Contributions from Biology Education Research

Michal Haskel-Ittah
Anat Yarden *Editors*

Genetics Education

Current Challenges and Possible Solutions

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Preface

This book is the outcome of a genetics education workshop that took place in 2019 at the Weizmann Institute of Science in Israel. The workshop brought together researchers studying genetics education from seven different countries with the aim of discussing challenges in this field and to offer possible solutions.

Genetics in the twenty-first century is a complex multilevel domain that has a key role in the scientific pursuit of a mechanistic understanding of biological phenomena. Due to its central importance to understanding biology, biotechnology, and medicine, in the past several decades, knowledge from this domain has begun to cross over from scientific research into our everyday lives. Genetic tests, genetically modified food, and genetically based therapies are all examples of how genetics are affecting our lives and forcing us to make decisions based on our understanding of this domain. The question is, can laypeople make informed decisions on genetic issues? Do they have the basic genetics knowledge for this purpose? Studies on the current public understanding of science suggest that the answer is no. Laypeople do not have the basic genetics knowledge needed to understand, criticize, or make decisions in this field. As educators, our goal is to find ways to change this. The first step toward achieving this goal is to understand what is wrong with current genetics teaching and what effective means are available to overcome these challenges.

In the last decade, studies focusing on analyses of genetics teaching and learning have identified the strengths and weaknesses of genetics education. The aim of this book is to present the state of the art of these genetics education studies in order to pinpoint the major challenges in genetics education, and to draw on the literature for possible solutions. Each chapter in the book presents a challenge of genetics literacy, discusses that challenge, and suggests possible solutions. The proposed solutions and implications are focused on how to make genetics education better in terms of preparing students to deal with genetics in the twenty-first century in order to inform teachers, curriculum developers, and researchers in science education.

The book deals with various challenges of genetics education, while reflecting upon three major issues: ***the content of genetics curriculum, processes for constructing students' understanding in genetics, and the relationships between***

genetics learning and related conceptions and beliefs. These three themes express three major concerns in teaching and learning – what to teach, how to teach it, and how students' prior knowledge and beliefs may affect or be affected by the learning process. In fields like genetics, in which the scientific content is constantly advancing, dealing with these core questions is inevitable and pivotal.

In the first part of the book, **Reflecting upon the Content of Genetic Curriculum**, we delve into specific new contents that can be used to promote genetics understanding in the current era (e.g., molecular-level mechanisms, epigenetics).

Chapter 1 starts with one of the fundamental questions in genetics education, namely, should we include Mendelian genetics in the genetics curricula that are taught in schools. In this chapter, **Kostas Kampourakis** provides strong evidence for the claim that Mendelian genetics provides a distorted view of both heredity and the nature of science. He suggests that Mendelian genetics should be replaced by developmental genetics.

Chapter 2 suggests that epigenetic concepts can provide a new mechanistic understanding of the interactions between environmental and genetic factors. In this chapter, **Niklas Gericke** claims that learning about how the environment can affect organisms' behavior and other biological characteristics may enable abolishing the nature versus nurture dichotomy. Moreover, it may allow overcoming the common notion of causal and deterministic understandings of genes and their functions. Thus, it is claimed that the inclusion of epigenetics in genetics curricula provides a way to counter genetics deterministic conceptions among students.

In **Chap. 3**, educationally significant factors such as general intelligence, reading ability, and examination success are claimed to be as important to learn in the course of genetic studies in schools as the standard school science topics of, for example, eye color, sickle-cell anemia, cystic fibrosis, and height. **Michael J. Reiss** argues that issues such as the role of genetics in intelligence are often discussed in the media, and therefore educators should provide students with the knowledge needed to critically evaluate these issues. Reiss suggests that the general public has not been well-served by much of the genetics that they were taught in school, and that school genetics teaching has the potential to advance social justice.

In the second part of the book, **Reflecting Upon Processes for Constructing Students' Understanding in Genetics**, we explore various suggested approaches for supporting students in developing their understanding of genetics (e.g., learning progressions, and the use of authentic scientific experiences).

In **Chap. 4**, we delve deeper into the actual use of Mendelian genetics and its relation to molecular genetics in four different learning progressions suggested for the teaching and learning of genetics. **Ravit Golan Duncan** and **Moraima Castro-Faix** discuss a central common question among researchers and practitioners, namely whether students need to first understand mechanisms at the cellular and molecular levels before they can understand ideas about Mendelian inheritance patterns, or vice versa. The arguments raised in this chapter are based on research that was carried out on the actual use of four learning progressions for teaching and learning, as well as on how these progressions can be used as tools to improve genetics literacy.

Chapter 5 provides an in-depth examination of the development of mechanistic understanding in genetics, thus enabling to delve more deeply into the relationships between the level of the observable trait and the cellular and molecular levels, which are included in the learning progressions proposed in the previous chapter. **Michal Haskel-Ittah** describes three milestones that she identified in the progression toward a mechanistic understanding in genetics, and proposes novel scaffolds for moving along the progression of mechanistic understanding.

Based on recent advances in genetic research, in **Chap. 6**, it is claimed that high-school students should know how genetics knowledge has been acquired and understand its applications to societal issues, so that they can make informed decisions regarding genetics-related issues as future citizens. **Bat-Shahar Dorfman** and **Anat Yarden** suggest that one way to provide these opportunities is through practicing authentic scientific experiences, and they provide examples of such experiences in genetics that have been practiced in high schools. Through these, they demonstrate the benefits of authentic scientific experiences for promoting genetics literacy, while outlining the challenges involved.

In the third part of the book, **Reflecting Upon the Relationship Between Genetics Learning and Related Conceptions and Beliefs**, we turn to deal with the mutual relationship between learning genetics and related conceptions such as genetic essentialism, teleology, or beliefs in genetic determinism. This relationship is discussed as a mutual effect in which learning genetics might be hindered by existing conceptions and, on the other hand, learning genetics may hinder the development of such conceptions. Thus, this relationship is not only important for learning genetics but may also have social implications.

Chapter 7 raises the difficulty associated with ascribing more causal power to genes in the formation of traits than available scientific knowledge suggests, or Belief in Genetic Determinism (BGD). **Niklas Gericke, Charbel N. El-Hani, Gena C. Sbeglia, Ross H. Nehm,** and **Neima Alice Menezes Evangelista** examined the distribution of BGD among university undergraduates across three countries (Brazil, Sweden, and the United States) for 14 different traits. They present evidence for BGD being potentially restricted to particular traits.

Chapter 8 explores the question of why multiple and interactive causation of human traits and behaviors is difficult to comprehend. **Marcus Hammann, Tim Heemann,** and **Johannes C.S. Zang** provide insights into individuals' thinking about the causal attributions of genes, environment, and personal decision to the formation of traits. They suggest reorganizing the curriculum and emphasizing gene–environment interactions to counteract the misconceptions that genes or the environment alone determines traits, thus strengthening the conclusions raised in Chap. 2 regarding the teaching of epigenetics.

Chapter 9 presents findings that in the context of genetics, teleological and essentialist conceptions are common among secondary-school students, and that they are dependent on students' age. **Florian Stern, Kostas Kampourakis, Marine Delaval,** and **Andreas Müller** outline previous research on the obstacles raised by teleological and essentialist conceptions among learners, (re)define them, and suggest what teachers might do to overcome these obstacles in the classroom.

Based on social-psychology theory and genetics education studies that elucidate how genetic information influences social cognition of race, in **Chap. 10**, genetics education is claimed to have the power to perpetuate as well as help prevent racial prejudice. **Brian M. Donovan, Brae Salazar,** and **Monica Weindling** argue for a more humane genetics education, which attempts to reduce racial prejudice by helping students understand the complexity of genomic science.

Taken together, we believe the collection of chapters in this book will provide novel ideas for biology teachers, curriculum developers, and researchers that will enable them to advance genetics education in the twenty-first century.

Rehovot, Israel

Michal Haskel-Ittah
Anat Yarden

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Part I
Reflecting Upon the Content of Genetic Curriculum

Chapter 1
Should We Give Peas a Chance? An Argument for a Mendel-Free Biology Curriculum

Kostas Kampourakis

1.1 The Problem: Mendelian Genetics in Today's Classrooms

Many people have probably had some genetics lessons in middle school and high school (hereafter referred to simply as school). Even if they have forgotten most of what they learned at that time, they probably remember Gregor Mendel and his experiments with peas. If they open any biology textbook and look for information about Mendel, they will likely read that he is an important figure in the history of biology: a man working in isolation and discovering the laws of nature. As textbook accounts often explain, Mendel discovered the laws of heredity but did not receive the recognition he deserved during his lifetime.

Here are some examples of such accounts from some widely used biology textbooks: "The study of genetics which is the science of heredity, began with Mendel, who is regarded as the father of genetics" (Biggs et al., 2009, p. 277); "The modern science of genetics was started by a monk named Gregor Mendel" (Miller & Levine, 2010, p. 262); "The ground work for much of our understanding of genetics was established in the middle of the 1800s by an Austrian monk named Gregor Mendel" (Nowicki, 2012, p. 167); "Modern genetics had its genesis in an abbey garden, where a monk named Gregor Mendel documented a particulate mechanism for inheritance (Reece et al., 2012, p. 262); "Gregor Mendel is considered to be the founding father of genetics" (Ward et al., 2008, p. 98); "For the next 35 years, his [Mendel's] paper was effectively ignored yet, as scientists later discovered, it contained the entire basis of modern genetics" (Walpole et al., 2011, p. 86). Note the common assumption in all textbooks: Mendel was doing genetics.

These textbooks also describe how Mendel discovered that characteristics are controlled by hereditary factors, the inheritance of which follows two laws: the

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law of segregation and the law of independent assortment. In the first case, when two plants that differed in one character, e.g., plants having seeds that are either round or wrinkled, were crossed, their offspring (generation 1) resembled one of the two parents and had round seeds. In generation 2, there was a constant 3:1 ratio between the round and the wrinkled character. Round shape was controlled by factor *R* that was dominant, whereas wrinkled shape was controlled by factor *r* that was recessive. The explanation of these results is that the factors (*R*/*r*) controlling the different characteristics (round/wrinkled) were segregated during fertilization and recombined in the offspring. This is described in textbooks as Mendel's law of segregation. When Mendel simultaneously studied the inheritance of two characteristics, e.g., both the shape of the seed and its color, he observed a similar but more complicated picture. When he crossed plants with yellow/round seeds and plants with green/wrinkled seeds, in generation 1, all offspring had yellow/round seeds. However, when those plants were crossed with each other, a constant ratio of 9 yellow/round: 3 yellow/wrinkled:3 green/round:1 green/wrinkled emerged in generation 2. Plants with factors *YY* or *Yy* had yellow seeds, whereas plants with *yy* had green seeds. The results suggested that the factors (*R*/*r* and *Y*/*y*) controlling the different characteristics (seed shape and seed color, respectively) were assorted independently during fertilization. As a result, all possible phenotypic combinations were obtained, and this is why they were observed in generation 2. This is described in textbooks as Mendel's law of independent assortment.

There are two problems here. This account distorts both the relationship between genes and traits, and the actual contribution of Mendel's work. In this chapter, I argue that teaching Mendelian genetics gives students a distorted view of heredity, without any actual gain in understanding. The imposed view is that genes determine traits, without any reference to the complexities and contingencies of developmental processes. At the same time, it also imposes a distorted view of the nature of science (NOS), portraying science as done by individuals. Therefore, it should be dropped altogether from genetics curricula, despite its intuitiveness for understanding and its heuristic value for research. In the following sections, I first explain why Mendelian genetics is misleading and then, after reviewing the relevant empirical research, I suggest that future research look into students' understandings in school curricula where Mendelian genetics have been replaced by developmental genetics. This chapter is not the first call for reconsidering what is taught in genetics (see, e.g., Jamieson & Radick, 2013; Redfield, 2012; Smith, 2014a, b). However, I must note that in this chapter, I am concerned with genetics teaching at schools and the misunderstandings that non-biologists may end up with because of it.

1.2 Current Knowledge About the Problem: Why Mendelian Genetics Is Misleading

1.2.1 Comparing the Effects of Different Teaching Approaches Is Not Informative

The question then becomes: what should teachers teach their students in school? One approach has been to reconsider the typical genetics curriculum, where teaching about genetics begins with Mendelian genetics and molecular genetics is taught afterwards. Another approach has been to compare the effect of a Mendelian genetics curriculum with that of a more development-oriented one. Overall, the empirical research in this domain is very limited, and therefore the conclusions that can be drawn are also limited.

One study compared two learning progressions which differed in the order in which Mendelian genetics and molecular genetics were taught. The aim was to see whether learning about molecular genetics first facilitates students' understanding of Mendelian genetics and vice versa. The study involved 117 students in the 7th grade. The researchers concluded that understanding molecular genetics may support students' subsequent understanding of Mendelian genetics. In contrast, understanding Mendelian genetics seemed to confer a weaker advantage for the subsequent learning of molecular genetics. The conclusion was that contrary to the usual practice, students might gain more if molecular genetics were taught before Mendelian genetics (Duncan et al., 2016).

In a similar study with undergraduate students, the instructor taught two groups of 52 students in two consecutive years following a different curriculum in each year. In the first year, Mendelian genetics was introduced before molecular genetics, whereas in the second year, it was the opposite. The comparison of the two courses showed no statistically significant differences in exam scores or final grades between the two approaches. The conclusion was that it might be better to focus on how to best present the content in Mendelian and molecular genetics, rather than on whether learning in one domain has any impact on learning in the other (Deutch, 2018).

In another study, 56 undergraduate students in their 1st and 2nd year in the UK took part in a study comparing two curricula: half of the students were assigned the classical Mendelian curriculum characterized by a strong genetic determinism component; the other half were taught a Weldonian curriculum with a weak genetic determinism component, emphasizing the importance of environment and developmental processes. The comparison of students' conceptions of genetic determinism before and after teaching showed that the students who had been taught the Mendelian curriculum did not undergo any change in their genetic determinism conceptions, whereas the students who had been taught the Weldonian curricula had less deterministic views about genes (Jamieson & Radick, 2017).

As already noted, the available research is limited, so limited conclusions can be drawn about whether or when Mendelian genetics should be taught. Empirical

research notwithstanding, there is another reason for which I think we should drop Mendelian genetics altogether from the genetics curriculum: it is simply inaccurate.

1.2.2 Mendelian Genetics Is an Oversimplified Representation of Heredity

In biology textbooks, inherited traits are conceived as being of two major types: (1) simple ones, which are considered to be determined by single genes, and (2) complex ones, which are considered to be affected by either several genes or one or more genes and environmental factors. In the second case, the complexities of development are often acknowledged in textbooks, and reference is also often made to multiple genes and environmental factors affecting a trait, as for example in the case of human height. So far so good. The problem now is that in the first case, the case of so-called monogenic traits, it is assumed that only one gene is involved and that its alleles determine one or the other version of the trait. Typical textbook examples are the shape and color of Mendel's peas, or eye color in humans. In the usual account found in textbooks, there only exist a few alleles "for" a particular version of a trait. For example, in the case of peas, round shape is controlled by the dominant allele *R*, whereas wrinkled shape is controlled by the recessive allele *r*. In the case of human eye color, the typical account is that brown color is controlled by the dominant allele *B*, whereas blue color is controlled by the recessive allele *b*. However, in both cases, the phenomena are quite different and much more complicated.

Let us begin with peas. The typical account of Mendelian genetics that there exist "factors" (alleles) for yellow or green seed color, or round or wrinkled seed shape is simply inaccurate. Wrinkled seeds have higher amounts of sucrose, fructose, and glucose, resulting in higher water uptake due to osmosis. What happens is that the starch-branching enzyme SBE1, which is involved in starch synthesis, is missing in wrinkled seeds, due to an interruption of the *SBE1* gene by an insertion of 800 nucleotide pairs. Because of the lack of SBE1, there is reduced starch biosynthesis, and therefore higher amounts of sucrose, fructose, and glucose (Bhattacharyya et al., 1990). Seed color (yellow/green) is regulated by alleles *I* and *i*, with the latter retaining seed greenness not only during seed maturation but also during senescence. This suggests that allele *i* is related to the *stay-green* (*SGR*) gene, which encodes the SGR protein that is involved in the chlorophyll catabolic pathway (the exact function is not clear). Three different *i* alleles have been found; one results in the insertion of two amino acids in the SGR protein, whereas the other two result in low or no production of this protein (Sato et al., 2007). Overall, it has been shown that the genes related to the traits that Mendel studied are not entirely independent as often assumed, and that the alleles result from a range of changes at the molecular level (see Reid & Ross, 2011 for an overview).

More broadly, the idea of "genes for" is incorrect anyway. Single genes are difference makers, that is, they bring about a change in the phenotype. But these genes alone are not sufficient to produce the phenotype (see Waters, 2007 for a detailed discussion). Adding salt to a meal makes the meal salty, but salt does not produce the salty meal as a whole. Other ingredients besides salt are also necessary. Therefore, salt is a difference-maker in turning a meal into a salty meal, but is not sufficient on its own to produce it. In short, salt is not a factor "for" a salty meal. This was already evident to Thomas Hunt Morgan and his colleagues more than 100 years ago, but we nevertheless persist in not teaching toward this kind of understanding:

> Mendelian heredity has taught us that the germ cells must contain many factors that affect the same character. Red eye color in Drosophila, for example, must be due to a large number of factors, for as many as 25 mutations for eye color at different loci have already come to light . . . Each such color may be the product of 25 factors (probably of many more) and each set of 25 or more differs from the normal in a different factor. It is this one different factor that we regard as the "unit factor" for this particular effect, but obviously it is only one of the 25 unit factors that are producing this effect … The converse relation is also true, namely, that a single factor may affect more than one character … Failure to realize the importance of these two points, namely, that a single factor may have several effects, and that a single character may depend on many factors, has led to much confusion (Morgan et al., 1915, pp. 208–210).

Therefore, the genes related to Mendel's characteristics are not "genes for" these characteristics. There is no allele for wrinkled or round peas; the shape of the seeds is the outcome of the function of an enzyme that synthesizes starch; wrinkled peas are produced because of a physicochemical phenomenon (osmosis) and not because there is an allele that determines this particular shape. Similarly, the color of seeds depends on the metabolism of chlorophyll and the proteins involved in it. Green color is therefore not produced by an allele "for" this color, but when the normal chlorophyll metabolism changes because of changes in the respective proteins. In other words, these two traits are not determined by specific genes, but they are affected by genes producing proteins involved in plant metabolism. In this sense, wrinkled shape and green color are the by-products of altered metabolic reactions, and not the products of any "genes for" seed shape or color. But this is not what students are taught at school. Rather, they are taught that there exist alleles for particular traits.

In addition to the characteristics that Mendel studied, another typical textbook example is the inheritance of human eye color. Textbook accounts often explain that a dominant allele *B* is responsible for brown color, whereas a recessive allele *b* is responsible for blue color. According to such accounts, parents with brown eyes can have children with blue eyes, but it is not possible for parents with blue eyes to have children with brown eyes. This pattern of inheritance was first described at the beginning of the twentieth century (Davenport & Davenport, 1907), and it is still taught in schools today, even though it became almost immediately evident that there were exceptions, such as two parents with blue eyes having offspring with brown or dark hazel eyes (e.g. Holmes & Loomis, 1909). The color of the eye

depends on the outer tissue layer of the iris, which is called the anterior stroma. It is the density and cellular composition of this layer that mostly affects the color of the iris. The melanocyte cells of the anterior stroma of the iris store melanin in organelles called melanosomes. When white light enters the iris, the latter can absorb or reflect a spectrum of wavelengths, giving rise to the three common iris colors (blue, green–hazel, and brown) and their variations. Blue eyes contain minimal pigment levels and melanosome numbers; green–hazel eyes have moderate pigment levels and melanosome numbers; and brown eyes are the result of high melanin levels and melanosome numbers (Sturm & Frudakis, 2004).

