Genetic Variations in Tibetan Populations and High-Altitude Adaptation at the Himalayas,* 1
[35] To explore these position researchers examined "SNP variants in EPAS1". This "showed significant associations with hemoglobin levels in the expected direction in several of these studies; individuals carrying the derived allele have lower hemoglobin levels than individuals homozygous for the ancestral allele." (Peng et al. *Genetic Variations in Tibetan Populations and High-Altitude Adaptation at the Himalayas,* 1)
[36] See footnote 2 for definition of allele and usage of molecular gene/ molecular gene sequence.

If an individual without the Tibetan copy of EPAS1 molecular gene (allele) were to try to live at high altitude, they would suffer from increased blood viscosity. This increased blood viscosity could put them at risk of cardiovascular conditions such as heart attack, stroke and among pregnant women, an increased risk of pre-eclampsia. Under this macro-context, an allele normally considered to be good, lowland ESAP1, would be detrimental to an individual's wellbeing. Under this same macro-context, the abnormal allele of ESAP1 (common in Tibetan populations but falls outside what is considered the normal sequence for ESAP1) is advantageous. Researchers have determined that the high altitude created strong selective pressures that favored the Tibetan variant of the EPAS1 gene.

The example of different variants in the EPAS1 molecular gene shows how the macro-context in which these genes are operating informs whether the phenotypic traits they contribute to are designated as positive or negative. In isolation, the variants of EPAS1 gene are merely different. In low altitude, Tibetan EPAS1 and lowland EPAS1 are genes involved in hemoglobin and vascular function. At high altitudes, lowland EPAS1 becomes an allele for heart attack, stroke, and preeclampsia, while Tibetan EPAS1 becomes an allele for improved acclimatization and maternal health[37]. Fundamentally, all alleles do is produce differently structured proteins and by extension, phenotypes. It is this critical link, between genes, phenotype and context, that is currently missing within the paradigm of strong genetic determinism. It is only by examining a

[37] Redundancy of the specific variation of EPAS1 are assumed in these examples.

gene's functional efficiency[38] in specific contexts (micro-, organismal-, and macro-) that allows for the language of positive and negative to come into play.

Thus far, I have argued that the context for genes encompasses a broad assortment of situations including intracellular interactions between other genes and cell machinery, cell and tissue level expression of genes, and the biological environment of the organism at large, such as the environmental context. The context of a gene is not limited to the biological examples above. The purpose of these examples has been to illustrate how the functional efficiency of a gene depends on the interaction between a given gene and the specific context being examined. The designation of a particular molecular gene sequence as negative cannot remain fixed to this molecular gene sequence once the context has changed. I argue that the discussion of context should also extend from the pure biological to the social-biological. The paradigm of strong genetic determinism especially disregards the role of both the biological and social environment that the body and its genes are situated within.

Before I engage with how the social-environmental-context affects a phenotype's designation as positive or negative, I must refocus the conversation. Thus far, my argument has focused on examples that utilize an evolutionary biology understanding of how genes can be a benefit or a detriment, specifically with regard to evolutionary fitness. The evolutionary fitness view of genes is concerned with an organism's ability to survive and reproduce as the sole determinate of wellbeing. I have previously chosen functional efficiency centered examples because scientists, physicians and evolutionary

[38] "The functional efficiency of a part or process is both a matter of how well it measures up to the demands of the moment, and a matter of its *capacity* or *disposition* to make a specific contribution when it is needed." (Hausman, In *Valuing Health: Well-Being, Freedom, and Suffering* ch2, p. 9)

biologists recognize that these interactions between genes and their contexts have biologically measurable effects. In the case of multicellular organisms, the effects can lead to decreased survival, which impacts the production of viable offspring. When looking at the biologically measurable effects related to a single cell, one could examine the decreased ability for cells to produce certain biologically necessary proteins which prevents them from undergoing mitosis or meiosis (replicating themselves and by extension their DNA). This might or might not increase the evolutionary fitness depending on the larger context. While the ability to appeal to evolutionary finesse's conception (through functional efficiency) of a gene's benefit or detriment within a given context is useful, it is not the goal of this paper. Rather, this paper is focused on molecular gene's relationship to wellbeing. The view of wellbeing that I explore in this paper, namely authentic happiness, encompasses a number of variables outside of functional efficiency as it relates to evolutionary fitness.