The inheritance of eye color, actually of iris pigmentation, is not as simple as is usually presented in textbooks. More than one gene has been found to be significantly associated with eye color. The strongest associations were initially found between eye color and the *OCA2* gene, which is located on chromosome 15 and encodes the melanosomal transmembrane protein OCA2 which affects melanosome maturation (Frudakis et al., 2003). Eye color is therefore best described as a polygenic character, i.e., one to which multiple genes contribute (Sturm & Frudakis, 2004). Nevertheless, it seems that particular genetic variants, located close to one another, account for blue eye color. It has been found that three single-nucleotide polymorphisms (SNPs) in intron 1 of the *OCA2* gene have the highest statistical association with blue eye color (Duffy et al., 2007; see also Frudakis et al., 2007). Other studies have shown that SNPs in the introns of gene *HERC2,* also on chromosome 15, are strongly associated with blue color. It is assumed that the variants within the *HERC2* gene are related to the expression of *OCA2*, and that it is the decreased expression of the latter in iris melanocytes that is the cause of blue eye color (Eiberg et al., 2008; Kayser et al., 2008; Sturm et al., 2008; Sulem et al., 2007). Therefore, certain variants in *OCA2* and *HERC2* genes are significantly associated with blue eye color; however, it must be noted that these associations differ in different populations (Edwards et al., 2016). In this sense, one could say that there exist alleles "for" blue color, but these are not the alleles of a single gene. Furthermore, even if such a model worked in many cases, it would still be insufficient to explain the full range of eye color phenotypes that has been documented in research (Sturm & Larsson, 2009).

1.2.3 The (Stereo)typical Story of Mendel Provides a Distorted View of the NOS

Mendel's results were presented at the meetings of the Brno Natural Science Society on February 8 and March 8, 1865, and were published in the society's journal in 1866. At the beginning of his paper, Mendel expressed his aim "to follow the development of hybrids in their descendants." He also noted that "a universally valid law describing the formation and development of hybrids has not yet been established." It is important to note that Mendel was interested in studying the transmission of

characteristics over generations bred from hybrids, and to better understand how this happens. In his paper, Mendel described the transmission of characteristics rather than that of hereditary particles such as genes. In particular, Mendel observed that the hybrids obtained from the various crosses between different varieties were not always intermediate between the parental forms. In contrast, some hybrids exhibited certain characteristics exactly as they appeared in the parent plants. Mendel called dominant the parental characteristics that appeared in the hybrids, and recessive those that did not appear in the hybrids but that reappeared fully formed in the next generation. Thus, Mendel studied and wrote about characteristics and not about hereditary particles, and so did not discover that heredity was particulate in nature. More generally, he studied hybridization and not heredity, and it should therefore come as no surprise that the term "heredity" does not appear in his paper. A careful study of his paper also shows that, strictly speaking, Mendel did not discover the two laws commonly attributed to him; rather, he observed their consequences under the particular experimental conditions. Statements that look like the laws of segregation and independent assortment can be found in his paper, but they are not explicitly described as such; they are also quite different from the way in which they are currently described in textbooks and elsewhere (see Kampourakis, 2015 for a more detailed account).

At that time, the mechanism of heredity was at the center of biological thought, in part because Charles Darwin's theory of descent with modification through natural selection (published in 1859 in the *Origin of Species*) lacked a complementary theory that could explain the origin and inheritance of the new variations that were so central to it. In response to this problem, Darwin proposed, in 1868, his *Provisional Hypothesis of Pangenesis*, according to which all parts of the body participate in the formation of the offspring by producing microscopic entities, the gemmules, which somehow carry the organismal properties from one generation to the next. This was followed by other theories, such as those by Herbert Spencer, Ernst Haeckel, Francis Galton, William Keith Brooks, Carl von Nägeli, Hugo de Vries, and August Weismann. All of these scientists were aware of one another's work and practically formed a scientific community, actively and interactively working to develop a theory of heredity. Mendel is nowhere in this picture. Only Nägeli came to know of Mendel's experimental work, through their correspondence from 1866 until 1873. Following Nägeli's advice, Mendel worked on *Hieracium* (hawkweed, a genus of the sunflower family) from 1866 to 1871, which gave different results from those of peas. Nägeli did not seem to pay much attention to Mendel's work, although he cited Mendel's 1866 paper on at least one occasion (Allen, 2014; Kampourakis, 2013).

Most importantly, the Brno Natural Science Society sent more than 100 copies of the journal that included Mendel's paper to scientific centers around the world. At least 10 references to Mendel's paper appeared in the scientific literature before 1900, some of them in books that were widely read by naturalists. Therefore, it would have been possible for Mendel's work to become more widely known during his lifetime. Why did it not? Probably because it was not an explicit attempt to develop a theory of heredity, which was of interest to naturalists at that time. Rather,

Mendel was interested in understanding hybridization and its patterns, which would be of practical, agricultural interest. It was in this practical, local context that Mendel's work made sense in his day (Olby, 1985; Orel & Wood, 2000).

However, important developments during the latter half of the nineteenth century would later provide a new context for reading Mendel's paper. On the one hand, first Galton and then Weismann developed frameworks of "hard" heredity, i.e., heredity characterized by discontinuous variation and nonblending characteristics. Both Galton and Weismann rejected the idea of the inheritance of acquired characteristics; moreover, Galton postulated and Weismann established the idea of the germline, i.e., that reproductive cells exist independently of the other cells of the body. In the late 1870s, Walther Flemming observed and described mitosis, and Oscar Hertwig observed and described meiosis. In the 1880s, Eduard Strasburger concluded that fertilization involves the fusion of two nuclei, and Edouard van Beneden described how this takes place at the chromosomal level. Theodor Boveri's experiments during the 1890s provided evidence for the role of the nucleus and chromosomes in heredity (Bowler, 1989, pp. 74–92; Carlson, 2004, pp. 23–28). In 1900, Hugo de Vries, Carl Correns, and Erich von Tschermak published the results of their research on plant hybridization, which agreed with those obtained by Mendel. This simultaneous "rediscovery" brought Mendel back on the scene. At that time, Mendel's paper was considered to bring together the findings of breeding experiments and cytology, showing that particulate determinants existing in the nucleus of the cell are segregated and independently assorted (see Brannigan, 1981, pp. 90–96; Olby, 1985, pp. 109–133).

After 1900, the work of Mendel guided the development of the new science of "genetics," a term coined by William Bateson. His book *Mendel's Principles of Heredity: A Defence,* published in 1902, contains the first English translation of Mendel's paper. In that book, Bateson presented Mendel's work as providing the solutions for various problems relevant to heredity. However, in the same year, Raphael Weldon showed that Mendel's "laws" might not actually work, even for peas. Weldon's studies of varieties of pea hybrids led him to conclude that there was a continuum of colors from greenish yellow to yellowish green, as well as a continuum of shapes from smooth to wrinkled. It thus appeared that in obtaining pure-bred plants for his experiments, Mendel had actually eliminated all natural variation in peas, and that characteristics were not as discontinuous as he had assumed (Jamieson & Radick, 2013; Weldon, 1902). So, less than two years after the rediscovery of Mendel's work, the generalizability of his conclusions was questioned.

However, Mendel's work helped new observations make sense, as well as producing new observations in the first place. Perhaps the most interesting immediate implication was the understanding of the role of chromosomes in heredity. In 1903, Walter Sutton provided cytological evidence that explained Mendel's ratios, based on the understanding of meiosis at the time. Sutton was concerned about recent observations indicating that the maternal and paternal chromosomes remain independent. This implied that reproductive cells should contain either the maternal or the paternal chromosomes. Sutton performed a careful study of the process of cell division and concluded that a large number of different combinations of maternal

and paternal chromosomes are possible in an individual's mature reproductive cells. Sutton's insight brought cytology and genetics even closer together, laying the foundations for explaining the physical basis of Mendel's ratios and for understanding the chromosomal nature of heredity (Hegreness & Meselson, 2007).

Getting the history of science right is very important, because the textbook section on Mendelian genetics provides numerous opportunities for teaching about the NOS. The usual presentation of Mendel's life and work in textbooks also includes instances of general NOS aspects that could be discussed in schools. In a study of how seven widely used high-school biology textbooks represented aspects of NOS knowledge and scientific inquiry in the Mendelian genetics sections, it was found that many such aspects existed but that most of them were implicit. Overall, 237 instances of NOS knowledge and 128 instances of scientific inquiry aspects were counted in 140 textbook excerpts. Of these 365 instances, 362 were implicit and only 3 were explicit (all scientific inquiry aspects). The conclusion of that study was that the sections on Mendelian genetics might provide teachers with a multitude of opportunities to teach about NOS knowledge and scientific inquiry explicitly, yet these opportunities are likely to be missed because almost all of the relevant aspects are implicit (Campanile et al., 2015).

1.3 Remaining Issues: Would a Developmental Genetics Curriculum Work as an Alternative?

Elsewhere, I explained in detail that if genes do anything at all, they are implicated in the development of traits and they account for variation in traits (Kampourakis, 2017). Genes do not determine traits in any sense similar to the simplistic Mendelian genetics account, and this is not new. See, for instance, the above-quoted text written by Thomas Hunt Morgan and his students more than 100 years ago (Morgan et al., 1915, pp. 208–210). Simply put, Morgan and his colleagues were aware, more than 100 years ago, that many genes contribute to the same effect and that a change in one of them might bring about a change in the trait. This is very different from the simplistic Mendelian genetics notion of genes determining traits, which is still taught in schools. Most interestingly, Morgan et al. (1915) noted that the failure to realize that a single gene can have many effects and that a single effect may depend on many genes has led to much confusion. Do we explain that a single gene can have many effects and that a single effect may depend on many genes when teaching Mendelian genetics? I think not.

I therefore suggest that what we should investigate is whether teaching genetics from a developmental perspective in schools would be more effective. The development of tissues and organs, and eventually, the production of the adult form, is not only controlled by genes or DNA but also by the exchange of signals among cells. These signals consist of gradients of signaling proteins. Whatever a cell does depends on the kind of signals it receives from its immediate environment. Therefore,

neighboring cells are interdependent, and it is the local interactions among cells that drive the developmental processes. These localized processes also make the development of different organs relatively independent, which allows for changes in each organ independently from other organs. During development, cells multiply, differentiate, and migrate to various parts of the developing organism. This happens in a coordinated manner, but without any centralized coordination of development; cells simply respond to signals from their local environment. Genes are involved in the production of proteins that are in turn involved in signal production, signal reception, and signal response. Genes are therefore implicated in this unconscious coordination of development, but they in no way determine its course or its outcomes (Davies, 2014, pp. 132, 251–252).

1.4 Implications for Teaching

Let me give a concrete example of how genetics could be taught from a developmental perspective. A textbook includes the following information about the *SRY* (sex-determining region on the Y chromosome) gene (Sadava et al., 2011, p. 257):

> These observations suggest that the gene controlling maleness is located on the Y chromosome. Observations of people with other types of chromosomal abnormalities helped researchers to pinpoint the location of that gene:
>
> - Some women are genetically XY but lack a small portion of the Y chromosome.
> - Some men are genetically XX but have a small piece of the Y chromosome attached to another chromosome.

The Y fragments that are respectively missing and present in these two cases are the same and contain the maleness-determining gene, which was named *SRY* (sex-determining region on the Y chromosome). The *SRY* gene encodes a protein involved in primary sex determination—that is, the determination of the kinds of gametes that an individual will produce and the organs that will make them. In the presence of the functional SRY protein, an embryo develops sperm-producing testes.

Another textbook provides a similar account (Raven et al., 2011, p. 1086):

> If the embryo is a male, a gene on the Y chromosome converts the indifferent gonads into testes. In females, which lack a Y chromosome, this gene and the protein it encodes are absent, and the gonads become ovaries. An important gene involved in sex determination is known as *SRY* (for "sex-determining region of the Y chromosome"). Once testes have formed in the embryo, the testes secrete testosterone and other hormones that promote the development of the male external genitalia and accessory reproductive organs. If the embryo lacks the *SRY* gene, the embryo develops female external genitalia and accessory organs. In other words, all mammalian embryos will develop into females unless a functional *SRY* gene is present.

What both textbooks imply, if not explicitly suggest, is that the *SRY* gene determines sex. If students have been taught Mendelian genetics before talking about this gene, they could even design crosses and show how this gene would be present in males but not females, and how it would be its presence or absence that determines

biological sex. However, the (developmental) story is more complicated than this. Whereas it is certainly the case that a mutation in the *SRY* gene is enough to make an XY individual develop as a female with underdeveloped reproductive organs (Jäger et al., 1990), it has also been found that a translocation of part of the Y chromosome including the *SRY* gene onto the X chromosome in humans makes an XX individual develop as a "true hermaphrodite" (a medical term for a form of *intersexuality*, i.e., carrying both male and female gonadal tissues) (Margarit et al., 2000). But these instances do not justify the statement that *SRY* is the gene for sex. The *SRY* gene is a gene on the Y chromosome that is indeed related to the development of male features. The default developmental outcome for the human embryo is to become a female. The expression of the *SRY* gene is what makes the difference in the outcome, because it affects a pathway that guides the development of the male or female sexual organs.

In this sense, it is more appropriate to state that *SRY* makes a difference for the development of sex. Embryos carrying the Y chromosome and the *SRY* gene develop testes and a male reproductive system, whereas those not having either the Y chromosome or the *SRY* gene develop ovaries and a female reproductive system (Davies, 2014, pp. 147–151). However, if one looks carefully at the details of the process, several proteins (and therefore genes) are involved in sex differentiation. The bipotential precursor of gonads (testes and ovaries) is established by various proteins, including SF1 and WT1, the early expression of which might also initiate that of SOX9 in both sexes. β-Catenin can begin to accumulate at this stage, and in XX cells, its levels can repress SOX9 production. However, in XY cells, increasing levels of SF1 activate the production of SRY that, along with SF1, enhances SOX9 expression. If SRY activity is weak, low or late, there is no SOX9 expression as β-catenin levels accumulate and shut it down. In the testis, SOX9 promotes the testis pathway, and it can do so even in the absence of SRY (Sekido & Lovell-Badge, 2009). Therefore, the *SRY* gene does nothing on its own. Sex is the outcome of a complex developmental process that involves several factors, and to understand their effect one has to consider the whole process of sex development. Of course, it is not possible to teach all the complexities of development to school students. Rather, they must be given a sense of these complexities.

Let me give an example of how this might be done, in order to show that genes do not control anything, but operate within cellular environments which affect their expression. If two people use the same cookbook (genome) and cook some food, the outcome (phenotype) could be very different, even though both followed the same recipe (genes). The expression of the information in the cookbook (DNA or genes) depends on the cook (developmental system) who implements it. Consequently, it is useful to mention development alongside heredity, particularly for multicellular organisms, as developmental processes may produce outcomes that differ from those expected by reading the DNA sequences alone. Simply referring to the recipe (genes) as that which determines the phenotype (meal) is wrong, because the outcome depends on how the recipe is implemented. But this is exactly what is taught in Mendelian genetics, and this is why a discussion of developmental processes alongside genetics might help.

It might therefore be more appropriate for genetics education to design curricula and teaching materials that present the role of genes in their developmental contexts, and to investigate how students can arrive at an understanding of this. If the aim is to educate scientifically literate citizens, then we should refrain from teaching students about genes that control or determine traits. Rather, we should try to teach students the biology of our times and give them a sense of the complexity of genomes. One suggestion, that still has to be empirically tested, is to teach students not about genes, but about the genetic material, based on the following definition (Burian & Kampourakis, 2013, p.624):

> Genetic material: any nucleic acid with the propensity to be inherited and to interact with other cellular components as a source of sequence information, eventually affecting or being implicated in cellular processes with local or extended impact.

This definition is more accurate and inclusive than the typical definitions of the gene. It allows a clear distinction between genetic and epigenetic inheritance, and it would free textbooks from referring to "gene(s) for" particular characteristics or diseases. Instead, it would allow them to refer to particular parts of the genetic material that, in identified contexts, interact with each other and with other cellular components to affect the production of molecular, cellular, or organismic characteristics, or to increase the susceptibility of affected individuals to acquiring certain traits or diseases.

It might then be simpler to drop Mendelian genetics altogether from the genetics curricula. In doing so, we do not dishonor Mendel and his contribution. As Greg Radick (2016) nicely put it: "If we want to honour Mendel, then let us read him seriously, which is to say historically, without back-projecting the doctrinaire Mendelism that came later. Study Mendel, but let him be part of his time." This means that if we teach about Mendel, we should teach about what he did and why he did it in its own context, and not present his work in anachronistic terms. Mendel's story can provide useful lessons for the NOS (e.g. Kampourakis, 2013), but not for today's genetics.

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Chapter 2
How Can Epigenetics be Used to Integrate Nature and Nurture in Genetics Education?

Niklas Gericke

2.1 The Problem

2.1.1 The Conception of Genetic Determinism

The 'nature versus nurture' debate is old and traditional, but to some extent outdated. All biologists today recognize that any organism's phenotype (set of traits) emerges from the interaction between its genome (nature) and environment (nurture). Arguably, this was always obvious. For example, everyone knows that children tend to look similar to their parents, but malnourishment stunts growth and overeating makes people fat. However, the degrees to which specific traits of an organism are influenced by its genes or the environment vary considerably at the population level. For example, human eye color is predominantly biologically determined, whereas high blood pressure is mostly attributed to environmental factors. Such examples highlight the importance of both nature and nurture, but can problematically imply a dichotomous, *either/or* relationship between them (as implied by the term *versus*). In teaching and education, this often translates to an additive model of nature and nurture relationships at the individual level, in which contributions of an organism's genetic composition and environmental factors are described in terms of percentages. This then clouds the scientific understanding that 'biological identities' (phenotypes) result from the constant reciprocal interaction between genes and environments (Forissier & Clément, 2003).

The separation of nature and nurture in the additive model could be a major source of learning problems in genetics education because it leads to the systematic neglect of environmental factors in the models used to teach gene function (Gericke & Hagberg, 2007, 2010a, b). Instead, the causal or biologically essential aspects of

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genes and DNA are emphasized in teaching genetics, as reflected in consistent reports of the prevalence of genetically deterministic conceptions among students (e.g., Jamieson & Radick, 2017; Lewis & Kattmann, 2004; Shaw et al., 2008). Here, genetic determinism is defined as the belief that genes influence organisms' traits more strongly than warranted by scientific consensus (Gericke et al., 2017). In this chapter, I argue that inclusion of epigenetics in genetics and biology curricula provides a way to counter genetic deterministic conceptions among students.

Epigenetics is a new and rapidly expanding field of biological research that focuses on heritable changes that play important roles in interactions between the environment and genes, and thus the environment's influence on organisms' (including humans') behavior and other traits. This fundamental new knowledge lies at the heart of the classical debate about the relative importance of genes and the environment, enabling our understanding to be strongly refined. The focus is no longer on the degree to which the environment or genes determine organisms' traits, but rather on the processes involved in the interactions between environmental and genetic factors that lead to observable epigenetic patterns in cells, which in turn regulate biological characteristics and social behavior. These processes provide biological memory systems,[1] in addition to DNA sequences, that respond to, and are affected by the surrounding milieu, which starkly conflicts with a genetic deterministic conception.

From a molecular perspective, epigenetics plays a major role in the regulation of gene expression without interfering with the nucleotide sequence of the DNA. Epigenetic mechanisms show that characteristics of humans and other organisms depend interactively on both inheritance (nature) and the environment (milieu). All this can be summarized in a definition of epigenetics as "the study of changes in gene function that are mitotically and/or meiotically heritable and that do not entail a change in DNA sequence" (Russo et al., 1996, p. 1). However, as outlined in this chapter, epigenetics encompasses complex concepts that can be described and explained in multiple ways.