The evolutionary fitness understanding of molecular genes does not allow us to explore the relationship between genes and wellbeing. If I were to focus on the evolutionary fitness view of genes, a genetic difference such as Huntington's disease could be neutral so long as the symptoms occurred after the person has reproduced successfully and the person's death did not have a measurable evolutionary detriment to the population. My account of a gene's functional efficiency does not commit me to an evolutionary fitness account of wellbeing. First, this focus on evolutionary fitness does not reflect our understanding of genetic disorders. If it did, diseases such as Alzheimer's, would be of no concern so long as it did not markedly reduce the number of viable off-spring an individual may produce. Furthermore, our inclination is that Alzheimer's has a

negative effect on wellbeing in more ways than potentially reducing evolutionary fitness. As Daniel Dennett states in his book *Freedom Evolves,* "Whereas all other living things are designed by evolution to evaluate all options relative to the summum bonum of reproductive success, we can trade that quest for any of a thousand others."[39] Only by examining the ways molecular genes affect wellbeing outside of the evolutionary biology context can we begin to give an accurate depiction of how a molecular gene's interaction with the social-environmental-context negatively affects wellbeing. We as a society have a more enriched understanding of wellbeing, which encompasses ideas of health, community connection, fulfillment, and autonomy. It is my hope that by undergoing this examination of how context determines a molecular gene's functional efficiency, one can incorporate this into a more complex conception of wellbeing. This more complex understanding of wellbeing better speaks to our intuition that wellbeing encompasses more than evolutionary fitness.

[39] Dennett, *Freedom Evolves*, 179

# Chapter 3 Beyond Biological Environment: The Social Environment of Genes

Thus far, I have focused on how the functional efficiency of genes is dependent on biological-context. In this next section, I discuss how strong genetic determinism is informing ableism in genetic editing. Under this view, bad genes produce certain disabling or diseased phenotypes because they reduce a person's wellbeing. However, I want to push back against the view that molecular genes should be the focus of alterations to improve wellbeing in the ethics of genetic editing. I will argue that instead of the paradigm of strong genetic determinism, we ought to adapt a paradigm of nuanced interactionism that acknowledges both genes and social-environmental contexts are causes of phenotypes.[40] Moreover, a person's wellbeing is also affected by the interaction between one's phenotypes and their environment.

Before I can begin to present my positive view, it is first important to understand the relationship between genes, phenotypes, and wellbeing under the paradigm of strong genetic determinism. I argue that the paradigm of strong genetic determinism informs a form of genetic ableism when it comes to the consideration of wellbeing in genetic editing. Melinda C. Hall makes the argument for the existence of gene-based ableism stemming from the persistence of genetic determinism in her paper *Reconciling the*

[40] A more detailed understanding of the interactionist position will be given later in the chapter. For now, our working definition of interaction is "a view merely saying that every feature of every organism is due both to its genes and environment and there is no way to distinguish their importance" (p. 86 Godfrey-Smith). A more nuanced position will be discussed later on.

*Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection.* Ableism and genetic determinism are critical concepts for Hall. Ableism and genetic determinism both work to collapse distinctions between genes, phenotypes, and wellbeing. Hall defines ableism as the "discrimination against persons on the basis of perceived disability."[41] She defines genetic determinism as "the evaluation of genetic factors to the level of autonomous causation; in other words, genetic determinism is the view that individual traits and behavior and often, social circumstances or problems can be explained solely through genetic factors."[42] Hall locates one clear example of the combined effect of ableism and genetic determinism in the practice of synecdone or "the identification of one trait with the whole, the fetus."[43] Synecdone occurs when parents or doctors advocate for termination of a pregnancy or do not select embryos because they contain a single molecular gene sequence seen as bad. Hall goes on to describe the case of Down Syndrome that is often selected against in a synecdone fashion. Hall notes that persons with Down Syndrome exhibit a diverse set of phenotypes and often have cognitive abilities far exceeding those explained by doctors to potential parents.[44]

From Hall's argument presented above, I want to make clear that there is an understanding of genetic ableism: the selection against people or embryos based on their genetic makeup. Genetic ableism is linked to the idea that certain genes constitute a disability or solely cause a negative influence on wellbeing. This genetic ableism is

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[41] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 122
[42] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*,124
[43] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*,128
[44] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 134

exemplified in acts of synecdone, where one trait is used to represent the worth of the whole individual. Bioethicists such as Julian Savulescu[45] and Laura Purdy[46] subscribe to a form of genetic ableism and actively argue for negative genetic selection[47] based on this view.