The field of epigenetic research has grown exponentially in recent decades, and this remarkable development in the biological sciences has been transferred, to some degree, into a societal discourse (Meloni & Testa, 2014). Hence, it has been covered in the last decade by both popular science books (e.g., Francis, 2011) and television documentaries (such as the BBC's "Ghost in your genes" and "The hidden life of our genes"). Nevertheless, the very few studies that have addressed the public understanding of epigenetics have generally found it be very weak. For example, a study published 10 years ago found that the lay public's knowledge of the topic in the USA was so limited that investigators first had to teach focus groups what epigenetics was before asking them what they thought of it (FrameWorks Institute, 2010). As concluded by Landecker and Panofsky (2013, p. 351) "compared with the public understanding of genetics, epigenetics remains a drop in the

[1] The term 'cell memory' here refers to acquired epigenetic marks that regulate gene activity, are stable and survive mitotic, and possibly also meiotic, cell divisions.

bucket." One major reason for epigenetics' lack of penetration into the public consciousness may be that it is not included in the school curriculum, and thus not taught in secondary schools. In this chapter, it is argued that the omission of epigenetics should be addressed to promote a more contemporary understanding of genetics, moving away from genetic deterministic conceptions.

Genetic determinism, with associated causal explanations such as 'one gene for intelligence' or the 'cancer gene', has been widely disseminated in the public discourse, both as personal convictions (Nelkin & Lindee, 2004) and in the media (Carver et al., 2008). A problem with this misconception is that research has found that genetic determinism is used to defend established differences between social groups defined in terms of, for example, gender, ethnicity, or class (Shostak et al., 2009). Hence, these differences are referred to as genetically determined, i.e., 'natural', and therefore justifiable. As a result, genetic determinism becomes a democratic problem since this misconception seems to sustain undemocratic values. Thus, it is important to identify the reasons why this misconception is so strong, and ways to counter it (Dar-Nimrod & Heine, 2011). A major line of argument in this chapter is that current school biology curricula, and how they are enacted, facilitate rather than hinder the development of genetic deterministic conceptions among students and the public. A second line is that epigenetic mechanisms provide an explanatory model that shows how the environment and genes have strongly interactive functional effects, which could refute genetic deterministic beliefs.

Previous studies have shown that much of the genetic knowledge conveyed in comprehensive school and upper secondary school is partly based on outdated explanatory models rooted in classical and Mendelian genetics (Dougherty et al., 2011; Gericke & Hagberg, 2010a, b). These kinds of models promote a causal and deterministic understanding of genetics and biology because they portray genes as the causes of the occurrence of specific traits, as extensively discussed by Gericke and Smith (2014). In accordance with this selection and teaching of genetics content, studies have abundantly and repeatedly shown that students mainly have a causal and deterministic understanding of genes and their functions (e.g., Jamieson & Radick, 2017; Lewis & Kattmann, 2004; Venville & Treagust, 1998).

To assist efforts to counter the establishment of genetic determinism, in this chapter I advocate a shift in the teaching of school biology to a more integrated model, including much more attention to the interactions between genes and the environment. As further outlined below, epigenetics provides new teaching models that clearly highlight these interactions and demonstrate the shortcomings of genetic deterministic beliefs.

2.2 Current Knowledge About the Problem

2.2.1 Teaching and Learning Genetics

Research in genetics education has repeatedly shown that students have difficulties distinguishing between genotype and phenotype (e.g., Haskel-Ittah & Yarden, 2017; Lewis & Kattmann, 2004). Studies of students' understanding of this relationship have found indications of a set of underlying and partially overlapping mental models of genes and their functions, summarized by Gericke and Smith (2014) as follows. Genes are:

- inherited particles that are transferred from one generation to the next (Duncan & Reiser, 2007; Lewis & Kattmann, 2004; Venville & Treagust, 1998);
- the sole determinants of characteristics (Lewis & Kattmann, 2004);
- objects with inherent activities, i.e., a gene is a physical object that acts in an unalterable way in an organism (Martins & Ogborn, 1997);
- sets of commands that control characteristics (Martins & Ogborn, 1997; Venville & Treagust, 1998);
- active particles that control characteristics (Duncan & Reiser, 2007; Venville & Treagust, 1998); and/or
- biochemical sequences of instructions connecting genes and protein synthesis, and protein synthesis and phenotype (Venville & Treagust, 1998).

In the first category, no link is made to traits at all; instead, only the hereditary aspect is mentioned. The next four categories (the most frequently found conceptions among secondary-school students in all studies) display a strongly causal and deterministic understanding of gene–trait relationships. These conceptions are strongly aligned with genetic deterministic views, attributing such importance to genes that students often view them as independent actors that control traits with no environmental influence. The conception expressed in the last category in the bullet list more resembles a mechanistic understanding of gene function, recognizing that other biochemical molecules in cells participate in the processes involving genes. This understanding of gene function should be promoted in secondary education, as it is most consistent with current scientific understanding. However, as shown in the cited studies, this understanding of gene function is rare among secondary students, and the environmental aspect is still missing. Why, then, are genetic deterministic conceptions so widespread? Some possible explanations are described in the following paragraphs.

In psychological research, it is well-established that children have a tendency to think essentialistically, i.e., they believe that organisms have a fixed essence (Gelman, 2003). For example, children commonly believe that organisms of the same taxonomic group share underlying common invisible properties from which inferences about the organisms' characteristics can be drawn (Gelman & Markman, 1986); even if the organisms undergo substantial changes, they keep their original essential identity (Rosengren et al., 1991). Hence, it seems that children believe that

organisms are characterized by an underlying essence. Dar-Nimrod and Heine (2011) suggested genes as the physical entity to which this essence could be linked, which would be manifested in genetic deterministic beliefs. In line with that conclusion, Stern and Kampourakis (2017) suggested that the underlying genetic essentialist ideas might be a cause for students' genetic deterministic thinking. Hence, this might be one explanation for the results in many studies of students' understanding in genetics, as outlined here.

Another explanation for students' recurrent genetic deterministic conceptions, proposed by Gericke and Hagberg (2007), is that they are promoted by current curricula and teaching that focus on explanatory models rooted in Mendelian and classical genetics, and the central dogma when teaching molecular genetics and gene function. This argument is empirically supported by findings of curriculum studies that (for example) 85% of 'standards' set by the US states and District of Columbia for school biology paid excessive attention to Mendelian genetics (Dougherty et al., 2011, p. 318). Hence, the standards in virtually every state have failed to keep pace with changes in the discipline as it has become genomic in scope, omitting concepts related to genetic complexity, the importance of environment to phenotypic variation, differential gene expression, and the differences between inherited and somatic genetic diseases (Dougherty et al., 2011). Furthermore, it has been shown that genetic deterministic conceptions are common in textbooks (Clément & Castéra, 2013; Gericke et al., 2014) and among teachers (Castéra & Clément, 2014). Hence, current curricula and teaching practices seem to promote, rather than hinder, simplistic and genetic deterministic conceptions.

Regardless of whether genetic deterministic conceptions and associated problems stem from students' essentialist preconceptions or the way in which we select content and organize its teaching, the conceptions can be countered. For example, Jamieson and Radick (2017) found that a teaching intervention focused on a developmental and interactionist perspective of gene function resulted in undergraduate students having fewer genetic deterministic conceptions than peers taught according to the traditional Mendelian curriculum. However, and this is less encouraging, a large-scale Brazilian study detected no correlation between knowledge of gene–environment interactions, or genomics, and genetic deterministic conceptions (Gericke et al., 2017). Hence, the strength of the relationship between the level of knowledge about genes and genetic deterministic beliefs is still an open question. An often suggested way of improving teaching about genes' functions is to highlight the roles of proteins as intermediates between genes and traits, in order to promote mechanistic reasoning (Duncan et al., 2009; Thörne & Gericke, 2014; van Mil et al., 2013). Accordingly, recent studies have shown that various learning activities can increase students' ability to relate the concepts of gene/DNA and trait by introducing the protein concept (Haskel-Ittah & Yarden, 2017), and that it might be productive to teach students about proteins' functions (Haskel-Ittah et al., 2020). However, environmental factors are still neglected in these approaches.

In a couple of articles, Pierre Clément and colleagues addressed how the environmental factor can be addressed in different teaching models (Clément & Castéra, 2013; Forissier & Clément, 2003). In a purely genetic deterministic model,

environmental factors are totally omitted, as shown in Fig. 2.1a. Nature (genotype) and nurture (environment) are regarded as mutual, but independent, contributors to the phenotype in an additive model (Fig. 2.1b). The phenotype is regarded as being jointly and interactively produced by the genotype and environment in a simple integrated model (Fig. 2.1c). Finally, in an interactive multilevel model, or what I would refer to as an epigenetic model, both the genotype and phenotype interact with the environment and each other across the multiple organizational levels spanning genes and associated traits (Fig. 2.1d). This far from deterministic model

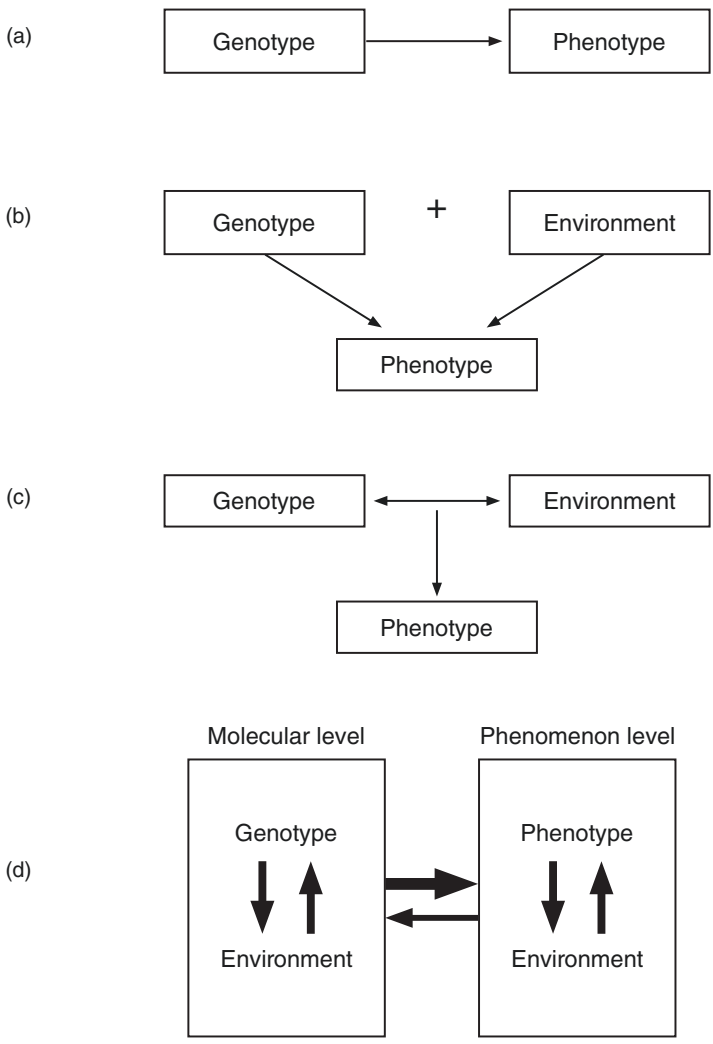

Fig. 2.1 Visual representations of four models—(**a**) purely genetically deterministic, (**b**) additive, (**c**) simple integrative, and (**d**) interactive multilevel—of the relationship between genotype and phenotype. (Modified by Gericke from Clément & Castéra, 2013)

encapsulates the way that we would like our students to understand genetics. Moreover, as I claim here, epigenetic models provide potent teaching tools that can portray and foster a combination of mechanistic reasoning at the molecular level and conceptual understanding at the phenomenological level.

Most current education reproduces the first two models of nature–nurture relationships, seldom mentioning gene regulation and feedback processes connected to the environment (e.g., Clement & Castéra, 2013; Gericke et al., 2014). Thus, it is easy to get the impression that genes reign supreme, and there is a clear need to develop genetics education to include an epigenetic perspective. However, first we need to address what epigenetics really is and how it can be included in the biology curriculum.

2.3 Remaining Issues

2.3.1 Transforming Epigenetics for School Biology

In the previous section, I proposed that an understanding of the epigenetic model could counter genetic deterministic conceptions, but this raises a question: how should epigenetic concepts be included in school biology curricula and teaching to accomplish that goal? A school subject is not a mere distillation of the corresponding academic discipline. The content must be transformed in implicit and explicit processes at various levels, first into a school curriculum and then into taught knowledge in the classroom (Gericke et al., 2018). Various theories of teaching have addressed and highlighted the importance of addressing these transformation processes, which have been called (for example) recontextualisation by Bernstein (1971) and didactic transposition by Chevallard (1989). As outlined in the previous section, and discussed in detail by Smith and Gericke (2015), the problem today is that this transformation process leads to genetics being portrayed in school as fundamentally deterministic (genes cause traits to occur). Thus, the key lies in identifying ways to transform school genetics by including epigenetic elements of the academic genetics discipline regarding the trait-forming interactions between genes and their milieu. This is far from trivial, and to do it in a systematic and informed way, we first need to define and discuss what epigenetics is from a disciplinary perspective. Epigenetics, like most sciences, has a long history, evolving within different academic sub-disciplines with different ontological and epistemological assumptions (Haig, 2004). It is important for both researchers and biology educators to be aware of these historical developments in the discipline, so that they can make informed decisions about the transformation of epigenetics into school curricula. Thus, in this section, I provide a general overview of the historical development of epigenetics, the two main coexisting traditions, and associated meanings and definitions of epigenetic concepts.

In genetics, the gene is the most fundamental concept, and it is operationally defined on the basis of four phenomena: genetic transmission, genetic recombination, gene mutation, and gene function (Portin, 1993). These phenomena are interdependent. Thus, for example, we typically cannot observe gene function or gene mutation without transmission. Function refers here to the roles that a specific gene plays in the manifestation of specific phenotypic characteristics or traits in a given organism (or taxon). It should be mentioned that the term phenotype may, unhelpfully, refer to either the entire set of an organism's traits (all observable characteristics, such as its morphological, biochemical, physiological and behavioral properties) or just a single trait (Lewontin, 1974).

For a long time, there was no understanding of how genes function, and the process involved was treated as a black box, with knowledge restricted to the end points, i.e., the identification of steadily increasing numbers of genes on chromosomes and associated traits. This resulted in a causal or even deterministic explanatory model of gene function in classical genetics because genes were described as the causal agents for biological traits, with no acknowledgement of any environmental influence on those traits (Schwartz, 2000). With the shift to molecular genetics in the 1950s, the black box of gene function was cracked open by establishing that genes encode proteins. These findings triggered a major paradigm shift, i.e., presentation of the pathway from information embedded in genetic material to mature proteins, leading to the so-called "central dogma of molecular biology" (Crick, 1958). The original expression of the central dogma of molecular biology states that proteins are constructed in a specific unidirectional order. This means that information that has been transcribed and translated into proteins cannot make its way back to the genetic material. Thus, information cannot be transferred from protein to DNA or between proteins (Crick, 1958). This portrayal of gene function provided a biochemical process explanation of what happens in the black box, but reinforced and strengthened a deterministic explanatory model for the relationship between genotype and phenotype. Moreover, the idea explicitly evolved into a dogmatic view of protein synthesis, which was subsequently considered one of the cornerstones of biology education (Cobb, 2017; Wahlberg & Gericke, 2018) and, as argued here, might be a major cause for the widespread deterministic conceptions among students.

In recent decades, advances in gene technologies have further increased our understanding of the molecular processes involved in genes' functions, and the recognition of their complexity and flexibility (Gerstein et al., 2007). The sharp focus on the end products of the central dogma, genes and proteins, has not faded, but the importance of other structures and molecules (inter alia, RNA, ribosomes, and myriad signaling agents) has become increasingly recognized. Accordingly, more attention is being paid to interactionist models over causal ones. The center of interest has shifted to ways in which the genetic material interacts with itself, other biological molecules and the surrounding environment, rather than how genes produce proteins in a causal way. At the heart of this development is the research field of epigenetics that has emerged as perhaps the most promising area of research in unpacking the black box of gene function. Epigenetic concepts not only provide an

interactive framework for understanding genetics; by including the environment in the explanation, they greatly extending the linear model of the central dogma. Hence, epigenetics provides an integrated model combining nature with nurture and, as I argue in this chapter, the possibility of counteracting students' prevalent and resistant genetic deterministic conceptions. However, before elaborating on that line of argumentation we first need to discern what epigenetics is, and how this field of knowledge evolved.

2.3.2 Epigenetics at the Organism Level

Epigenetics is a polysemous concept,[2] and there are historical reasons for this (Haig, 2004). The term was first coined nearly 80 years ago by Waddington (1942) in an attempt to merge ideas emerging from research on organisms' development (experimental embryology) and inheritance (genetics). Waddington (1942) explicitly used the term 'epigenetics' to stress that an organism is not pre-formed in the zygote to simply unfold during ontogenesis, as it would be if genetic deterministic views were valid. Rather, he thought that the zygote was progressively constructed during development in an interactive process involving genes, other biological constituents of the fertilized egg, and the milieu (Nicoglou & Merlin, 2017). Hence, Waddington's ideas stemmed from a developmental perspective. He explicitly defined epigenetics as the study of the relation between phenotypes and genotypes by which "the genes of the genotype bring about phenotypic effects" (Waddington, 1942, p. 18). In other terms, he concluded that epigenetics was the study of the epigenotype, the whole complex of developmental processes that dynamically link genotype and phenotype. Waddington introduced the ideas of 'phenotypic plasticity' and 'epigenetic landscape', which meant that the developmental process of a cell or organism could take different paths, depending on environmental influences (Noble, 2015). He visually represented the epigenetic landscape with 'valleys' and 'forks' and the developmental process as a series of 'decisions' leading to various 'canals' separated by 'ridges' (see Fig. 2.2). A small change in a slope in the landscape could lead to one canal (developmental pathway) being favored over another, as illustrated in Fig. 2.2 by the passage of a ball. At the top of the slope it may be 'canalized' to the left (Fig. 2.2a) or right (Fig. 2.2b), depending on the environmentally determined gradients at each fork, thereby irreversibly locking the ball (developmental process) in a specific path as it proceeds down the slope. This clearly visualizes the idea that phenotypic outcomes (traits) are determined not only by the starting points—the genes, but also by how the genes interact with the environment and hence the paths taken.

According to Haig (2004), Waddington's work led to one traditional view within the biological sciences of epigenetics that focused on phenomena at the level of

[2] A concept with multiple meanings.

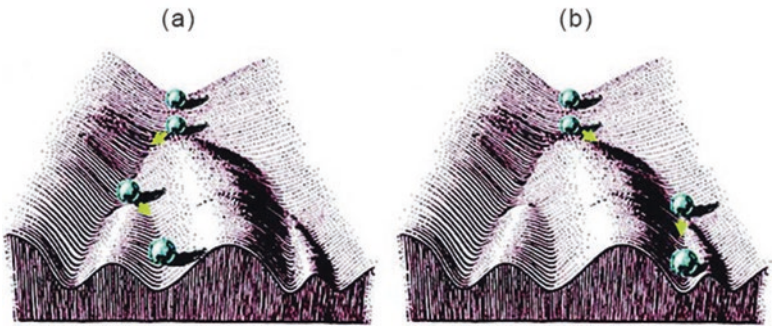

Fig. 2.2 Waddington's landscape of epigenetic development. The landscape and the ball at the top are from the original figure in Waddington (1942). Subsequent positions of the ball have been added to illustrate that development may follow different routes (**a** and **b**) through environmental 'canalization'. (Reproduced with permission from The Journal of Experimental Biology, modified diagram by K. Mitchell from Noble, 2015)

whole developing organisms, i.e., a holistic notion of epigenetics (at least for multicellular organisms). Huxley (1957, p. 1) followed this tradition and used epigenetics "to denote the analytic study of individual development (ontogeny) with its central problem of differentiation." Thus, according to Haig (2004), this tradition of epigenetics studies was concerned with the problem of how a constant genotype gives rise to differentiated cell types and tissues, and how perturbations of those processes could lead to diseases such as cancer. A more contemporary definition of epigenetics according to this view, presented by Herring (1993, p. 472) is "the entire series of interactions among cells and cell products which lead to morphogenesis and differentiation."