Now that I have made clear the nature of genetic ableism, it is necessary to understand how genetic ableism understands genes as the determinates of wellbeing. I argue that genetic ableism, like strong genetic determinism, understands genes as the strong determinant of disability that negatively correlates to one's wellbeing. Under this view, even in an ideal world (one in which people currently considered to have disabilities would not experience the social stigma and prejudice present in our current society) would still be worse off than their nondisabled counterparts, and therefore should be selected against. This view of disability and wellbeing is the "medical model of disability."[48] Hall states that "the medical model of disability, in its strongest version, orients all limitations due to disability in the biological facts of the particular disability; discomforts and lack of freedoms are all, on this model, due solely to disability's character as a medical or health problem."[49] The medical model of disability is subject to the very same form of reductionism pervasive under the paradigm of strong genetic determinism. The medical model may acknowledge that the structure of society affects

[45] "We should allow the selection for non-disease genes" (*Savulescu, Procreative Beneficence*, 415)
[46] "gene for disease" (Purdy, Reproductive risk, 43) and "those who are opposed to abortion must be especially careful to avoid conception if they are to behave responsibly" (Purdy, Reproductive Risk, 48)
[47] Negative genetic selection refers to the act of selecting against a certain genetic trait. (Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 123)
[48] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 126
[49] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 127

someone's lived experience with disability. However, the medical model seeks to improve a person's lived experience by removing the biological feature of the person that causes the individual to experience the negative effects of societies' structure. This model does not allow individuals to advocate for social change as a means to improving wellbeing. This is deeply problematic because it unnecessarily limits avenues to improving wellbeing. Take for example the way the Americans with Disabilities Act of 1990 vastly improved the lives of those with disabilities. The act required that employers make reasonable accommodations for disabled employees, such as wheelchair ramps, which improved accessibility. The act also prevented employers from discriminating in the hiring process based on disability. More than 25 years after the ADA's implementation, reviews of the ADA have found that the act "has influenced knowledge, attitudes and perceptions about the employment of people with disabilities with regards to: (1) knowledge of the law; (2) the perceived employability of people with disabilities; and (3) workplace culture."[50] Seeing the positive changes made by the Americans with Disabilities Act of 1990, many disability rights activists reject the medical model of disability in favor of the social model of disability.

The social model of disability states, "the difficulties of differences in the quality of life experienced by persons with disabilities are due to a lack of social accommodation rather than inherent qualities of the individual who experiences disability."[51] This stance is not the same as saying that disabilities are not limiting. As Hall states, "This shift in

[50] ADA National network, *ADA Systematic Review: Summary of Year 2, Rapid Evidence Review*, https://adata.org/ada-systematic-review-summary-year-2-rapid-evidence-review
[51] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 127

emphasis can still consider certain traits limiting when it comes to activities while maintaining that available alternative modes of activity can ensure high quality of life."[52] This social model of disability better speaks to the improvements in wellbeing we have seen in the lives of disabled people as accommodations have become more accessible. Take for example the use of study aids, recording devices, and computers used to improve the outcomes of dyslexic students. Without these devices and awareness of the nature of dyslexia many students struggle in classes, their teachers make them feel as though they are less than and many internalize feelings of stupidity and low self-worth. The negative effect on wellbeing experienced by dyslexics is mostly due to the frustration they experience when they are unable to perform as well as their peers. Now, imagine the same dyslexic student encountered teachers and parents who understood dyslexia and tailored the child's education in order to utilize the student's strengths and build ways to cope with their weaknesses. Accommodations such as extended time, one-on-one reading tutoring to improve literacy, and education on dyslexia can greatly reduce or remove this frustration dyslexics experience, and thus the negative effect on wellbeing.

Ultimately, the difference between the medical model of disability and the social model of disability comes down to how one conceives of wellbeing. Strong genetic determinism and genetic ableism "places unwarranted emphasis on the size of opportunity range rather than the possibility for meaningful choice and rewarding outcomes within that range."[53] This is supported by the adherence to a "nature fulfillment" view of wellbeing, whereby what it means to be able to achieve wellbeing is

[52]Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic Selection*, 127
[53] Hall, *Reconciling the Disability Critique and Reproductive Liberty: The Case of Negative Genetic* Selection, 127

directly connected to what it means to be human[54]. Any deviation from the conception of human, especially in the less than direction, is seen as an inherent loss or absence of what is good. Under this conception of wellbeing, the ultimate decider of wellbeing stems from the ability to have the greatest range of choice, the most opportunity. However, as Adrienne Asch, a renowned disability rights' activist, points out "virtually everyone with a disability can participate in many everyday activities, experience relationships, discover the world beyond themselves, and contribute to familial, social, political, and economic life."[55] Asch's statement speaks to a different understanding of wellbeing, one based on authentic happiness theory.