Transformed into school biology, this tradition of epigenetics provides a framework for describing cell differentiation. However, it does not include any mechanistic explanations at the molecular level of how the genes interact with the environment, so it is difficult to integrate into a course in molecular genetics.

2.3.3 Epigenetics at the Molecular Level

The second tradition of how to view and understand epigenetics can, according to Haig (2004), be traced back to a seminal paper by Nanney (1958) entitled "Epigenetic control systems." Like Waddington, Nanney was interested in development, but rather from a molecular mechanistic perspective. Nanney addressed the same problems as Waddington, but focused on the intracellular level, whereas Waddington considered multicellular phenomena (Nicoglou & Merlin, 2017). Nanney's term 'epigenetic control systems' referred to auxiliary molecular mechanisms outside the central dogma of molecular biology. These systems, he argued, have different operational principles from genetic control systems, regulate gene expression, and are

governed by other factors, such as biological markers and the environment. Nevertheless, the term 'epigenetic' was chosen "to emphasize the reliance of these systems on the genetic systems and to underscore their significance in the developmental processes" (Nanney, 1958, p. 712). Hence, cells with the same genotype can have different genotypes because of variations in the activities of epigenetic control systems that regulate gene expression.

Another important contribution of Nanney's theory was the claim that epigenetic control systems are *mitotically stable*, leading to the maintenance of differences in gene expression among cells during cell division, and thus allowing lineages of cells to develop different tissues and organs within a developing organism (Nicoglou & Merlin, 2017). Nanney (1958) denoted this phenomenon 'cellular memory', and it is now an important part of epigenetic theory. As epigenetics emerged as a distinct field of research in the 1990s, Russo et al. (1996, p. 1) provided a more precise definition of epigenetics as: "the study of mitotically and/or meiotically heritable changes in gene function that cannot be explained by changes in DNA sequence." Today, this definition is very common and is widely used in contemporary epigenetics studies.

According to Nicoglou and Merlin (2017), molecular and cellular definitions of epigenetics commonly highlight three specific defining features. One is the cellular stability or 'memory' of epigenetic changes (often expressed by the term 'heritable', although most often referring to cell lineages within an individual). The others are the impact of epigenetic changes on gene expression/gene function, and their occurrence with no modifications of the DNA sequence.

In recent decades, many regulatory gene–gene and gene–protein mechanisms have been discovered (Deans & Maggert, 2015). These can be categorized into four main categories that are often defined as epigenetic mechanisms. First, the previously mentioned **DNA methylation**, in which methyl groups bind to the DNA molecule and may change the activity of a DNA segment without changing its sequence. Second, **histone modifications**, where post-translational modifications of histone proteins, including methylation, phosphorylation, and acetylation, can impact gene expression by altering chromatin structure or recruiting histone modifiers. Third, **chromatin remodeling**, which is a dynamic modification of chromatin architecture that changes the genomic DNA sequences that can be accessed by regulatory transcription machinery proteins, and thus controls gene expression. Fourth, species of **regulatory RNA**, of various lengths, are paired with complementary sequences of DNA or RNA or enzymes, thereby regulating their expression. These include small microRNAs (species of noncoding RNA that regulate gene expression in either the cell cytoplasm or the cell nuclei). These four mechanisms are related, and often interact, and all of them explain changes in gene expression, but they also clearly differ in location and regulation (Deans & Maggert, 2015). Moreover, the degree to which they meet the three criteria stated above varies. It is especially difficult to identify to what degree some of the mechanisms meet the first criterion of cellular stability or 'heritability'. Regulatory RNA in particular often has effects in the cytoplasm, and the strength of the retention, if any, of those effects ('memories') over cell divisions is difficult to establish. Therefore, some recent definitions of

epigenetics omit this criterion, though I would not when teaching epigenetics in school as discussed in the following section.

This second molecular tradition of epigenetics provides possibilities of linking nature and nurture at a molecular mechanistic level in genetics education. At the same time, the level of complexity and number of concepts are great in this tradition, which creates a high level of abstraction of the content knowledge.

To summarize, epigenetics profoundly challenges the way genetics has been conceptually portrayed in school biology through the central dogma, i.e., that information flows unidirectionally from genes to proteins and traits. It challenges genetic deterministic beliefs by providing integrated mechanistic explanations for the interactive controls of gene expression by nature and nurture. However, I also show that epigenetics is a polysemous concept with multiple definitions and uses stemming from two main disciplinary traditions. In the next section, I address ways of how to transform epigenetics into school curricula and of integrating it into genetics teaching.

2.4 Implications for Teaching

2.4.1 Teaching Genetics Using an Epigenetic Model

As discussed in the previous section, contemporary epigenetics has mostly evolved toward the molecular genetics tradition. Therefore, in attempts to transform disciplinary knowledge of epigenetics into school biology content, I would build on that tradition and include epigenetics mainly in molecular genetics elements of secondary biology courses that cover concepts of gene function and expression. This would reduce the risk of students perceiving that traits are solely controlled by genes, a deterministic conception that is reportedly the most common among students (Lewis & Kattmann, 2004; Venville & Treagust, 1998). Instead, traits can be described as aggregated quantitative effects of multiple genes, which can vary over time and in different contexts, depending on effects of multiple environmental factors, thereby providing a much more flexible conception of gene function. Aspects of the Waddingtonian perspective, such as phenotypic plasticity, could also be included and discussed in other parts of the biology curriculum, but I would recommend the introduction, definition and description of the underlying molecular mechanisms in the realm of genetics. In that context, it would be important to convey the three main features of molecular epigenetics as identified by Nicoglou and Merlin (2017): the cellular memory of epigenetic changes; the impact of epigenetic changes on gene expression/gene function; and their occurrence with no modifications of the DNA sequence. In teaching these features, it would be important to relate micro- and macro-level phenomena and explanations to each other, which is regarded as crucial for promoting a genuine understanding of genetics among students (Duncan & Reiser, 2007; Knippels, 2002).

At the macro level, the teaching could start with comparisons of identical twins who generally have greater phenotypic differences in old age than when they are young, despite their DNA being the same. The differences that develop between them over their lifespan (in the absence of major injuries or changes in weight, etc.) can be explained by the differential accumulation of epigenetic markers at the molecular level, i.e., cell memories, leading to changes between them in gene expression and hence traits. In that way, the three main features of molecular epigenetics could be integrated.

It would be important to teach how environmental and lifestyle factors (such as food, exercise, drugs, and social interaction) induce signals in the body which trigger micro-level epigenetic mechanisms that create cell memories. Therefore, I would restrict the teaching to molecular mechanisms that occur in the nucleus and are more closely related to DNA and cell memory. Teaching about the mechanisms involved could therefore focus on DNA methylation, and possibly histone modification. Another advantage of focusing the teaching on the epigenetic mechanism of DNA methylation is that it can easily be linked to the existing teaching of protein synthesis, and thereby modify the genetic deterministic notion portrayed via the central dogma, as previously explained. DNA methylation is also one of the most thoroughly explored epigenetic mechanisms, facilitating the inclusion of examples and cases in teaching from real studies of epigenetic gene–environment interaction.

In teaching epigenetics, it would then be important to leave the mechanistic molecular level, of DNA methylation, and show the implications at a conceptual level of genetics and epigenetics to counteract genetic deterministic conceptions. Therefore, I suggest that the integrated model of epigenetics, as presented in Fig. 2.1d, be used in conjunction with molecular mechanisms to illustrate the impact of environmental signals on our characteristics, i.e., show that environmental and genetic factors interactively influence traits. Similarly, the molecular mechanisms and their conceptual implications should be used in phenomenological level explanations of the increasing differences as identical twins age. In addition, there may of course be many other fruitful ways to implement epigenetics in secondary genetics teaching and this is just one example, but the benefits of developing genetics education in this manner can be summarized in five main contributions.

2.4.2 Main Contributions by Integrating Epigenetics in Genetics Education

A first main contribution of epigenetics is that it provides an integrated model of gene–environment interaction, as visualized in the epigenetic model in Fig. 2.1d. In educational contexts, an additive model is often applied when teaching nurture aspects of gene–environment interactions, focusing on responses of biological organisms or cells triggered by changes in their surrounding milieu (Fig. 2.1b). In contrast, by teaching gene expression, nature is highlighted as the predominant

biological force in a deterministic model (Fig. 2.1a). Epigenetics provides another perspective in biology education, where genes and environmental factors interact directly in an integrated explanatory model through phenotypic plasticity.

The second main benefit of epigenetics, in comparison to the more commonly used concept of gene–environment interactions, is the addition of cell memory or notion of mitotic heritability. Deans and Maggert (2015) suggest that we should use the term 'heritable memory' to promote the understanding that environmental experiences of an organism (physical, biotic and/or social) may leave persistent epigenetic marks that are materialized in the molecules; this is missing in more classical gene–environment regulatory models.

The third benefit is that epigenetic heritable memory acts on much longer timescales than physiological reactions, which in cases such as some photosynthetic responses to changes in light level may be in the picosecond range (Holzwarth, 1986). The effects of many such responses are transient, and when the stimulus is gone, the effect rapidly diminishes, leaving no traces in the affected organism or cell. At the opposite end of the timescale, there are mutations, i.e., random changes in the DNA sequences of genes, and chromosomal rearrangements. These changes are by definition heritable, and can be favored by selective pressure in evolutionary processes that often span many generations and thousands of years, although exceptions such as chromosomal rearrangement may result in fast evolutionary processes (Pérez-Ortín et al., 2002). Moreover, these changes are fixed within an individual. Thus, epigenetics provides an explanatory model with a timescale between those of classical gene–environment regulation and evolutionary explanations.

A fourth contribution is that epigenetic processes are dynamic: epigenetic patterns can be reversed, so epigenetic effects are not deterministic and epigenetic processes are not definitive. This could offer hope for students and empower them regarding epigenetics' societal implications, such as that tailored medical treatments and lifestyle choices could have potent epigenetic effects. Here, though, lies one of the most conceptually demanding aspects of epigenetics. The effects of different epigenetic mechanisms are context-dependent and their stability varies considerably. Some epigenetic phenomena, such as genetic imprinting, are not reversible, while other epigenetic effects persist for parts of minutes to hours, days, years, entire lifespans and possibly generations. This dynamic semi-stable attribute confers the key advantage of teaching epigenetics, in its potential power to loosen students' prevalent deterministic understanding of gene function, but it is highly conceptually demanding. If we integrated the epigenetics perspective in the genetics syllabus of a revised biology curriculum, I would expect this aspect to raise substantial teaching challenges.

A fifth, and very specific contribution is that epigenetics has the potential to be a useful topic for teaching cell differentiation. Several studies have shown that students have difficulties understanding that different cell types within an individual contain the same genetic information, but use different parts of it. Instead, secondary-school students tend to think that differences in cell types are due to the cells containing different genetic information (Lewis & Kattmann, 2004; Lewis et al., 2000). Explaining cell differentiation by epigenetic regulation through DNA methylation

could be a powerful tool for countering these misconceptions by showing that genes can be switched on and off by these mechanisms.

As already noted, the idea of phenotypic plasticity can also be used to promote an epigenetic model integrating nature and nurture. Kitcher (2001) argues that highlighting genotype–environment interactions is important in neutralizing genetic deterministic conceptions. He discusses the possibility of drawing on the concept of 'reaction norm' that describes the pattern of phenotypic expression of a single genotype across a range of environments. For every genotype, phenotype, and environmental variable, a different reaction norm can be portrayed in a diagram, as illustrated in Fig. 2.3. Epigenetic regulation relies on the activation (switching on) and deactivation (switching off) of specific sets of genes, thereby influencing levels of corresponding RNA species (encoding proteins or RNA species involved in transcription). These processes govern traits' levels of expression, as shown on the Y-axis of diagrams of reaction norms, and are influenced by the level of exposure to environmental variables, as shown on the X-axis. Figure 2.3a shows an example of the relationship between a genotype and corresponding phenotype of an organism according to a genetic deterministic representation. The influence of the environment is zero, regardless of the strength and duration of the exposure, so the environmental factor will not induce any change in the trait. In Fig. 2.3b, there is great environmentally linked variability in gene expression, due to epigenetic mechanisms. Figure 2.3c shows contrasting interactions of two genotypes (organisms carrying different alleles) with the environment. One genotype (X) is more sensitive than the other (Y), leading to different activation rates of epigenetic mechanisms. Figure 2.3d shows healthy and unhealthy ranges of a trait's expression, demonstrating how epigenetic mechanisms might be triggered by environmental factors that

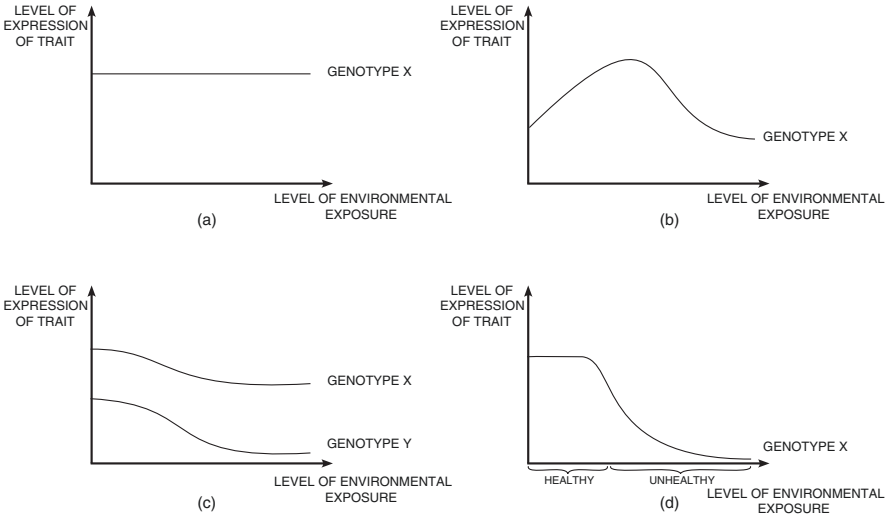

Fig. 2.3 The nature–nurture relationship portrayed through visualizations of a reaction norm according to conceptual frameworks presented in the text. (Modified by Gericke from Kitcher, 2001)

cause diseases such as cancer at exposure levels beyond a certain threshold. Hence, using visual representations such as these, it is possible to link expressions at the phenomenological or macro level with molecular epigenetic processes, such as DNA methylation, as already described in this text. Moreover, by including the symbolic representations of Fig. 2.1 it can be shown that the portrayed molecular mechanisms and phenomena have profound conceptual implications for the integration of nature and nurture.

To conclude, epigenetics provides genetics education with an explanatory model that integrates nature and nurture in a manner that has the potential to promote an understanding of gene function that counters genetic deterministic conceptions. Further, as outlined in this chapter, epigenetics could be used to unpack the black box of gene function in genetics education in a way that connects mechanistic molecular explanations with conceptual explanations at the phenomenological level. This chapter is a call for curriculum development as well as future studies that could unravel ways in which epigenetics can be enacted in school biology teaching.

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Chapter 3
How Can We Teach Genetics for Social Justice?

Michael J. Reiss

3.1 The Problem

Learning about genetics can result in misconceptions; one of these is that when genetics plays a part in the development of a characteristic, that characteristic is genetically determined (e.g., Kampourakis, 2017). When such misconceptions are about matters of deep personal significance to us, such as human sexuality and intelligence, there is a danger that teaching about such issues could lead to individuals being disadvantaged and social justice retarded. Under these circumstances, one response might be to avoid such teaching in formal education at schools. However, might this amount to abdicating our responsibility as genetics educators? In addition, students are likely to have such misconceptions reinforced through what they learn from other sources, including the media (Carver et al., 2017). Could it be that good-quality genetics education in schools will not only help students gain a better understanding of genetics, but will also help advance social justice? In this chapter, I explore this idea, with particular reference to teaching about such educationally significant factors as general intelligence, reading ability and examination success.

It is well established that many people, including school students (Gericke & El-Hani, 2018) and the general public (Gadjev, 2020; Kampourakis, 2020), find the topic of genetics cognitively difficult (e.g., Kampourakis, 2017; Haskel-Ittah et al., 2020). There are many reasons for this. For a start, some of what we (as science educators) want learners to understand takes place on scales that are too small for visualisation, even with electron microscopes—the sequences of bases on DNA, in particular (cf. Marbach-Ad & Stavy, 2000; Rotbain et al., 2006). Then there is the fact that a deep understanding requires knowledge at a number of different levels—a change in DNA structure may lead to a change in protein structure, which may affect the phenotype of an organism, which may result in it leaving fewer or more

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copies in future generations, which may have consequences for the population as a whole and ultimately, the species. These levels operate over huge ranges of time and space. In addition, and related to this, some of genetics is abstract and makes high cognitive, including mathematical, demands on learners, resulting in numerous student misunderstandings (misconceptions) about genetics in particular and evolution more generally (Harms & Reiss, 2019).

In this chapter, I address an issue that has to do with difficult genetics and lies at the heart of scientific literacy, namely, the relevance of much of genetics education to the lives of learners. By relevance, I mean more than interest; I mean the extent to which contemporary school genetics education does a good job of enabling students to understand the ways in which genetics affects their lives and the lives of others—now, in the past and in the future. My particular focus, for reasons that I explain below, is the specific issue of the genetics of (general) intelligence (and related characteristics, such as reading ability); the broader context deals with how genetics can be taught for (i.e., to advance or promote) social justice. Back in 2000, I wrote an article asking whether it would be wise to undertake research on the genetics of intelligence. I concluded: "The history of the debate on intelligence does not make one very optimistic that the fruits of such research would be used wisely" (Reiss, 2000, p. 1).

I do not want my comments to relate only to my own country, so there is no analysis here of the National Curriculum for science as it applies to England, Wales and Northern Ireland, nor of school textbooks and examinations in these countries. Instead, I start with the 'Big Ideas' of science education—as articulated by Wynne Harlen and her colleagues (Harlen et al., 2010, 2015). At the time of writing, the documents about 'Big Ideas' are not as highly cited as the Next Generation Science Standards (National Research Council, 2012). However, the latter are intended specifically for the United States, whereas Harlen's 'Big Ideas' are intended internationally and have been taken up in a number of countries; they are currently perhaps the nearest we have to an international agreement on what should be in school science education (Appendix 1).

When one looks at Appendix 1—with its suggestions for Big Idea 9 "Genetic information is passed down from one generation of organisms to another"—a number of things strike me.[1] For a start, I am rather surprised now to read "other features, such as skills and behaviour, are not passed on in the same way and have to be learned." This is, at best, a major oversimplification—though I realise that one does need to simplify for 7–11 year olds, the intended age for this learning objective. But I note two bigger things. One is that, perhaps inevitably, what is written in Appendix 1 is written at a high level of generality—it is not clear, for instance, which characteristics of organisms are being talked about. The second is that there is nothing in Harlen et al. (2010) or Harlen et al. (2015) about the history of the use and misuse of genetics, or on the relative contributions of genes and the environment to the

[1] This is not meant to be read as an attack on this Big Idea. Indeed, if I am critical, I am in large measure self-critical as I was part of the team that wrote the Big Ideas and, as a biologist, I share particular responsibility for what is in the biological Big Ideas.

determination of phenotypes—aside from the cited sentence for 7–11 year olds. It would be possible to read all of the text in Appendix 1 and conclude that characteristics are either entirely determined by genes or completely independent of them—a false conclusion that is likely to be reinforced by the introductory statement "Genes determine the development and structure of organisms."

The nearest the Big Ideas come to acknowledging the importance of the use and misuse of genetics is in Big Idea 14: "Applications of science often have ethical, social, economic and political implications." Here we read, for example (for 11–14 year olds), "There are generally both positive and negative consequences of the applications of science. Some negative impacts can be anticipated but others emerge from experience." However, the examples that immediately follow have nothing to do with genetics. Indeed, there is nothing in Big Idea 14 about genetics for any student age group.

3.2 Current Knowledge About the Problem

It is widely accepted that the material, cultural and social benefits that children receive from their parents play an important role in how well they do at school. However, there is a disconnect between what most academics in education and what many academics in biology think about the role of genetic inheritance in many areas of human life, including how well children do in schools (Reiss, 2018). Here, I first look at why there is this disconnect and then examine the core issue of the role of genetic inheritance in school performance. As a result, I hope to show three things:

1. Genetic inheritance can contribute to how well children do in schools.
2. This does not mean that children's school performance is predetermined, i.e., fixed in advance; environments are important too.
3. Education needs to stop putting its head in the sand about the possible role of genetic inheritance in school performance.

3.2.1 Inheritance Plays a Role in How Well Children Do in Schools

Geneticists determine the extent to which inheritance plays a role in the manifestation of a trait in much the same way, whether we are considering the height of plants, the milk yield of cows or the reading ability of children. Saying that inheritance 'plays a role' is not to minimise the importance of environmental factors or to ignore the ways in which environmental and genetic factors may interact. Throughout, of course, by 'inheritance' is meant 'genetic inheritance'. Everyone realises, for example, that family background is important. If one is brought up in a home with lots of books and where reading is valued, it is hardly surprising that one

is likely to do better at reading as a child than another child of the same age who has not enjoyed such benefits. Indeed, much of the skill in arriving at measures of 'heritability'—the extent to which genetics plays a role—is precisely to do with disentangling the effects of shared environments.

Without going into a full-scale statistical treatment of how biologists and statisticians determine the importance of genes for the expression of any trait (e.g., Walsh & Lynch, 2018), what is needed is:

- to obtain reasonably objective measures of the trait in question. This is fairly easy for milk yields in cows; it is harder—but not impossible—for most things of educational interest, such as reading ability or musicality;
- to collect such data from a large number (ideally many thousands) of individuals;
- to get a measure of the extent to which these individuals have similar genetic constitutions;
- to get a measure of the extent to which these individuals have similar environmental backgrounds.

It is the last two of these that are the most difficult to achieve and for this reason, a number of human studies have relied on twin studies. Twin studies are of value because there are two sorts of twins—identical and non-identical. Non-identical twins are no more genetically similar than any two non-twin siblings but, by virtue of having been born from the same pregnancy, they have shared an early environment that is more similar than that shared by non-twin siblings. Identical twins have an early environment that is at least as similar as that shared by non-identical twins; but, in addition, they are virtually identical genetically. What this means is that by looking at the extent to which monozygotic (identical) twins are more similar in certain traits than are dizygotic (non-identical) twins, one can obtain a measure of the heritability of a trait. In a comprehensive review of the causes of individual differences in human traits, Polderman et al. (2015) concluded that across all such traits, the reported heritability was 49%. For 69% of the traits, the observed twin correlations were consistent with a simple model in which twin resemblance is solely due to additive genetic variation: the data were inconsistent, with substantial influences from shared environment or non-additive genetic variation.

To give an extreme example (and one that oversimplifies as heritabilities are normally calculated on characteristics that vary continuously not discretely): identical twins typically have very similar eye and hair colour—more similar than is the case for non-identical twins. We therefore conclude that eye and hair colour have high heritabilities. However, the language (e.g., French, Urdu, Mandarin) spoken best by identical twins is no more similar than in the case for non-identical twins. In most cases, of course, siblings, twins or not, have the same mother tongue but if they are separated at some point in their childhood—for example, because they are adopted by families in different countries—they may end up speaking different languages best. We therefore conclude that the language one speaks best has a very low heritability.

Nowadays, there are various ways of calculating heritabilities and they give similar values—which is encouraging from a scientific point of view. A widespread

consensus is that human behaviours tend to have heritabilities of about 0.3–0.6 (Bouchard, 2004). Heritabilities lie between 0 (e.g., the language one speaks best) and 1 (e.g., eye colour). This means that human behaviours are moderately heritable—not as heritable as height (with a heritability in the West of about 0.9), but more so than religiosity (which has a heritability of about 0.1–0.2). Examples of human behaviours include personality, intelligence, artistic interests and the chances of developing a psychiatric illness.

However, the term 'heritability' is often misunderstood. To calculate it, as the bullet points above indicate, it is necessary to look at quite a large number of individuals, and the calculated values therefore apply to the level of groups of individuals, and not to the individual level (Moore & Shenk, 2017). Indeed, it simply does not make any logical or biological sense to attempt, for any individual, to apportion its characteristics between its genes and its environment. As I once wrote:

> I was fortunate enough when an undergraduate in the late 1970s to be taught animal behaviour by Pat Bateson, among others. Pat sometimes likened the role of genes to the role of a recipe in making a cake. Genes and recipes are essential but it makes little sense to ask what proportion of a good (or a bad) cake is due to the recipe. (Reiss, 2003a, p. 51)

Moore and Shenk (2017) helpfully spell out a thought experiment from Lewontin (1974) in which plants are grown from seed under one of two sets of environmental conditions—one with high levels of nutrients and one with poor nutrients. The important point is that within each experimental set up, there is negligible environmental variation, so that any differences in, for example, plant height must be due to genetic differences between the plants, resulting in calculated heritabilities of (close to) 100% (1.0). However, there may be major differences between the results of the two experimental set ups, with, for instance, plants typically being substantially lower in height when grown in poor nutrients. So, it is a mistake to conclude that just because heritability is high, environments cannot make a difference.

Turning specifically to issues connected with school performance, a thorough summary of the argument that human genetics plays an important role is provided by Asbury and Plomin's (2014) *G Is for Genes: The Impact of Genetics on Education and Achievement* and Plomin's (2018) *Blueprint: How DNA Makes Us Who We Are*. Robert Plomin set up the Twins Early Development Study (TEDS) in 1994 when he moved to the UK from the United States. TEDS is now one of the largest and longest-running twin studies in the world, with about 13,000 pairs of twins in 2019.

As is well-known, twin studies have historically been of great value in inheritance research as they do not require the sort of DNA mapping that has only fairly recently become widely available (and affordable). Estimates of heritability can be made using data from monozygotic twins reared apart (but there are only a few hundred such pairs of twins who have been studied) or by using data from monozygotic twins brought up together and from dizygotic twins brought up together.

Today, other approaches, in addition to twin studies, are becoming increasingly valuable for determining human heritabilities. In particular, the rapid decrease in the cost of DNA sequencing means that it has become possible to screen large numbers of people (genome-wide association studies) to see if they have particular gene

sequences that are of interest with regards to particular characteristics. Because they involve large numbers of people (typically in the tens of thousands), genome-wide association studies are good at identifying genes and combinations of genes that have only small effects on the characteristic(s) in question.

One conclusion from these various studies seems clear: it is no longer possibly to validly conclude that genetics plays no part in educational success (e.g., Morris et al., 2018; Savage et al., 2018; Sniekers et al., 2017). For example, in the UK, there is a genetic component to university examination success (Smith-Woolley et al., 2018). Furthermore, it is not just 'intelligence' that is heritable. For instance, genetic factors are implicated in mathematical anxiety (Wang et al., 2014).

However, it may be that the standard ways of calculating heritabilities underestimate the importance played by the environment and therefore overestimate the importance of genetics (Rosenberg et al., 2018). Twin studies often produce higher estimates of heritabilities than do genome-wide association studies, which suggests that, despite the best efforts of those undertaking twin studies research, it remains difficult to untangle the effects of genes and the environment. We are in the early days of research on the genetics of intelligence and it is very possible that some of today's confident assertions will be tempered by time.

3.2.2 Children's School Performance Is Not Predetermined

As every biology educator knows, calculating heritabilities and stating that differences between genes are involved in school success does not mean that genes alone are important—an organism's genes do not *determine* its characteristics. For a start, there is the obvious truth that genes need the rest of the cell to work. Then there is the fact that we could just as well talk about the roles that proteins (and other gene products) play in school success. There are two biologically valid reasons for why we more usually talk about genes: it is genes that are inherited; and the Central Dogma (DNA makes RNA makes proteins), so that changes to RNA or protein structure that are not the result of changes to DNA structure are not passed on to the next generation.

Even those who emphasise the importance of genetics in the development of human characteristics fully acknowledge that sometimes, genetics plays less of a role than is commonly presumed. Plomin himself points out that whereas people typically presume that breast cancer is strongly influenced by genetics, in fact, it has a heritability of only about 10% (Plomin, 2018).

Then, focusing on intelligence, there is the well-known Flynn effect. Throughout the twentieth century, there were steady and substantial increases in IQ (intelligence quotient) scores over time in just about every country where such data were collected. Each decade, average IQ scores increased by about 2.5–3 points. That is not much year to year, but over the twentieth century, it amounts to 25–30 points, almost two standard deviations. A number of factors are believed to contribute—better health, better education, better nutrition among them—but the important point is

that such data emphasise the extent to which intelligence has an important environmental component (cf. Bratsberg & Rogeberg, 2018). Flynn's more recent work explicitly attacks the notion that genetics is of overriding importance in the determination of intelligence (Flynn, 2016).

It is hardly surprising that education enhances intelligence. But it might be that students with a greater propensity for intelligence go on to complete more education, or that more years of education increase intelligence (Ritchie & Tucker-Drob, 2018). In a large (over 600,000 participants) meta-analysis, Ritchie and Tucker-Drob (2018, p. 1358):

> found consistent evidence for beneficial effects of education on cognitive abilities of approximately 1–5 IQ points for an additional year of education. Moderator analyses indicated that the effects persisted across the life span and were present on all broad categories of cognitive ability studied. Education appears to be the most consistent, robust, and durable method yet to be identified for raising intelligence.

Some of the most trenchant criticism of the argument that genes are important determinants of educational success has been raised by the veteran biologist, Steven Rose. One of Rose's key points is that calculations of heritability depend on the extent to which the environment varies in some relevant way—this is well-known but easy to forget (Rose, 2014). A classic example is that human height shows higher heritability in high-income countries than in low-income ones where nutrition and disease play a greater role (Perkins et al., 2016). In the same way, Turkheimer et al. (2003) concluded that "in impoverished families, 60% of the variance in IQ is accounted for by the shared environment, and the contribution of genes is close to zero; in affluent families, the result is almost exactly the reverse" (p. 623). Another point Rose makes is that gene–environment interactions (possibly of particular significance in human characteristics such as learning) make it even more difficult (less meaningful) to partition out effects between genes and the environment (Rose, 2014; cf. Tucker-Drob & Bates, 2016).

3.2.3 Education Needs to Stop Ignoring the Possible Role of Genetics in School Performance

Ever since the publication of Darwin's momentous *On the Origin of Species* in 1859, biologists have accepted that inherited variation plays a central role in the manifestations and evolution of the enormous number of characteristics exhibited by organisms. The early twentieth century advances in genetics, followed by the mid-twentieth century advances of neo-Darwinism and the subsequent developments in molecular biology, have emphasized this conclusion (Klug et al., 2019; Roberts et al., 2000).

In the case of humans, along with other organisms, this means that just about everything of interest about us has an inherited component. It does not matter whether one considers height or weight or reaction time or longevity or the

likelihood of developing heart disease or anything else, inheritance generally plays a role. And this is true, too, of such educationally significant factors as general intelligence, reading ability and examination success. This, of course, is not to ignore the influence of environmental factors on all of these characteristics.

Many people—including parents and teachers—are happy to accept that children differ greatly in their abilities or potential (e.g., at music, mathematics or sports). However, with certain exceptions (e.g., Ingram, 2019), educators have generally been reluctant, to put it mildly, to accept the mounting weight of evidence for the importance of genetic inheritance in school performance (e.g., White, 2006). There are a number of reasons for this reluctance—most of them understandable and indeed well-intentioned.

For one thing, there is a terrible legacy of genetics and human history. Historians of science and evolutionary biologists (e.g., Gould, 1981; Lewontin, 1991) have shown how genetics has been used, both consciously and unconsciously, in attempts to argue for the inferiority of women, of black people and of those not in the ruling classes. Faced with this legacy of sexism, racism and cultural imperialism, it is hardly surprising that educators have rejected genetics as a way of understanding differences between humans. What has happened is that genetics, rather than the misuse of genetics, has been rejected. It is as if books in general were rejected because some books are harmful. The reality, though, is that a *better* understanding of genetics, not the *abandonment* of genetics, is what is needed.

A second major reason for the widespread scepticism among educators, certainly in the UK, concerning the importance of inheritance in educational attainment is due to the legacy of Cyril Burt. Cyril Burt (1883–1971) was an educational psychologist who played an important role in the development of an examination (the '11-plus') in schools in England to determine whether students were educated from the age of 11 in more (grammar schools) or less (secondary modern) academically demanding schools. Although there have been quite a number of revisionist accounts (e.g., Fletcher, 1991; Tredoux, 2015), it is generally thought that Burt systematically engaged in scientific fraud, falsely claiming to have collected data in his studies on the heritability of intelligence (Tucker, 1997). However, the findings that he produced on the extent to which intelligence is inherited were in line with other studies at the time (Rushton, 1997). In other words, even if we ignore all of Burt's work, there would be no effect on the conclusions to be reached from the literature about the role of inheritance in the manifestation of intelligence, namely that inheritance and the environment both play a part (Johnson, 2010).

A third major reason why educators have tended to ignore the ever-increasing growth in what is known about the inheritance of intelligence is, I believe, due to the widespread, often implicit, presumption that *inheritance* is to be equated with *determinism* (e.g., Gericke et al., 2017; Jiménez-Aleixandre, 2014; Kampourakis, 2017), as discussed above.

3.2.4 Social Justice in Science Education

Traditionally, there have been two main aims for school science education. The majority aim has simply been for students to come to a good knowledge and understanding of science, typically understood as both the content of science (the specifics of biology, chemistry, earth science and physics) and the way in which science is undertaken (often referred to as the nature of science). The second aim has been that school science education should in some way contribute to the well-being of both the individuals who are learning it—now and/or in the future—and more collectively, society (Reiss & White, 2014).

This second aim can be characterised in a number of ways but one that has a good pedigree is 'science for social justice' (Reiss, 2003b). Social justice is about the right treatment of others [what Gewirtz (1998) identifies as the relational dimension of social justice] and the fair distribution of resources or opportunities (the distributional dimension). Of course, considerable disagreement exists about what precisely counts as right treatment and fair distribution of resources. For example, some people accept that an unequal distribution of certain resources may be fair provided certain other criteria are satisfied (e.g., the resources are purchased with money earned, inherited or obtained in some other socially sanctioned way—such as gambling in some, but not all, cultures). At the other extreme, it can be argued that we should ensure either that all resources be distributed equally or that all people have what they need. Such distributions might be achieved through legislative coercion, social customs or altruism on the part of those who would otherwise end up with more than average.

An important element of teaching for social justice is what Freire (1970) termed 'conscientization' (or 'consciousness raising'). This can be seen in feminist pedagogy, where students develop the ability to question gendered inequities and their causes and perpetuation, in anti-racist education, in education that seeks to undermine heteronormativity, in critical pedagogy in general and in science education more specifically (Reiss, 1993).

Teaching in school science for social justice should help promote flourishing, for both humans and other organisms, and for the environment more generally. We want, for example, people to want other people, as well as themselves, to live fulfilling lives. Negatively, this means not hurting them, not lying to them, not breaking one's word or in other ways impeding them in this. Positively, it means helping them to reach their goals, respecting their autonomy and being fair, friendly and cooperative in one's dealings with them. Schools can reinforce and extend what parents and others family members do in developing morality in children, and school science has a particular place in this given the fact that many contemporary ethical issues have a techno-scientific element to them (genetic modification, climate change, artificial intelligence, etc.). Schools can expand students' moral sensitivity beyond the domestic circle to those in other communities, locally, nationally and globally, and beyond this to other species and the whole of the environment.

Specifically with reference to teaching about educational success, there is a risk that teaching about the role of genetics in this might backfire, causing students to conclude that their educational ability is 'fixed' and that it is not worth them bothering much if they are doing poorly in school. This, of course, would retard rather than advance human flourishing. One possible response, therefore, is to continue to do what is being done at present, which is to avoid consideration of the issue. But I think that there is a risk to this response; in failing to address students' misconceptions about genetics in general and the genetics of educational success in particular, an opportunity is lost. My hope is that good-quality genetics education might enable students to reject the mistaken conclusion that educational ability is 'fixed'.

3.3 Remaining Issues

While there is, in my judgement, no doubt that there is a genetic component to educational success, several points need to be made. For a start, the contribution of any one gene locus is almost always extremely small. Even large numbers of genes considered together typically account for only a relatively small percentage of the observed variation. For example, a recent large study undertaken on over one million individuals identified 1271 independent genome-wide-significant single nucleotide polymorphisms (SNPs) (Lee et al., 2018). However, collectively, these only accounted for 11–13% of the variance in educational attainment and 7–10% of the variance in cognitive performance.

Then there is the fact that, as yet, understanding *how* certain genes affect cognitive and/or educational performance—i.e., their mechanisms—is only beginning. I have no doubt that these mechanisms will increasingly be worked out and that such elucidation will help reduce some of the over-the-top claims *and* fears around genetic influences; however, much remains to be done.

Perhaps the most important educational issue that remains is whether advances in genetics will prove to be of value in enabling educational interventions. I discuss this possibility in the section below 'Genetics and better diagnoses of educational issues'.

3.4 Implications for Teaching

Understood badly, realisation of the importance of genetics for education can paralyse teachers and students, leading them to think, mistakenly, that there is little that can be done to counteract the effect of genes. In this section, I discuss two main ways in which this belief is mistaken, firstly by discussing the 'growth mindset' movement and secondly, and more speculatively, by suggesting how genetics might one day be used in better diagnoses of educational issues.

3.4.1 The Growth Mindset Movement

'Growth mindset' refers to a learning theory most associated with the work of Carol Dweck. The key idea is that if learners believe that they can improve their performance (intelligence, subject attainment, skills, examination success and the like), they will do better than if they believe that their performance is predetermined (Dweck, 2017). When Dweck was a child in her 6th-grade class in Brooklyn, New York, students were seated in order of their IQ. Students with the highest IQ scores could erase the blackboard, carry the flag or take a note to the principal's office. In a 2015 interview, Dweck pointed to this 'glorification' of IQ as a key point in her childhood.

Dweck (2017) argues that individuals vary with respect to where they believe ability comes from, falling somewhere on a continuum with two endpoints. At one extreme, those with a 'fixed' mindset believe that ability is innate, and can be changed only a little. At the other extreme, those with a 'growth' or 'incremental' mindset believe that success comes from hard work and persistence. Dweck and her colleagues maintain that encouraging a growth mindset in students results not only in them learning more but also in better self-regulation, increased wellbeing and reduced helplessness.