The authentic happiness theory of wellbeing's focus is "that one's happiness should reflect a response of ones, to a life that is one's own."[56] Unlike a "nature fulfillment" conception of wellbeing, authentic happiness theory does not inherently view disabled individuals[57] as already lacking or less than. Authentic happiness theory focuses on how wellbeing is located in one's own life. This theory allows for disability and acknowledges that disabled individuals may be worse off in certain societies. This is not because they are inherently less human because of their molecular genes as nature fulfillment theory of wellbeing would state. Rather, disabled people often have less

[54] Some philosophers, including Agar, seek to make a distinction between genetic editing of human traits that are relatively close to what humans are capable now (this aligns with currently held human values) and gene editing the far exceed current human capabilities (traits far outside what humans currently value)
[55] Asch, *Where's the Sin in Synecdoche? In Quality of Life and Human Difference: Genetic Testing, Health Care, and Disability,* 320
[56] Haybron, *The Pursuit of Unhappiness*, 35
[57] Certain mental disabilities may pose a challenge to this position if individuals are unable to reflect on their own mental states. However, I argue that many people considered mentally handicapped or developmentally delayed are able to set goals for themselves and reflect on their mood (even if it is not in a sophisticated way).

wellbeing because they are in a position where the social environment undermines their response to one's own life.

Under the authentic happiness conception of wellbeing, molecular genes are not the sole cause of wellbeing. Rather, molecular genes, in so much as they help inform a person's life, along with a person's social environmental context determine wellbeing under the authentic happiness conception. In order to help understand the distinction between molecular genes sequence as the sole cause of wellbeing and molecular gene sequence as only one of many causes of wellbeing, the idea of niche construction may be helpful.

As Odling-Smee et al. writes, "It is self-evident that all organisms must interact with their environments to stay alive, and equally obvious that, when they do, it is not just organisms that are likely to be affected by the consequences of these interactions, but also environments."[58] Niche construction is the term used to describe the complex relationship between an organism and its environment, especially how each shapes the other. One benefit to adopting niche construction is that it allows for an expanded understanding of the relationship between social context (society) and molecular genes. Odling-Smee et al. states "Humans are not just passive vehicles for genes, they actively modify sources of natural selection in environments. They are the ultimate niche constructors."[59] In this way, humans are able to construct the biological and social environment in which their molecular genes interact. Whether we are aware of it or not, humans are one of the shapers of their gene's context. Given the great extent to which

[58] Odling-Smee et al., *Niche Construction*, Chapter 1, p. 2
[59] Odling-Smee et al., *Niche Construction*, 28

humans can engage in niche construction, it is foolish to regard our current paradigm of genetic ableism and strong genetic determinism as stagnant. We have the ability to shape how genetic factors affect a person's wellbeing. Under the social model of disability (which is compatible with niche construction), even genetic disorders that result in loss of a sense only have a negative impact on wellbeing due to the social-environmental context in which these phenotypes reside.

One of the most widely explored examples of the importance of social context's relationship to genetic disorders is deafness. Deafness has a diverse molecular nature and as such, deafness presents a critical case study for understanding a molecular gene's relationship to social-context. Certain deaf communities identify themselves as cultural groups with their own language, history, and artistic tradition. Through this example, I argue that both molecular genes contribution to deafness and deaf phenotype's effect on wellbeing are dependent on the biological and social context of the individual.

One prevalent argument among deaf activists concerning the debate to correct deafness is "that though it is true that deafness carries with it a number of significant disadvantages, it is not a disability because the disadvantages associated with deafness are not natural, but social in origin."[60] This is one reason people often draw connections between deafness and racial minorities. Being a racial minority in certain societies clearly carries negative effects on wellbeing due to discrimination. In order to combat this negative effect on wellbeing, society commonly accepts that the social context should be altered to reduce such negative effects, rather than altering racial minority's appearance.

[60] Levy, *Reconsidering Cochlear Implants: The Lessons of Martha's Vineyard,* 138

The analogy between deafness and racial minorities is useful because deaf advocates also think deafness' negative effect on wellbeing is a product of society and deaf individuals also experience "discrimination, a lower than average level of education… higher rates of unemployment."[61] In this way, deaf advocates argue that deafness is a disability, not a biological impairment. Deaf advocates use this analogy to challenge the idea that the negative effect on wellbeing ought to be mitigated by eliminating deafness. The Deaf community believes that eliminating deafness is akin to eliminating a racial or cultural minority because the Deaf community has a strong cultural and linguistic tradition[62].

Before I continue, it is important to understand the distinction between the concept of biological impairment and disability. Biological impairment is used akin to the medical model of disability discussed in the beginning of this section, while disability should be understood in terms of the social model of disability. Specifically, disability within this context refers to "the limitations faced by disabled people" [63] and are solely caused by "'contemporary social organization."[64] It is important to note that the social model of disability is not the same as the radical constructivism account of biological impairment whereby biological impairment is solely caused by the social context. Instead, I appeal to a practical account of biological impairment, offered by the WHO, "In the context of health experience, an impairment is any loss or abnormality of

[61] Levy, *Reconsidering Cochlear Implants: The Lessons of Martha's Vineyard*, 152
[62] Levy, *Reconsidering Cochlear Implants: The Lessons of Martha's Vineyard*, 138
[63] Wasserman, Asch, Blustein, and Putnam, *Stanford Encyclopedia of Philosophy*, "Disability: Definitions, Models, Experience"
[64] Such as the definition given by the Union of the Physically Impaired Against Segregation Wasserman, Asch, Blustein, and Putnam, *Stanford Encyclopedia of Philosophy* "Disability: Definitions, Models, Experience

psychological, physiological, or anatomical structure or function."[65] Under this view, biological impairment is deeply tied to a sense of species typical functioning or what it means to be human rather than a sense of wellbeing.

Humans, by using sound to signal danger, have engaged in niche construction whereby the inability to hear sound is now seen as a biological impairment within any context. While at this stage, niche construction is acting on the classical gene (deafness) one can imagine a future when niche construction will act on the molecular gene sequence through genetic selection and gene editing. In essence, we have changed our environment, which in turn changes us[66]. Neil Levy, a bioethicist and proponent of using cochlear implants, observed that currently sound is used as a means of altering people. However, it is possible to replace loud auditory alarms with other sensations such as touch or light. Current alarms for deaf and hard of hearing individuals utilize wearable bands that vibrate, beds that vibrate to wake someone from sleep and lights that flash. While a sound is the norm, it is not impossible to imagine its replacement with other sensations.

Levy uses the view that being deaf is an inherent biological impairment as a reason to advocate for the usage of cochlear implants and other measures to eliminate deafness, rather than altering the social-environmental context to remove deafness' negative effect on wellbeing.[67] Levy subscribed to a nature fulfillment understanding of wellbeing. Under this understanding, deafness viewed as a biological impairment will

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[65] WHO, *International Classification of Impairments, Disabilities, and Handicaps* (1980), 48 https://apps.who.int/iris/bitstream/handle/10665/41003/9241541261_eng.pdf?sequence=1&isAllowed=y?

[66] Later in this paper, I will go on to discuss the role of changing either the environment or our molecular genes.

[67] Levy, *Reconsidering Cochlear Implants: The Lessons of Martha's Vineyard,* 140

always be negative (even once it is no longer viewed as a disability) because deaf individuals are missing hearing which is an inherently human feature. Thus, Levy argues, "some significant disadvantages suffered by the deaf are natural, not social, in origin."[68] This roots Levy in the position that deafness has an inherently negative effect on wellbeing within every context because it will always be a biological impairment. As I have illustrated, Levy's view subscribes to a strong sense of genetic determinism by arguing that deafness will always have a negative effect on wellbeing.

Furthermore, Levy uses his stance that deafness is an inherent biological impairment and will always have a negative effect on wellbeing in order to argue that social-environmental-context should not be altered. He maintains this stance even though historical examples provide evidence that the social-environmental-context can be altered to eliminate deafness as a disability. Levy cites the community of Martha's Vineyard during the 18th and 19th centuries as an example of a context in which deafness was not a disability. Martha's Vineyard experienced a higher than average percentage of deaf individuals. This caused the community to incorporate sign language within the larger non-deaf community. Levy notes, "with the language barrier down the deaf were integrated fully in the community's life…the result was that deaf Vineyarders preformed as well as anyone."[69] In fact, deaf individuals experience higher literacy rates than their hearing members in this community. I argue higher literacy rates are a positive effect on wellbeing due to the way greater literacy impacts an individual's connection to information. Martha's Vineyard is an example of how changes in community culture can

[68] Levy, *Reconsidering Cochlear Implants: The Lessons of Martha's Vineyard,* 140
[69] Levy, *Reconsidering Cochlear Implants: The Lessons of Martha's Vineyard*, 141

change the degree to which certain phenotypes, like deafness, negatively affect well-being. Thus, both physicians and ethicist ought to acknowledge that there exists a wide range of possibilities to improving the wellbeing of a person with deafness outside of eliminating "deaf genes" or adding cochlear implants.