As a teenager, despite doing very well at mathematics and the sciences and reasonably well at English, with a passion for reading, I had convinced myself that I was not good at languages. In hindsight, it was simply that my performance at French and Latin—the two foreign languages I had been taught for many years—was mediocre, which probably says as much about my teachers as myself. I can still recall the first lesson I had at school (aged 13) in German. The teacher burst into the classroom and proceeded to speak only German. At the time this seemed revolutionary to us. "Ich bin Herr Martin. Wer bist du?" he began. By the end of the lesson we were all speaking a few simple phrases and I proudly said to my grandmother (who was German) when I next saw her "Das ist ein Kugelschreiber," as I took a biro from my jacket pocket. German ended up being one of my two best 'O' levels (examinations taken in England at that time at the end of compulsory schooling) while Latin was my worst, with French not much better. Having previously presumed that I suffered from some sort of innate shortcoming at languages, I now realise that this was not the case.

There is mounting evidence that interventions can enable students to move towards more of a growth mindset position, though not all interventions have proved successful (e.g., Foliano et al., 2019). Yeager et al. (2019) found that an online growth mindset intervention that took just under one hour and taught that intellectual abilities can be developed improved grades among lower-achieving students and increased overall enrolment in advanced mathematics courses in students in school education in the United States. The effect size was not large (0.10) but the sample was nationally representative and given that the intervention took under an hour, it represents excellent value for money; in addition, an effect size of 0.10 equates to about 6 months of progress with an average teacher.

Not everyone is convinced by the growth mindset argument. The same Robert Plomin who has done so much work on the genetics of intelligence and educational success is unimpressed with it:

> Growth mindset, I feel, is greatly over-played…If you try to tell kids who have trouble learning, "You can do it, you can change," you can actually do some harm. Because some kids are going to find it really difficult; it isn't just a matter of positive thinking. Kids aren't stupid. I don't believe the evidence base is all that strong. (Lee, 2015, p. 12)

Of course, defenders of the growth mindset argument would respond by saying that Plomin's characterisation of it as "you can change" at best misunderstands what growth mindset is all about (at worst, the phrase itself suggests an essentialist conception of individual performance that is precisely what growth mindset rejects). It is not a matter of students who are performing poorly "changing"—a sort of naïve positive psychology. It is about all students putting into practice the notion that each of us needs to persist and practise, thereby improving our performance. Such teaching requires appropriate resources and caring teachers. It is known that STEM faculty who believe that ability is fixed have larger racial achievement gaps and inspire less student motivation in their classes (Canning et al., 2019).

3.4.2 Genetics and Better Diagnoses of Educational Issues

It needs to be emphasised that, as yet, genetics has contributed virtually nothing of any value to teaching. Indeed, because of the common, albeit mistaken equation of genetics with destiny (the belief that genes are determinative), it is more likely that genetics has harmed education. Nevertheless, it is possible that genetics might eventually prove to have some direct educational value. Consider the analogy with medicine. For a long time, understanding the genetics of diseases was of no use in treating them. Gradually, however, certain diseases with a strong genetic component became treatable or, even better, preventable as a result of such knowledge. We are now in the early stages of gene therapy, but examples exist from long before gene therapy was even a pipe dream.

A classic example is the condition phenylketonuria, a congenital metabolic disorder in which the body is not able to manufacture the enzyme phenylalanine hydroxylase. As a result, the amino acid phenylalanine accumulates to levels in the blood that affect the brains of infants, resulting in severe mental retardation and other adverse consequences if left untreated. In 1962, Robert Guthrie invented the test that now bears his name, replacing a pre-existing but less effective test. The Guthrie test relies on the collection of a few drops of blood from one of the heels of a new-born. Individuals found to have the abnormalities in their blood that indicate that they will go on to develop phenylketonuria unless something is done are put on a diet that is low in phenylalanine. Used in many countries, this has prevented the development of phenylketonuria in tens of thousands of people.

In the same way, it is possible that genetics might one day be used to tailor intervention programmes more precisely so that—to give just one example—instead of a 4- or 5-year-old simply being identified as slow to start reading, it would be known whether to concentrate on helping the child distinguish between certain letters, learn the relationships between letters and sounds, read consistently and steadily from left to right (for left-to-right languages), etc. Another analogy would be with spectacles or hearing aids—find the right one and learning can take off.

3.4.3 Genetics Education

Finally, there are implications for genetics education. There isn't space here to flesh out a whole curriculum but, from the above literature and arguments, teaching about the genetics of intelligence might have a number of benefits:

- It provides an example of 'complicated' inheritance—so is better and possibly more interesting for students than the simplified stories they often get.
- It represents cutting-edge science.
- It provides a good example of evo-devo, including the role of learning (e.g., 'feral' children, children in certain orphanages).
- It has lessons for things like sporting success and musical aptitude.

There are a number of things we might want students to learn about the genetics of intelligence:

- intelligence is not a simple monogenic trait (cf. standard accounts of blue eye colour, phenylketonuria, cystic fibrosis, etc.) but a complex trait that is influenced by interactions between polygenic and environmental factors, including the family and the society in which one grows up;
- the distinction between heritability and determinism;
- growth mindset arguments and our ability to improve, given appropriate resources, support from others and effort on our part;
- whether there are likely to be any practical implications of research into the genetics of intelligence, reading ability or musicality;
- there have been and continue to be many instances of the misuse of genetics to the disadvantage of women, minority groups and those in general who are not in positions of power and privilege.

Teaching about the genetics of intelligence can therefore allow for explorations of socio-scientific issues and the role of ethics in science. It also potentially provides a good entry into consideration of the nature of science and the history of science—including disagreements among scientists. Nevertheless, it is important to emphasise that many people have a deterministic understanding of the genetics of human behaviour in which genes are presumed simply to 'cause' characteristics (e.g., Lynch et al., 2018). If done badly, teaching about the genetics of human characteristics might not only fail to overturn such misunderstandings; it might reinforce them.

In a classic study, Dar-Nimrod and Heine (2006) showed that women who read a passage about genetic causes of sex differences subsequently performed worse on math tests than those who read about experiential causes. Teaching matters.

3.4.4 Conclusion

There are many ways that good teaching, including good science teaching, might hope to advance social justice. We now have the beginnings of a literature compendium as to how genetics education can be of high quality and help to advance genetic literacy (Boerwinkel et al., 2017; Dougherty, 2009; Nowgen Centre, 2012) and, in particular, social justice, for example by tackling issues to do with determinism (Clément & Castéra, 2014), race (Sheth, 2019) and sex differences (Donovan et al., 2019). The argument of this chapter is that done well, good biology teaching about intelligence can help all learners learn well and flourish. However, done badly, genetics education can have the opposite effect.

Appendix 1

Genetic Information Is Passed Down from One Generation of Organisms to Another

Genetic information in a cell is held in the chemical DNA. Genes determine the development and structure of organisms. In asexual reproduction all the genes in the offspring come from one parent. In sexual reproduction half of the genes come from each parent.

5–7 years old

Living things produce offspring of the same kind, but offspring are not identical with each other or with their parents. Plants and animals, including humans, resemble their parents in many features because information is passed from one generation to the next.

7–11 years old

Other features, such as skills and behaviour, are not passed on in the same way and have to be learned.

11–14 years old

Inside the nucleus of animal and plant cells are structures called chromosomes which hold large complex molecules of DNA. When cells divide the information that is needed to make more cells is in the form of a code represented in the way that the parts of the DNA molecule are put together. A gene is a length of DNA; and hundreds

or thousands of genes are carried on a single chromosome. In the human body most cells contain 23 pairs of chromosomes with a total of about twenty thousand genes.

When a cell divides, as in the process of growth or replacement of dead cells, genetic information is copied so that each new cell carries a replica of the parent cell. Sometimes an error occurs in replication, causing a mutation, which may or may not be damaging to the organism. Changes in genes can be caused by environmental conditions, such as radiation and chemicals. These changes can affect the individual but only affect the offspring if they occur in sperm or egg cells.

In sexual reproduction, a sperm cell from a male unites with an egg cell from a female. Sperm and egg cells are specialised cells each of which has one of the two versions of each gene carried by the parent, selected at random. When a sperm and egg combine half the genetic material in the fertilised egg is from the sperm cell and half from the egg cell. As the fertilised egg divides time and time again this genetic material is duplicated in each new cell. The sorting and recombining of genetic material when egg and sperm cells are formed and then fuse results in an immense variety of possible combinations of genes, and in differences that can be inherited from one generation to another. These provide the potential for natural selection as a result of some variations making organisms better adapted to certain environmental conditions.

14–17 years old

Asexual reproduction, which occurs naturally in a wide range of organisms including some bacteria, insects and plants, leads to populations with identical genetic material. Biotechnology has made possible the production of genetically identical organisms through artificial cloning in a range of species including mammals.

The overall sequence of genes of an organism is known as its genome. More is being learned all the time about genetic information by mapping the genomes of different kinds of organisms. When sequences of genes are known genetic material can be artificially changed to give organisms certain features. In gene therapy special techniques are used to deliver into human cells genes that are beginning to help in curing disease.

Taken from Harlen et al. (2015, p. 28)

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Part II
Reflecting Upon Processes for Constructing Students' Understanding in Genetics

Chapter 4
How Can Learning Progressions Support the Development of Genetic Literacy?

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4.1 The Problem

Genetics is a complex, conceptually challenging domain to teach and learn (e.g. Freidenreich et al., 2011; Gericke & Wahlberg, 2013; Haskel-Ittah & Yarden, 2018; Lewis & Kattman, 2004; Marbach-Ad, 2001; Todd & Romine, 2018; van Mil et al., 2016; Venville & Treagust, 1998). Individuals' understanding of genetics, i.e., their level of genetic literacy, has ramifications for personal and civic decision-making (Bates et al., 2005; Christensen et al., 2010; Condit, 2010; Lanie et al., 2004; Moster et al., 2009; Pearson & Liu-Thompkins, 2012; Shea, 2015). Genetic literacy, or lack thereof, is also associated with cognitive biases that result in overestimating the influence of genes on trait variations and ascribing genetic differences, and consequently differences in cognitive and behavioral traits, to racial groups (Dar-Nimrod & Heine, 2011; Donovan, Chap. 10, this volume).

Although genetic literacy is critically important, current curricula and texbooks in many countries do not reflect advances in genetics research and technology, and continue to promulgate deterministic and essentialist views of genetics (Donovan, 2017; dos Santos et al., 2012; Dougherty et al., 2011; Gericke & Hagberg, 2007; Gericke et al., 2014). A recent consensus-building study for genetic literacy (Boerwinkel et al., 2017) proposed three core dimensions of knowledge that are needed for decision-making about genetic-related issues: conceptual knowledge of core genetic concepts, sociocultural knowledge of how applications of genetic research and technology impact society, and epistemic knowledge of the meaning and certainty of genetic information. In particular, that report highlighted, as a central message, the importance of helping students understand the contribution of multiple genes, in interaction with multiple environmental factors, to the observed variation in traits.

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Such an ambitious, yet necessary, mandate for genetics education raises the obvious question of how we should structure the curriculum and instruction, across K–12 (and beyond), to foster a more complex, nuanced, and socially responsible understanding of genetics. One clear implication is that genetics education needs to begin earlier in the course of schooling (Elmesky, 2012). A second implication, is that we need to progressively develop students' cognitive, sociocultural, and epistemic knowledge of genetics—a learning progression toward genetic literacy. Fortunately, there already exist multiple hypothetical learning progressions in genetics (Dougherty, 2009; Duncan et al., 2009; Elmesky, 2012; Roseman et al., 2006; Todd et al., 2017). However, they differ substantially from one another and thus engender very different recommendations for curricula and instruction. The field would therefore benefit from an attempt to synthesize insights across these progressions that can provide more coherent recommendations for curricula and instruction.

In this chapter, we review the research on these genetics learning progressions and compare their conceptualizations of learning in the domain. We then discuss some critiques of learning progression scholarship, and while these do not target the genetics progressions, they are nonetheless relevant. These critiques raise several questions about: (1) potential end points of a learning progression for genetics that go beyond conceptual knowledge, (2) how a progression can account for the differences in reasoning across different genetic contexts that are empirically documented, and (3) validity concerns regarding the learning progressions enterprise and their potential utility for education. We end by offering some provisional answers to these questions and suggesting some instructional implications and directions for future research.

4.2 Current Knowledge About the Problem

Learning progressions embody a developmental approach to learning that describes hypothetical paths to developing a progressively more sophisticated understanding of core ideas and scientific practices in a domain over time (Smith et al., 2006). The learning progression begins with a lower anchor that represents the knowledge and practices students bring with them and that serve as the starting point for learning. The target end points, or upper anchor, of the progression reflect the anticipated understanding by the end of the progression; a progression's upper anchor is often informed by societal expectations and expert analyses of the domain. The bulk of the progression describes the paths from the lower to the upper anchor and may encompass shorter (single unit) or longer (grade band) time periods. There are several key characteristics of learning progressions in science. First, progressions focus on a few big ideas and inquiry practices that are powerful and generative in the domain. Second, the descriptions of intermediate steps or levels between the lower and upper anchors of a progression are grounded in research on student learning in

that domain. Third, progress along a progression is not developmentally inevitable; rather, it is facilitated by carefully designed instruction.

Many learning progressions have been developed over the past two decades in science, including several progressions in genetics. The extent to which these genetics progressions are grounded in prior research, and the extent to which they have been empirically tested vary dramatically. However, they all offer important insights into what are considered important aspects of understanding in the domain and how these may develop over time. In the following, we provide a brief overview of four progressions, and then compare and contrast them to draw out key distinctions and similarities in how they conceptualize learning and development in genetics.

4.3 Molecular Basis of Heredity, Atlas of Science Literacy

This progression was developed by Roseman et al. (2006) as part of the American Association for the Advancement of Science Project 2061. It describes the ideas students should understand, and how ideas in one grade band contribute to development of ideas in the following grade band. It is focused on the middle- and high-school grade bands, but it does include foundational ideas from lower grade bands such as the understanding that offspring resemble their parents (grades K–2). The authors argue that to reason about heredity, students need a coherent understanding of two main functions of DNA: (1) determining the characteristics of organisms and (2) passing information from one generation to the next. A foundational assumption of this progression is that students need to first understand mechanisms at the cellular and molecular levels, that is, the role of DNA in determining protein structure and function, and the subsequent role of proteins in mediating genetic traits, before they can understand ideas about Mendelian inheritance patterns (alleles and modes of inheritance). This recommendation stands in contrast to the typical treatment of the topic in textbooks, where Mendelian inheritance is presented before molecular genetics, DNA is discussed mostly in terms of structure and its role in protein synthesis, and the role of proteins in genetic traits is scarcely discussed beyond a few examples involving genetic disorders (Kurth & Roseman, 2001). Roseman et al. (2006) argued that understanding how DNA stores genetic information and brings about its effects (i.e., observable traits) is a necessary prerequisite to understanding genes, chromosomes, and the more abstract ideas of classical genetics and inheritance patterns. Their progression thus engages middle-school students with molecular genetics and relegates Mendelian genetics to high school.

4.4 Modern Genetics Learning Progression

This progression was developed by Duncan et al. (2009). It describes the development of genetics understanding from upper elementary school (grade 5) to the end of compulsory education in high school (grade 10). The progression is organized around two questions: (1) How do genes influence how we, and other organisms, look and function? (2) Why do we vary in how we, and other organisms, look and function? It is comprised of eight big ideas (constructs). These ideas span three conceptual models in genetics described by Stewart et al. (2005) as important for genetic literacy: (1) the inheritance model—describing the patterns of correlations between genotype and phenotype; (2) the meiotic model—describing how genetic material is physically passed from one generation to the next; and (3) the molecular model—describing how genes (DNA) bring about their observed effects through cellular and molecular mechanisms involving proteins. In addition, Duncan et al.'s (2009) progression adds the idea that environmental factors can interact with genetic information and influence trait variation. Growth in sophistication, according to this progression, entails understanding each of these conceptual models more deeply, and the connections between them. In contrast to Roseman et al.'s (2006) progression, the modern genetics progression does not assume that understanding the molecular model must precede understanding the inheritance (Mendelian) and meiotic models. Rather, the eight ideas develop in concert, beginning with simpler versions of all eight in late elementary school (level 1) and the deepening of each idea across the middle-school (level 2) and high-school (level 3) grade bands. Since its inception, Duncan et al.'s (2009) progression has been empirically studied and refined iteratively, including the addition of sub-constructs (sub-ideas) and levels (Duncan et al., 2016, 2017; Shea & Duncan, 2013). It has also served as the basis for further investigation and refinement by other researchers (e.g., Todd & Kenyon, 2016; Todd et al., 2017). The modern genetics progression is the only one of the four progressions (described here) that has been studied in classrooms and at various grade levels (middle school to college). There are multiple instructional units and assessments for this progression developed by the two research groups that have studied it most extensively (Castro-Faix et al., 2018; Duncan et al., 2017; Todd & Kenyon, 2016).

4.5 Learning Progression in Genetics—Protein Expression

The third progression we discuss was developed by Elmesky (2012) and it positions the upper anchor as the understanding of inheritance as protein expression. This progression begins in the kindergarten and continues through high school. The progression towards an understanding of traits as the expression of proteins begins in the K–5 segment by supporting students' understanding of basic biology concepts, such as the distinction between living and non-living, the relationship between

biological structures and functions, and the cell as the basic unit of life. The upper-elementary to middle-school focuses on the transition from a theory of kinship that simply recognizes similarities between kin, to a theory of genetics in which genes provide the explanatory power underlying inherited traits (Springer, 1999). In this grade band, there is an extensive focus on cells with an emphasis on their structures and functions (which will be later explained by differential expression of proteins). The final segment, middle-school to high-school, begins by building on the understanding of cells as the basic unit of life and focuses on the processes of cell splitting and gamete production. Building on the idea of gametes, the progression proceeds to an understanding of patterns of inheritance and finally, to understanding genetic inheritance as protein expression. The main contribution of this progression is the more comprehensive specification of what ideas should be developed in the very early grades to support more sophisticated understanding in later grades.

4.6 The Inverted Genetics Curriculum

The last progression that we discuss is better described as an alternative paradigm to sequencing the genetics curriculum. While not grounded in extensive research about student learning in genetics, it nonetheless makes a compelling argument for inverting the curriculum to focus first on complex polygenic traits before teaching about the simpler, but rare, monogenic traits. The inverted curriculum was proposed by Dougherty (2009) in response to the poor performance of US students in genetic assessments, and in particular their lack of understanding of complex genetics. Dougherty (2009) argued that the focus on simple Mendelian inheritance, at the expense of teaching complex multifactorial inheritance, results in a poor understanding of genetics and contributes to a damaging deterministic view of inheritance. Therefore, Dougherty (2009) suggested beginning instruction with quantitative traits (height, arm length), which can be understood in terms of contributing factors (genetic and environmental) that cumulatively lead to a greater manifestation of the trait. Once students understand some of the complexity involved, they are ready to learn how genetic factors are inherited and associated with traits through the simplified models of Mendelian inheritance. While Dougherty (2009) does not explicitly specify the grades in which these ideas can be introduced, he postulates that middle-school students would be able to understand the basics of multifactorial traits.

4.7 Comparison of Progressions

In thinking across these progressions, we note several key distinctions; first, in the extent to which the progression assumes the need for a mechanistic understanding of molecular genetics (role of DNA and proteins in trait expression) to precede the

understanding of inheritance patterns (simple or complex). Roseman et al.'s (2006) progression makes the strongest claim for molecular understanding coming before the more abstract ideas of alleles and modes of inheritance. Elmesky's (2012) and Dougherty's (2009) proposals recommend the opposite sequence and advocate for understanding inheritance patterns first. Dougherty (2009) further argues that complex (multifactorial) patterns need to precede simple ones (Mendelian); however, he remains ambivalent about the placing of molecular genetics, and his proposal offers two options for incorporating ideas in molecular genetics (gene expression and protein synthesis) either before or after teaching the simpler Mendelian model. Elmesky's (2012) progression acknowledges the importance of understanding structure–function relationships and cells as precursors to meiosis and inheritance patterns, but it does not advocate for teaching about protein expression till later. Duncan et al.'s (2009) progression and its subsequent revisions by both research groups (Duncan et al., 2009, 2017; Todd & Kenyon, 2016) advocates for teaching all ideas from the start, at developmentally appropriate levels, with the assumption that ideas about molecular genetics and inheritance patterns can bootstrap each other. However, recent research by Duncan et al. (2017) has shown that there is a slight benefit to teaching molecular ideas first. In a comparison study of two instruction sequences that differed in which ideas were introduced first, Mendelian or molecular genetics, high-school students who had the molecular-first sequence performed slightly better (albeit not significantly so) across all constructs than their peers in the Mendelian-first sequence. While these findings are inconclusive given the lack of statistical significance, it is somewhat surprising that the advantage of the molecular-first sequence was seen for all constructs—both those learned early (molecular) and those learned later (Mendelian).

A second point of difference between the progressions is the emphasis on complex multifactorial traits and the role of environmental factors in trait expression. Dougherty (2009) takes a strong stance here, advocating for the need to deal with the more complex first (quantitatively) and to develop the underlying and simplified Mendelian model only after students understand that trait variation is explained by multiple factors—genetic and environmental. Duncan et al.'s (2009) progression does include ideas on the role of environmental factors in altering gene expression and influencing traits' expression. However, it does not include ideas about polygenic traits or the role of multiple genes in influencing trait expression. Ideas about polygenic inheritance are assumed to be learned in higher grades beyond the upper anchor of the progression. Elmesky's (2012) progression does not include either of these ideas—polygenic or multifactorial—and remains mute on when they should be taught. The jury is still out on when (and how) these ideas should be introduced to students as there is no study that juxtaposed complex vs. simple inheritance. However, it is clear that if we want students to develop more robust, non-deterministic and non-essentialist views of genetics, we need to engage them with multifactorial genetics in K–12.

The third and final point we raise is not one of contention, but one of omission. None of the proposed progressions tackle two of the core areas of knowledge identified by Boerwinkel et al. (2017) as important for genetic literacy: sociocultural

knowledge of genetics, and epistemic knowledge of genetics. Sociocultural knowledge refers to understanding the implications for citizens and society of advances in genetics (such as genetic screening, genetically modified crops, etc.). Given recent research in genetics education, it seems that another aspect of sociocultural knowledge that is highly relevant to genetic literacy is the conceptualization of genetic differences within and between populations, and the impact of environmental factors (including the social environment) on genetic traits (see Donovan, Chap. 10, this volume). Epistemic knowledge relates to reasoning about claims regarding genetic contribution to traits, genetic risk, and other uses of genetic information. In particular, understanding the certainty and validity of claims generated from genetic data is critical for genetic literacy given the prevalence of genetic technologies, including affordable genome analyses, in the public sphere. We currently lack the necessary research regarding reasoning about sociocultural and epistemic aspects of genetics to inform new constructs for these key areas of genetics knowledge.

4.8 Remaining Issues

We would be remiss if we did not discuss some of the current critiques of the learning progression enterprise in general, as these also apply to the genetics progressions. Originally conceived as an attempt to bring more coherence to standards, curricula, and assessments, learning progressions have, in fact, informed standards reform efforts in the US (National Research Council, 2012). While many remain hopeful about their prospects (Alonzo & Elby, 2019; Duncan & Rivet, 2018; Duschl et al., 2011), there have been several substantial critiques of progressions that question their validity as models of learning (Alonzo & Elby, 2019; Hammer & Sikorski, 2015; Sikorski & Hammer, 2010; Sikorski, 2019; Steedle & Shavelson, 2009). In this chapter, we discuss two of these critiques and their implications for the genetics learning progressions: the messy middle problem and expanding our conceptualization of the upper anchors.

4.8.1 Messy Middle

Learning progression research has shown that students at the ends of the progression perform consistently (i.e., reason at the same level across most tasks), but those in the middle show much less consistent performance; this makes it difficult, if not impossible, to "diagnose" their level of reasoning (Gotwals & Songer, 2010; Steedle & Shavelson, 2009). The phenomenon can be partly explained by reference to the context dependence of student performance, i.e., students' performance on an assessment task has been shown to depend on the contextual features of the task, such as the organism and trait involved (Schmiemann et al., 2017; Shea et al., 2015). For example, students tend to perform at a lower level of sophistication on genetics

tasks involving plants vs. humans (Shea et al., 2015). Current learning progressions in genetics do not model how contextual features of genetic phenomena influence students' reasoning or their demonstrated level of sophistication. Thus, there may be a lot more nuance to students' learning in this domain that is not being captured by current progressions, and this might explain why students' reasoning, in the middle levels of the progression, appears messy.

In addition to the issue of learning progressions missing the nuance of contextual features, there may be a second explanation for the observed messy middle—the tentative nature of learning and the "two steps forward one step back" process involved (Duncan & Rivet, 2018). The assumption of progression as succession, with each level representing a more sophisticated view that "replaces" the prior level (resulting in robust reasoning at the new level), is inconsistent with the available empirical data from learning progression research (Alonzo & Elby, 2019). Moreover, the assumption of consistent and robust reasoning at a particular level is also at odds with a situated, knowledge-in-pieces perspective on reasoning (Hammer & Sikorski, 2015). These inconsistencies represent serious problems with our construal of learning progressions and they will not be easily resolved without changing core aspects of how we conceptualize, and use, progressions.

However, learning progressions, including the progressions in genetics, can still be useful despite some of their empirical validity problems (i.e., inability to diagnose students' level of understanding due to the messy middle problem). They can be useful in helping teachers attend more fully to nuances in students' thinking. Research on teachers' use of progressions (Alonzo & Elby, 2019; Furtak, 2012; Furtak & Heredia, 2014) suggests that they can use them to inform their formative assessment practices. For example, teachers can generate more fine-grained analysis of student thinking and identify more specific and actionable ideas for subsequent instruction. To date, there has been no focus on teachers' use of progressions specifically in genetics. The genetics progression that has been more fully developed, and for which there are validated assessments (Duncan et al., 2016; Todd et al., 2017), will likely be more useful for teachers in terms of informing their assessment and instructional practices. Nevertheless, additional research is needed to identify the most useful representations of learning along this genetics progression for teachers.

4.8.2 Conceptualization of the Upper Anchors

The upper anchor represents the most sophisticated way of reasoning targeted by the progression and, as noted above, replacing prior, less sophisticated ways of reasoning. However, what qualifies as "most sophisticated" depends on the situation. Sometimes, using simple Mendelian genetics may be sufficient (e.g., to explain the likelihood of having a child with phenylketonuria given heterozygous parents); in other situations, it may not (e.g., explaining the risk of developing breast cancer if one carries the *BRCA1* gene). Even reasoning that (at face value) seems to be

broadly inappropriate, for example, ignoring anomalous data, is appropriate under some conditions, and scientists do employ such reasoning strategies themselves (Chinn & Brewer, 1993). Currently, our definition of the upper anchor is derived from normative (aligned with scientific cannon) expectations and is informed by experts' analyses of the domain and what students should know about it (i.e., the kinds of analyses that inform what ideas, and at what depth, should be included in textbooks). However, we may need to adopt a more expansive definition of the upper anchor that is much more sensitive to the situational appropriateness of reasoning (Sikorski, 2019). That is, what counts as sophisticated reasoning at the highest level (i.e., upper anchor) depends on the situation, and there may be a plurality of upper anchors that are appropriate for different situations.

This need to broaden and situate the upper anchor becomes an even bigger issue when we think of different contexts in terms of expert and lay reasoning. Genetic literacy is not about becoming a genetics expert. In fact, given the cognitive division of labor in society, lay persons will never have the deep disciplinary knowledge of theories and methods in a domain that would allow them to reason about it as experts do (Chinn & Duncan, 2018; Keren, 2018). Therefore, it is more productive to think of literacy in terms of becoming a competent outsider (Feinstein, 2011). The competent outsider is an individual who has the ability to make appropriate judgments about the credibility of claims based on cues such as source credibility (e.g., professional reputation, publication venue) and degree of consensus in the field; such reasoning is possible even when one does not understand the disciplinary details or technical nuances of the methods.

Positioning genetic literacy as being a competent outsider also expands what counts as "most sophisticated." Moreover, ways of reasoning that are sophisticated and productive for a lay person may not be viewed as such by disciplinary experts. This is because productive reasoning strategies for lay persons are very different from the reasoning strategies of disciplinary experts (Keren, 2018). For example, lay persons may not be able to expertly evaluate the methods used to generate evidence in support of a claim about a genetic issue, but they can evaluate whether the scientific community accepts this claim (extent of consensus), and whether the expert community views the methods used to generate it as reliable. Such distinctions between lay and expert reasoning should be taken into account when defining expectations for the upper anchor. Perhaps research that identifies the necessary knowledge for the competent outsider is a more useful way to define the upper anchor. That is, the upper anchor should include knowledge needed by the competent outsider to solve a genetic issue or problem relevant to the situation they are dealing with (Feinstein, 2011; Sikorski, 2019). For example, one might expect a competent outsider to be able to make sense of claims related to genetics issues that appear in the news. An attempt to identify these types of understanding was carried out by Shea (2015), who analyzed genetics-related articles in the New York Times science section. She found that those articles often entailed understanding molecular genetics ideas that corresponded to the highest levels of the Duncan et al. (2009) progression, but only a lower level understanding of Mendelian genetics ideas. Moreover, Shea (2015) found that some articles entailed an understanding related to

conserved genetic sequences, and comparisons of sequences across species, which went beyond the upper-level expectations of the modern genetics learning progression (Duncan et al., 2009). It is therefore clear that the upper anchor expectations of progressions do not always overlap with what is actually needed to be a genetically literate competent outsider.

The analysis conducted by Shea (2015) only addressed the content or cognitive dimension of knowledge. However, Boerwinkel et al. (2017) proposed three dimensions of knowledge—cognitive, sociocultural, and epistemic—presumed to play a role in being a competent outsider. We need to think about what might be the range of different "most sophisticated" ways of reasoning in different situations in terms of these three knowledge dimensions, while accounting for the aim of promoting a sophisticated lay person. Currently, the progressions in genetics only deal with the cognitive dimension, and even then, the definition of "most sophisticated" is unitary and does not take situational context into account. As already noted, whether one uses simple monogenic inheritance models or the more complex multifactorial ones depends on what one is trying to explain or understand in a particular situation, i.e., one's situated epistemic aims. This is not to say that the suitability of the model is subjective; rather, the determination of which model is appropriate varies and depends on the situation. Being able to know which model to use and when (i.e., to use them adaptively) is a core part of being genetically literate; such understandings should be encompassed by the epistemic knowledge dimension. The upper anchors of a comprehensive progression should reflect this notion of adaptiveness or situational appropriateness for all three knowledge dimensions (cognitive, sociocultural, and epistemic).

4.9 Implications for Teaching

In this section, we present three key recommendations for teaching genetics derived from our discussion of the merits and pitfalls of existing learning progressions in genetics. These recommendations are tentative in nature and therefore also reflect avenues for future research in genetics education to examine their fruitfulness and refine them further.

4.9.1 The Explanatory Role of Molecular Mechanisms

One of the longstanding questions in genetics education is whether teaching the molecular mechanisms of genetics (contribution of genes and proteins to phenotypic variation) should precede teaching about Mendelian inheritance patterns. Research addressing this question, while not conclusive, provides tentative evidence that students do fare better when they learn molecular genetics before Mendelian genetics (Duncan et al., 2016, 2017). Moreover, understanding the cellular and

molecular basis of inheritance may also reduce the tendency to think deterministically about genes and traits. By providing mechanisms to open the black box of how genotype brings about phenotype, students can better understand how genetic variation (mutations) can result in variations in phenotype, giving them a more nuanced view of inheritance (Duncan & Tseng, 2011), rather than considering genes and traits as almost synonymous (Lewis & Kattmann, 2004).

4.9.2 Tackling the Complexity of Multifactorial Traits

Given the problems associated with deterministic and essentialist thinking in genetics (e.g. Carver et al., 2017; Dar-Nimrod & Heine, 2011; Jiménez-Aleixandre, 2014; Keller, 2005; Donovan, Chap. 10, this volume), it behooves us to begin teaching about multifactorial traits as a core part of genetics education in K–12 (Dougherty, 2009). Reasoning about multifactorial traits, and the contribution of genes and environmental factors to trait variation, requires understanding some statistical ideas, including randomness, distributions, and probability. Thus, it may be appropriate to begin such instruction in the middle grades once students have some statistics knowledge. Donovan et al. (2019) and Dougherty (2009) provide some ideas about instructional activities that can support student learning about multifactorial inheritance and trait variation.

Research on students' understanding of multifactorial inheritance is sparse and certainly insufficient to inform a progression. In particular, we know little about how students conceive of the interactions between environmental factors and genetic factors, including mechanisms involving changes to gene expression and epigenetic changes to the genome. Recent effort suggests that high-school students and even undergraduates struggle to explain how environmental factors impact phenotype through alterations in gene expression (Haskel-Ittah et al., 2020; Todd & Romine, 2018). The notion that organisms can sense and respond to environmental factors in ways that alter which proteins are expressed and when is central to understanding phenotypic plasticity. In general, teaching about regulation of gene expression and epigenetic modifications is not prevalent at the K–12 level. Much more research is needed to understand how students reason about, and how to teach, these core ideas.

4.9.3 Lay Reasoning in Genetics: Fostering Competent Outsiders

As noted earlier, none of the current genetics progressions tackle the sociocultural and epistemic dimensions of knowledge (Boerwinkel et al., 2017). Moreover, as a field, we lack a robust and specific theory of what the knowledge dimensions really entail; nor do we have sufficient research on how students reason about the

epistemic and sociocultural considerations involved in current genetic issues and controversies. However, there is relevant research, albeit not specific to genetics, which can inform our efforts along these lines. For example, Samarapungavan (2018) provides a framework for unpacking what it means to reason with evidence in the context of a disciplinary domain. She argues that reasoning with evidence in a particular discipline requires the integration of disciplinary knowledge of theories with epistemic considerations related to inquiry practices in that domain. This framework provides a sense of how the interactions between disciplinary knowledge of practices, theories (cognitive), and epistemology play out in a life science domain and could be useful for thinking about similar interactions in the specific domain of genetics. However, the focus of this framework is on expert evidentiary practices—the kinds of epistemic considerations that govern the practices of scientists. The extent to which students can develop the level of disciplinary sophistication necessary to reason about the kinds of epistemic considerations advocated by Samarapungavan (2018) is unclear. It is also not clear whether such reasoning would also serve lay persons in their engagement with genetic issues as competent outsiders.

A second, similar framework, developed by Duncan et al. (2018), is more explicit about the distinction between lay and expert evidentiary reasoning. The grasp of evidence framework (Duncan et al., 2018) posits five dimensions of reasoning with and about evidence in science; four of the dimensions deal with expert evidentiary practices and the fifth specifically addresses lay persons' evidentiary reasoning, taking into account the substantial difference between such reasoning and that by experts, given the division of cognitive labor (Keren, 2018). For instance, a lay person may need to determine the credibility of scientific claims in news articles. To do this, they may have to rely on their capacity to determine the trustworthiness of the source, the relevance of the authors' expertise, and the extent of consensus in the scientific community regarding the claims. The grasp of evidence framework can also be used to think about the epistemic considerations that are important for lay reasoning in genetics; for example, what lay people should understand about the potential confounding factors in determining the risk of developing a particular disorder based on data from DNA sequencing, such as provided by 23andMe. Obviously, lay individuals are not in a position to understand the nuances of the methods used by the company, nor do they understand the implications of these methods for interpreting the results in terms of personal risk. However, lay individuals can develop an understanding of the large complexity involved in the method with its sources of uncertainty, and that the results account only for genetic makeup but not for environmental factors, meaning that the ultimate risk cannot be surmised from these results alone. Lay individuals should understand that they need to consult the relevant experts to determine their own risk, and that they should be skeptical of encompassing claims made based on genetic data alone given that most (if not all) traits are multifactorial. Again, operationalizing the framework in terms of genetic literacy and testing its utility in educational settings will require additional research.

To conclude, recent advances in genetic technology are placing a greater demand for genetic literacy on the public. As genetics education researchers, we need to figure out ways to better prepare students for personal and civic engagement with current and future genetic dilemmas. The existing learning progressions offer important and substantive insights on learning and how to support more sophisticated reasoning in genetics. However, none of these progressions fully operationalize what it means to be genetically literate. Future research is needed to examine how to support student learning of the cognitive, sociocultural, and epistemic dimensions of genetic literacy—specifically, how to differentiate lay reasoning from expert reasoning in the domain (fostering competent outsiders) and what this entails instructionally in terms of fostering adaptive use of genetic models (knowing which models are best for which situations) and adequate reasoning with evidence in this domain. It is an exciting time for the field, one that will usher in important shifts in how we conceptualize the teaching and learning of genetics for socially responsible genetic literacy.

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Chapter 5
How Can We Help Students Reason About the Mechanisms by Which Genes Affect Traits?

Michal Haskel-Ittah

5.1 The Problem

We have entered an era in which genetic tools are constantly being developed and used by researchers and industry. In this era, every individual can obtain information about his own DNA sequences and consume genetically engineered products. This rapid invasion of genetic issues into our everyday lives has not escaped the media's attention, where these issues are frequently raised, albeit not always in a scientifically correct manner (Stern & Kampourakis, 2017). Hence, today, knowledge about genetics is essential for the ability to make informed everyday decisions and to engage in discussions about ethical issues and the health benefits of applying these genetic tools. Mere familiarity with genetic concepts is not enough. A deeper understanding of genetic mechanisms, the limits of our understanding of them and the boundaries of their predictive power is required (Boerwinkel et al., 2017; Gelbart, 2012; Stern & Kampourakis, 2017).

Almost 20 years ago, it was already being acknowledged that education that wishes to prepare the public for the genetic revolution should itself undergo a change in the oversimplified description of genetics in school (McInerney, 2002). Haga (2006) suggested that educational resources in genetics must be continuously updated and revised to narrow the gap between concurrent scientific findings and public knowledge. The purpose of narrowing this gap is not merely to be up-to-date with current scientific knowledge, but also to comprehend how scientists' understanding of genetics may affect citizens' everyday lives.

For the last 20 years, the understanding of the mechanisms by which genes affect traits has been continually growing. Scientists have revealed the complexity of these mechanisms which involve multiple genes, regulatory pathways, and interactions with the environment. These mechanisms are the basis for technologies and genetic

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tools, and they provide the limits for what can and cannot be learned from the DNA sequence. However, when examining how the public today acknowledges the complexity of the relationship between genes and traits, many people seem to hold an oversimplified conception of this relationship (Chapman et al., 2019; Gericke et al., 2017). For example, people suggest that complex traits such as autism are the product of a single genetic variant, and that a person's behavior can be predicted from looking at their DNA sequence (Chapman et al., 2019).

If our aim is to help students reason about gene-to-trait mechanisms to support everyday decision making and discussion, we should assist them in understanding these mechanisms and their complexities. This is also important for the goal of developing a mechanistic understanding in general, an issue that has been acknowledged for its contribution to critical thinking (Ahn & Kalish, 2000; Koslowski, 1996), as well as to a deep understanding of science (Chin & Brown, 2000; Grove et al., 2012). The question is, how can we support students' mechanistic understanding and mechanistic reasoning in these genetic contexts? To answer this question, we must review what we know about teaching genetics from a mechanistic perspective.