The main takeaways from this brief foray into deafness are to establish that the paradigm of genetic determinism is linked to genetic ableism. Both genetic determinism and genetic ableism subscribe to the idea that genes themselves solely cause disability. Disability under this view subscribes to the medical model of disability that understands genes as having a negative effect on wellbeing through the "nature fulfillment" understanding of wellbeing. I hope that along with establishing the connection between genetic determinism, genetic ableism, and natural fulfillment theory of wellbeing, I have come to disrupt the certainty of this idea. I have done this through an exploration of deafness as disability under the social model of disability. I have used niche construction to challenge the way we view deafness as an inherent disability. Furthermore, I have offered an alternative view of wellbeing, the authentic happiness theory of wellbeing, which allows people with disabilities to obtain as great a sense of wellbeing as able-bodied individuals. Finally, I offered Martha's Vineyard as an example of how deafness, in a different societal context may not be considered a disability. This example leaves open the possibility that under some construction of society, deafness could have a neutral effect on wellbeing under the authentic happiness theory of wellbeing.

Thus far, I have established the existence of a paradigm of strong genetic determinism that informs genetic ableism, especially concerning genetic selection. I have argued that genetic ableism locates genes or genetic disorders, as the determinate of

wellbeing. Through the example of deafness and niche construction, I have sought to undermine genetic ableism, specifically the idea that genes are solely responsible for the negative effect on wellbeing a disabled person may experience. I have sought to undermine this by supporting both the social model of disability and authentic happiness theory of wellbeing, which view disabled and abled-bodied individuals as neutral. This is in direct contrast with both the medical model of disability and the nature fulfillment theory of wellbeing, which view disabled individuals as fundamentally less then. Under this model, neither molecular genes nor phenotypes are the sole determinates of wellbeing. This section supports the environmental-context dependent nature of molecular genes while also expanding interactionism to include the social-environmental-context and this in turn effects wellbeing.

Now that I have established the problems that abound with an ethic based on strong genetic determinism and the ways this leads to the prevalence of genetic ableism, I will turn my attention to an alternative. Instead of the ethics of genetic selection based on strong genetic determinism, the ethic should instead be based on one of interactionism. Here, interactionism locates both the environment (both social and biological) and molecular gene sequence as equal determinates of wellbeing. As such, both the environment and genes ought to be considered equal loci of improving wellbeing.

# Chapter 4 Nuanced Interactionism

Interactionism allows one to consider both molecular genes and environment as legitimate causes of wellbeing and loci of improvement, while opening room for context dependent evaluation. On the surface, interactionism places importance on the interaction between genes and their environment. Within specific contexts, either molecular genes or environment can make larger contributions to wellbeing depending on how they are viewed as causes. What I hope to avoid here is a "bland interactionism" whereby molecular genes and environment are equal causes within all contexts. As discussed in chapter 2, negative or positive judgements can only be made within specific contexts. Under specific contexts, it may be that molecular genes or environments are playing a larger causal role. Therefore, I want to present a more nuanced interactionism from Peter Godfrey-Smith in which "genes and environment both affect every trait, but there are coherent ways to distinguish their roles based on difference-making and specificity."[70] In order to understand how this nuanced interactionism differs from strong genetic determinism, it is important to explore the concept of difference maker and specificity.

Godfrey-Smith defines a difference-maker broadly as a type of cause. An example of this is given in the statement "C caused E because if not for C, E would not have happened."[71] Another way of understanding a difference maker would be that a cause is something "that produces an effect through some local connection between the

[70] Godfrey-Smith, *Philosophy of Biology,*Genes, 88
[71]Godfrey-smith, *Philosophy of Biology,* 86

two."[72] Furthermore, a difference maker can act through redundancy or by omission. Redundant causation occurs when "C1 produced E, but C2 was ready as a backup if C1 had failed."[73] Causation by omission occurs when something fails to interact in a chain of events. These conceptions of causation are already present in our discussion of the nature of molecular genes in chapter 2 section ii. Upon reflection, redundant causation can be applied to our previous example of the recessive mutation, while causation by omission can be applied to the case of the failed polycomb repressive complex.

In addition to the environment and molecular genes being evaluated on the strength of their difference-making, they can also be evaluated for specificity. Difference-makers can be specific or more general. One way to think of this is "X is a specific difference-maker for Y if variation in X over many different values leads to variation in in Y over many different values."[74] An example of an entity being a more specific cause can be shown by reexamining the high-altitude acclimatization in individuals of Tibetan ancestry. Both the oxygen concentration in the air and the molecular sequence of the EPAS1 gene contribute a person's wellbeing at high altitudes. However, EPAS1 gene's molecular sequence is what may allow a person to adapt properly to high altitudes without adverse effects, and thus is the more specific difference maker.[75] In this case, the oxygen concentration and the molecular sequence of EPAS1 gene play a causal role in one's wellbeing at high elevations; however, the molecular sequence of the EPAS1 gene specifically effects one's ability to acclimatize to the low oxygen concentration.

[72] Godfrey-smith, *Philosophy of Biology*, 86
[73]Godfrey- Smith, *Philosophy of Biology*, 86
[74] Godfrey- smith, *Philosophy of Biology*, 88
[75] This example was adapted from an example found in Godfrey-smith, *Philosophy of Biology*, 88

Nuanced interactionism is able to address the pitfalls of strong genetic determinism by allowing the consideration of both environment and molecular gene sequences as causes, while simultaneously allowing for context dependent evaluation of the strength of each as a cause. The strength of the cause is evaluated based on difference-making and specificity. Furthermore, nuanced interactionism can be used to identify the most effective way to intervene and improve wellbeing. Nuanced interactionism is able to effectively avoid genetic ableism while addressing the concerns from disability rights critics. Difference-makers can be examined within specific contexts that allows for both genes and environment to play larger or smaller roles in the causation of different traits or aspects that contribute to wellbeing. For example, Huntington's disease may be a condition where the most effective way to improve wellbeing would be through the alteration of the HTT molecular gene sequence. However, molecular genes may not be a strong or specific difference maker in seeking to improve intelligence, especially now when we have little understanding of how genes influence intelligence. Nuanced interactionism does not simply evaluate genes and biological environment, but also evaluates social-environmental context and causes of wellbeing. Take again the example of the dyslexic student. The difference-maker in their life may be social conditions in general or the one person, teacher, parent, or learning disability specialist, who finally diagnoses the child as dyslexic and provides the support for the child to develop coping mechanisms. This one person serves as a difference-maker in the child's wellbeing because they are able to give the child the tools and resources to achieve wellbeing under the authentic happiness theory of wellbeing.

I argue for the adoption of a nuanced interactionism based on difference making and specificity because this interactionism 1) holds environment and molecular genes as both loci for improving wellbeing 2) while also allowing for context dependent evaluation of difference-makers. Thus, this new paradigm of nuanced interactionism directly addresses the roadblocks the current paradigm of strong genetic determinism left in the way. The paradigm of nuanced interaction especially needs to be adopted in this age of genetic selection and the beginning of genetic engineering.

The paradigm of strong genetic determinism professed by many authors writing on genetic selection allows for one gene to speak for the whole in every context. Nuanced interactionism breaks this down. It complicates ethical discussion because instead of focusing on molecular genes, it emphasizes the interaction between genes and environment and is able to examine specific contexts. Take for example a common ethical conundrum for genetic selection. In this situation, a dwarf couple asks a fertility specialist to help them conceive a dwarf child. Many people's gut reaction is that this family should not be allowed the selection of a dwarf child based on a strong genetic determinist's informed view that their child's life will be worse off due to their molecular gene sequence. The paradigm of nuanced interactionism does not just look at molecular gene sequence. It is able to see past molecular gene sequence to view the specific context in its entirety and make a decision given the specifics of each individual case. It may be that there is nothing morally problematic with a dwarf family intentionally trying to conceive a dwarf child, especially if they live within a community where dwarfism

connection to disability has been minimized or eliminated[76]. Nuanced interactionism opens doors for us to consider these possibilities.

Furthermore, nuanced interactionism may provide support for those who advocate for social-environmental changes that may in fact be better difference makers for more individuals. Nuanced interactionism in its weakest form legitimizes previously dismissed ways to improve wellbeing, such as the exploration of changing society. In its strongest form, nuanced interactionism may demand social change in order to improve wellbeing. Nuanced interactionism is able to recognize that many different entities, including molecular genes and environment, can lead to the same phenotypic presentation. This may be especially important for improving the wellbeing of individuals who have the same or similar phenotype obtained from a variety of different causes.

Deafness as a biological category is broad, comprising wide variation in hearing ability as well as causes of hearing loss. About 50% [77] of the causes of deafness are attributed to genetic factors; the remaining cases are attributed to other causes, such as nutrition and trauma. According to Shearer et al. "Approximately 80% of prelingual deafness is genetic… The most common cause of severe-to-profound autosomal recessive non-syndromic[78] hearing loss in most populations is mutation of GJB2."[79] GJB2 encodes a protein for gap junction beta 2, also known as connexin, involved in the transport of nutrients between cells. Since deafness has a diverse molecular nature, altering molecular

[76] However, in the current society that has not made the social-environmental alterations to improve dwarf individual's wellbeing, it may still be morally problematic.
[77] Angeli, Lin, and Liu, X. (2018). *Genetics of Hearing and Deafness*. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4523052/
[78] Non-syndromic refers to hearing loss with no other signs or symptoms whereas syndromic which refers deafness associate with other symptoms
[79] Shearer, *Hereditary Hearing Loss and Deafness Overview*, https://www.ncbi.nlm.nih.gov/books/NBK1434/#deafness-overview.Causes_of_Hereditary_H

gene sequences may not be the most effective intervention to improve wellbeing. Instead, changing the social-environmental context may be a far greater difference-maker in the lives of many deaf individuals, especially given the fact that deafness can occur at any age and due to physical trauma, as well as genetic causes. Types of intervention could be early sign language learning opportunities during critical language learning period for prelingually deaf individuals born outside deaf communities, or the adoption of translation apps for businesses and educational institutions that employ signing interpreters to ease conversation. Another way to improve wellbeing would be to implement at least basic sign language skills in all public schools so there is at least basic sign language competency in the general population. This has already begun to happen as some parents use sign language to communicate with children too young to speak. Furthermore, sign language has applications for non-verbal individuals common in special needs programs. This brings us to the broader topic of physical or intellectual disabilities.

Some of these disabilities may have their roots in molecular gene sequences, but others may be due to trauma or environmental circumstances. Nuanced interactionism is able to give legitimate credit to the alteration of the social-environmental context that can act as a broad difference-maker for many individuals with diverse intellectual and developmental disabilities. Specifically, alterations such as elevators, wheelchair accessible ramps, and diversity of educational models can improve the wellbeing of people who fall under the broad category of disabled who would not benefit from the alteration of their molecular genes. In some circumstances, alterations of context ought to be advocated for because of its ability to be strong difference-makers in terms of

wellbeing for many individuals with diverse phenotypes who may be experiencing negative effect on wellbeing due to social structure

# Conclusion

The current debate surrounding the use of genetic selection and genetic modification is still framed by outdated notions of strong genetic determinism[80]. The outdated paradigm of strong genetic determinism clouds our understanding of issues in genetic selection and modification. I have argued that strong genetic determinism is 1) mistaken about the interplay between molecular genes and environment in the creation of phenotypes and 2) when informing the ethics of genetic selection and modification, it ignores viable solutions to improve people's wellbeing, such as the alteration of cellular and social-environmental context, which are causal factors.

I supported my position that strong genetic determinism is mistaken about the interplay between molecular genes and environment through my discussion of molecular genes as "bad genes" or "disease genes". I argued that this understanding is informed by strong genetic determinism, which in turn produces the language of genetic disorder. Current biological research suggests that terms such as "genetic disorder" or "bad genes" are misleading at best. I argue that the creation of phenotypes requires molecular genes to work together with environmental factors. It is this interplay between molecular genes and environmental factors that is not considered when the term "bad" is applied to a single molecular gene. The term "bad" or "disordered" cannot be applied to a molecular gene in isolation; this designation of good or bad can only be understood within a given context.

[80] Resnik & Vorhaus, *Genetic Modification and Genetic Determinism*, 2

I supported my second critique of the paradigm of strong genetic determinism, that it ignores viable solutions to improve people's wellbeing (such as the alteration of cellular and social-environmental context that are causal factors) through my discussion of disability. I argue that the paradigm of strong genetic determinism supports a form of genetic ableism when it comes to the natural fulfillment theory of wellbeing. Both strong genetic determinism and genetic ableism subscribe to the idea that molecular genes themselves solely cause disability. Disability under this view subscribes to the medical model of disability that understands genes as having a negative effect on wellbeing through the "nature fulfillment" understanding of wellbeing. I used niche construction to challenge the way we view deafness as an inherent disability. Furthermore, I have offered an alternative view of wellbeing, the authentic happiness theory of wellbeing, which allows people with disabilities to obtain as great a level of wellbeing as able-bodied individuals.

Finally, I offered my own positive position of nuanced interactionism as a replacement for strong genetic determinism. I argued for the adoption of a nuanced interactionism based on difference making and specificity because this interactionism 1) holds environment and molecular genes as both loci for improving wellbeing 2) while also allowing for context dependent evaluation of difference-makers. Nuanced interactionism is able to recognize both molecular genes and environment as causes of phenotype, and by extension, wellbeing.

This paradigm of nuanced interactionism opens up new possibilities for solving the roadblocks the current paradigm of strong genetic determinism left in the way. The paradigm of nuanced interaction especially needs to be adopted in this age of genetic

editing, genetic selection, and the beginning of genetic engineering. An ethic based in nuanced interactionism opens room for evaluations of concrete recommendations in context specific situations as to how parents, doctors, and society ought to engage in genetic selection and genetic engineering. Yet, the most significant contribution an ethic based in nuanced interactionism makes is that it legitimizes alteration of social--environmental context as a possible avenue to improving wellbeing. Nuanced interactionism exposes a path forward that strong genetic determinism had obscured.

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