5.2 Current Knowledge About the Problem

All natural phenomena can potentially be explained by underlying mechanisms. Such mechanisms consist of entities with specific properties that enable them to conduct certain activities (Craver & Darden, 2013; Machamer et al., 2000). These activities cause changes which eventually lead to the observed phenomena (Craver, 2001; Darden, 2008). Hence, to understand a mechanism and to reason about similar mechanisms, one should be able to identify and characterize the entities involved in the mechanism, and recognize the activities and functions executed by those entities (Russ et al., 2008). Those entities and interactions should be at least one organizational level lower than the phenomenon itself, and they should be able to explain how interactions between entities at this level lead to the final outcome—the phenomenon (Krist et al., 2018).

In biology, mechanisms typically extend across multiple levels (Craver & Darden, 2013). In the case of the effect of genes on traits, the characterization of the phenomenon (gene to trait) suggests that multiple levels should be addressed in the mechanism: from the molecular gene level to the organismal trait level. In multicellular organisms, this requires a deep understanding of the central entities that act at each level (e.g. Haskel-Ittah & Yarden, 2017; Marbach-Ad & Stavy, 2000; van Mil et al., 2013). However, in the case of genetics teaching, the representation and description of the entity—the gene—is not consistent among textbooks, making it hard for students to thoroughly understand its properties (Gericke et al., 2014; Livni-Alcasid et al., 2018). For example, an analysis of eight textbooks from three different countries revealed inconsistencies in allelic symbols throughout all of them with no explicit explanation for the symbol alterations (Livni-Alcasid et al.,

2018). Gericke and Hagberg (2007) identified five historical models that included different descriptions of the concept of gene. In each model, the properties and function of genes differed. For example, in one model, the gene's function was to hold information for traits (Mendelian model) and in another, it was to manufacture specific proteins (neoclassical model) or code for RNA (modern model). In one model, the gene was an abstract idea with no structure or composition (Mendelian model) and in another, it had the property of being located at a specific locus on the chromosome (classical model). An analysis of science textbooks from six different countries revealed the sporadic appearance of the five different models among textbooks (Gericke et al., 2014). From a mechanistic perspective, this is highly problematic because an entity's description should include its properties and function. When genes are described inconsistently in each historical model, with different properties and functions, it may be confusing in terms of which properties and activities should be used for the purpose of mechanistic reasoning. Such a description may lead to confusion regarding genes as entities in the mechanism.

Proteins, which are central entities in genetic mechanisms, are also difficult for students to understand and use in genetic explanations (e.g. Duncan & Tseng, 2011; Haskel-Ittah & Yarden, 2017; Marbach-Ad & Stavy, 2000). Teachers often refer to proteins as merely gene products without further denoting their active involvement in the mechanism (Thörne & Gericke, 2014). Aside from the inaccuracy of describing proteins as if they were side products of genetic mechanisms, this description offers students a fragmented mechanism that does not explain most aspects of genetics. These problems might explain why students often regard genetic phenomena as cause-and-effect relationships between genes and traits, and not as the result of a complex causal mechanism (Duncan & Tseng, 2011; Haskel-Ittah & Yarden, 2017; Haskel-Ittah et al., 2019).

No less important than these central entities—genes and proteins—is their connection. A comparison between how biology and chemistry textbooks describe the connection between genes and proteins revealed that in the former, the mechanistic connection is not emphasized (Wahlberg & Gericke, 2018). Wahlberg and Gericke (2018) characterized the description of protein synthesis in biology textbooks as more focused around the concepts in isolation, rather than on how their interactions lead to a final outcome. Although characterizing the entities involved in a mechanism is an important part of mechanistic reasoning, it is not enough to provide a mechanistic explanation. With no description of the dynamics between entities and how this leads to the emergence of a certain phenomenon, there is no connection between set up and final conditions, and therefore, no actual mechanism (Krist et al., 2018; Russ et al., 2008).

Another problem emerges from the erroneous descriptions of the relationships between genes and traits: the complexity of the gene-to-trait mechanism is often ignored in the classroom, suggesting that there is a one-to-one relationship between genes and traits. A study with preservice biology teachers showed that they tend to ignore the complexities of these mechanisms. These teachers' explanations about the origin of traits in individuals mostly referred to genes as causes, but not to a process or mechanism (Kampourakis et al., 2016). Moreover, textbooks often

provide examples of classical genetic disorders or traits and rarely deal with multifactorial disorders and traits, leading to the misconception that multifactorial traits are the exception rather than the rule (Dougherty, 2009; Hicks et al., 2014). Such examples outline a simple one-to-one relationship that will, later on, be discordant with knowledge about other components in the mechanism (e.g. proteins). This again may lead students to abandon a mechanistic explanation in favor of a simple cause-and-effect one.

While not prevalent in high-school education, it seems that at a young age, children can already think of genetic phenomena as the result of a mechanism. Although preschoolers may provide inconsistent and sometimes teleological explanations for kinship (Williams, 2012), when they are asked to choose between a mechanistic explanation which involves physical entities and a teleological one that involves intentions, they display a preference for the mechanistic one (Ergazaki et al., 2014; Springer & Keil, 1991). A study conducted with high-school students showed that a mechanistic explanation for genetic phenomena, but not memorization of genetic concepts, was retained in the students' memory even 18 months after instruction (Todd & Romine, 2018). In addition, studies show that causal knowledge plays a major role in the ability to provide a mechanistic explanation. In the absence of causal knowledge, other intuitive explanations (e.g., teleological) emerge (Kampourakis & Zogza, 2009; Trommler et al., 2018). This implies that at least some of the difficulties encountered by students in reasoning about genetics are not because they do not recognize the superiority of a causal mechanism over other explanations in this context, but because they lack the domain-specific knowledge to build one. Indeed, domain-specific knowledge has been shown to play a major role in students' ability to reason in genetics (Duncan, 2007).

5.2.1 A Possible Solution

If we acknowledge that the ability to reason mechanistically in genetics is based on conceptual and mechanistic knowledge in genetics, then conceptual knowledge and mechanistic understanding in genetics should be intertwined and mutually developed. By reviewing some of the literature in the field of genetics education, I came up with three milestones in the development toward a mechanistic understanding of the effect of genes on traits. These three milestones represent levels in the progression from a more naïve phenomenal description of the relationship between genes and traits to a complex understanding of the entities and activities that are involved in these mechanisms.

I. **Establishing a Correct Causal Connection between Genes and Traits.** The first step in the search for mechanisms is a description of the target phenomenon. This description outlines the borders for the space of the mechanistic explanations (Craver & Darden, 2013; Darden, 2008). In the gene-to-trait mechanism, this space is defined between genes and traits. Thus, the first milestone in under-

standing genetics (prior to learning about the mechanism) is an acknowledgment of this space.

Studies in genetics education have mapped numerous alternative conceptions of genetic concepts. Among others, they found a misconception regarding the relationship between genes and traits: the notion that genes are trait-bearing particles or genes and traits are synonyms (Lewis & Kattmann, 2004; Venville & Treagust, 1998). This alternative conception is an incorrect description of the phenomenon of causal relationship between genes and traits. More than a decade later, we decided to explore both the prevalence of this alternative conception, and its effect on mechanistic understanding in genetics. In our study (Haskel-Ittah & Yarden, 2018), we used concept maps to explore the manner in which students describe the relationship between genes and traits. We found that this alternative conception was prevalent among 9th graders. Namely, 28% of the 152 students who participated in the study described genes as containing traits or as being synonyms for traits. Thus, almost a third of the students described genetic phenomena as resulting from a direct connection, which might not provide a space for a mediating mechanism.

According to the knowledge-integration perspective, the identification of a gap in one's knowledge is a crucial step toward constructing connections between new knowledge and prior knowledge (Linn et al., 2004). Describing genes as affecting traits suggests causal relationships with a mediating gap that may be further explained by searching for the mediating mechanisms. In contrast, suggesting that genes are traits or trait-bearing particles means that the gene–trait relationship need not to be explained by a mechanism because there is no gap to be filled. Knowledge about entities and activities in the mechanism would thus be fruitful, and can be linked to prior knowledge in the first case, but is irrelevant for the latter. Indeed, we found that when students described a causal relationship between genes and traits prior to instruction, they used the taught gene-to-trait mechanism as the missing link between genes and traits. These students' knowledge of the involvement of proteins improved and they were successful in reasoning mechanistically about new genetic phenomena. On the other hand, students who did not describe the gene–trait relationship as causal prior to instruction were significantly less successful in learning about proteins and in reasoning about the mechanism.

As a phenomenal description, "genes affect traits" is correct, and seems to be an important stage prior to learning about gene-to-trait mechanisms. Nonetheless, this description holds the risk of imposing a gene-centered deterministic view, i.e., that genes are the only entities determining traits. Such a view can create an educational problem because it may hinder the learning of other ideas in genetics (Jiménez-Aleixandre, 2014), and it can pose a social problem because it may lead to racism and prejudice (Donovan, 2016; Keller, 2005). Thus, teachers and educators should be aware of this risk and possible solutions for avoiding it (Stern & Kampourakis, 2017).

One suggested solution for the problem of genetic determinism is presenting multifactorial genetic traits before the description of Mendelian traits (Dougherty, 2009). Another suggestion is to focus on the developmental aspect of the mutual

effect of genes and environment on traits (Jamieson & Radick, 2017). My suggestion is to implement these two ideas at the first stage of the phenomenon description suggested here. For example, students can sort effectors on traits and conclude that genes affect traits—sometimes this involves more than one gene and sometimes genes are not the only effectors. This, of course, does not provide the mechanisms for this multifactorial effect, but it describes a multifactorial phenomenon in a manner that sets the placeholders for a mechanism (see Fig. 5.1A).

II. **Establishing the Understanding of Genes–Proteins–Traits Mechanisms.** Following the description of genetic phenomena, the mechanistic space can be filled with entities and activities. This might include entities such as RNA-polymerase, mRNA, ribosome, amino acids, various proteins, etc. As more entities and functions are incorporated into the mechanism, the cognitive load in learning this mechanism increases. Thus, in most cases, not all entities and functions will be presented. An important question is what are the key components for allowing mechanistic understanding and mechanistic reasoning in genetics? To answer this question, more empirical studies are needed. Nevertheless, in this chapter, I describe the importance of focusing on entities that are largely overlooked by teachers: the proteins (Thörne & Gericke, 2014).

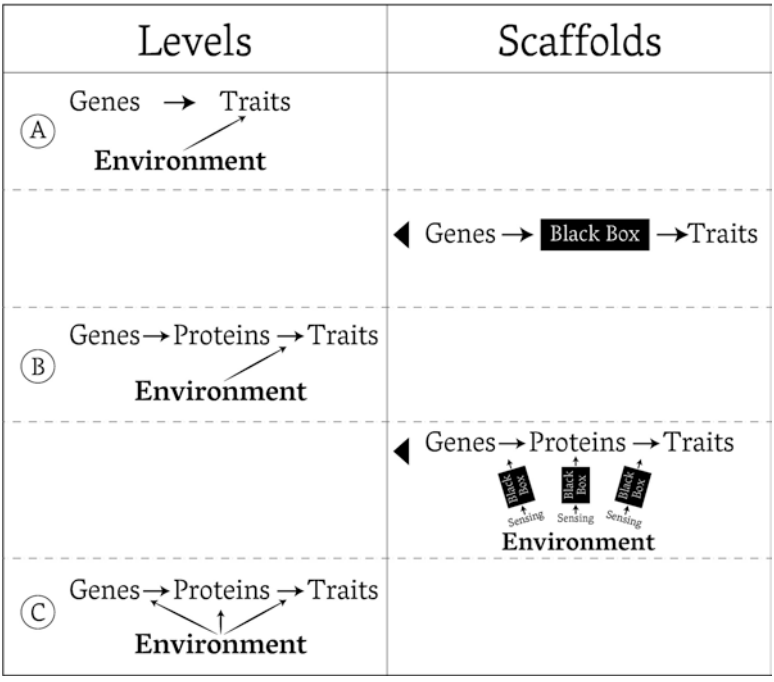

Fig. 5.1 A progression scale toward mechanistic understanding in genetics. Three milestones (A, B, C) in the development of mechanistic understanding in genetics are shown on the left, and suggested scaffolds for facilitating the progression along the scale are shown on the right

Several studies have reported that instructions that focus on protein function lead to gains in the mechanistic understanding of genetics (Freidenreich et al., 2011; van Mil et al., 2013). For example, Duncan and Tseng (2011) developed an instructional module in which 10th-grade students explored multiple phenomena involving proteins with different functions (e.g., enzymes, transporters, and channels). Following instruction, students proposed proteins as key players in their accounts of molecular genetics phenomena and were able to describe the roles of those proteins in the suggested mechanism. Van mil (2016) pointed out that learning about three general protein activities (colliding, binding and changing shape) can bridge an explanatory gap between the molecular and cellular levels. An implementation of this approach with high-school students showed improvement of students' reasoning about cell biology. In a study conducted with 7th graders, we examined how students who had learned about different protein functions (e.g., receptor, enzyme) reasoned using those functions (Haskel-Ittah et al., 2019). Our findings revealed that although the students acknowledged that genes code for proteins, their use of proteins when asked to explain the result of a genetic mutation on a specific trait was inconsistent: they used proteins to explain the given genetic phenomenon in cases that could be explained by a protein function they had learned. However, when the case could not be explained by a previously learned function, students turned back to merely describing a causal relationship between genes and traits without specifying a mechanism.

Altogether, these studies suggest that students are more likely to establish the idea of proteins as mediators between genes and traits via learning about the involvement of proteins as functioning entities and their contribution to trait formation, rather than via learning about how the DNA code is transcribed and translated into proteins. This notion is in accordance with the mechanistic perspective, whereby the presence of an entity in an explanation is important if it changes the predicted behavior of the mechanism (Craver & Darden, 2013). A change in a gene sequence may result in a change in the predicted trait if the protein's function, quantity or stability is affected by the sequence change. The RNA is also changed in this scenario, but it is impossible to explain the trait change only by the RNA change. In the case of the protein, we may describe the gene as coding for a protein whose function was changed without mentioning the RNA. Hence, proteins are central for explaining genetic phenomena.

This does not mean that translation and transcription mechanisms are unimportant, but it implies that these mechanisms may be less fruitful for the purpose of allowing students to reason about the effect of genes on traits using proteins. These mechanisms may be added later to provide a more detailed linkage between genes and proteins (see Fig. 5.1B).

III. Identifying Regulation Points and Understanding How Environmental Signals May Lead to the Modulation of Gene-to-Trait Mechanisms. The final milestone toward a mechanistic understanding in genetics includes a more complex understanding of biological mechanisms that interact with, affect and regulate genetic mechanisms. Genetic mechanisms are subject to multilevel regulation,

> from regulation of gene expression to regulation of RNA translation, protein activity and its stability. Such regulation is the outcome of mechanisms that involve multiple inputs from the internal and external environments, which are sensed and transmitted as signals that affect one or more regulation points. Understanding that gene-to-trait mechanisms are regulated by other mechanisms is important for understanding the complexity of genetics and to demarcate the limits of genetically based predictions.

In a Delphi-like study conducted to ascertain the knowledge needed for genetics literacy in the twenty-first century, the interaction between environmental factors and genetic mechanisms was considered one of the core ideas in modern genetics (Boerwinkel et al., 2017). However, in that study, similar to recommendations in other studies and reports, the interaction between genetic mechanisms and the environment was only vaguely described. This vague description included mentioning that it exists or suggesting that the environment may affect gene expression, while overlooking all other regulation points, such as protein stability (Boerwinkel et al., 2017; Dougherty, 2009; Duncan et al., 2009; National Research Council, 2012).

The mechanism by which a change in the environment may result in a change in traits is also regarded. For example, in the genetics learning progression (LP; Duncan et al., 2009), the environment is first described as influencing our traits; at higher levels, it is described as influencing cell function, and then as causing mutations or altering gene expression. There are no descriptions of any mechanisms that might link the environment to the alteration in the gene's expression. A disregard for the question of how environment might affect genetic mechanisms is also apparent in textbooks. An analysis of five Spanish textbooks showed that, although four of them defined phenotype as a result of gene–environment interactions, the texts provided only a few examples of the mechanisms for environmental influences (Puig & Jiménez-Aleixandre, 2011).

Overall, such a description provides limited knowledge about possible regulation points (only the point of gene expression) and fragmented knowledge about the mechanisms by which environmental signals can lead to the modulation of traits (only an input–output description of the environment as influencing traits). This may form a serious obstacle to understanding the effects of the internal and external environment on the regulation of gene-to-trait mechanisms.

Puig and Jiménez-Aleixandre (2011) analyzed 10th-grade students' explanations for gene–environment interactions when involved in a group discussion about a physical trait. Those students studied with different teachers about how the environment may affect genetic traits. Four out of five groups suggested that either the environment has no effect at all, or that genes and environment affect the phenotype separately, with the former having a greater influence. The two groups that suggested genes as the only influential factor studied with the same teacher, and the authors suggested that they had not had the opportunity to develop their ideas regarding possible mechanisms of gene–environment interactions in class. This indicates the importance of providing at least a general description of the possible mechanisms for gene–environment interactions.

In a study conducted with undergraduate students (Haskel-Ittah, Duncan and Yarden (2020), we provided students with four phenomena in which an environmental change led to a change in trait (e.g., skin color change as a result of UV light, low growth rate as a result of undernutrition). We interviewed these students and asked them to reason about these cases. We found that students with presumably less knowledge about biological mechanisms (most 1st-year students and some 2nd-year students) suggested that the environment affects the trait independently from the genetic mechanism or by causing mutations. Students with a higher level of knowledge were able to explain how sensing the environment might activate signals that can modulate genetic mechanisms via regulation of gene expression or other regulatory points. In addition, we found that knowledge about mechanisms of gene expression, where they were not connected to environmental signals, did not assist students in reasoning about gene–environment interactions.

From these studies, we can conclude that it is not enough to mention that the environment might affect genetic mechanisms. Without establishing a mechanistic connection between the environment and regulation of genetic mechanisms, the possibility of an environmental effect on genetic traits is not considered. For this reason, knowledge about gene regulation that is not described as the output of regulatory signals may not be fruitful in terms of reasoning about genetic phenomena that involve gene–environment interactions. Accordingly, students should first identify possible points of regulation in gene-to-trait mechanisms and acknowledge that they include mechanisms of sensing the environment, which can result in activating/inhibiting these regulation points. Only then can specific mechanisms be taught as an example of regulatory mechanisms, such as regulation of gene expression (e.g., transcription factors, epigenetic modifications) or protein modifications (e.g., ubiquitination, phosphorylation) (see Fig. 5.1C).

How to scaffold movement between the milestones of mechanistic understanding in genetics? Moving between the three suggested milestones of mechanistic understanding might be challenging for students. In this section, I suggest theoretical scaffolds for moving along this progressive scale of mechanistic understanding in genetics. These scaffolds are based on a concept that has been characterized and studied in computer science education: the black box. The term "black box" refers to knowledge which is unknown to the person receiving the explanation and/or to the one who is giving the explanation (Haberman et al., 2002). Thinking in terms of black boxes includes both the process of removing details with the aim of simplification, and the process of identifying the core essence with the aim of generalization (Kramer, 2007). Hence, thinking in terms of black boxes in explanations enables thinking in terms of conceptual ideas, rather than details (Armoni, 2009).

Black boxes are also central to mechanistic explanations in biology, because "every description of a mechanism bottoms out at some point where the gain in detail makes no difference to the researcher" (Craver & Darden, 2013, p. 90). For example, one can provide an explanation involving an enzyme catalyzing a reaction without explaining precisely how the chemical interaction between the enzyme and its substrate leads to the catalysis. In other cases, black boxes may exist simply because their contents, namely the mechanisms, are not yet understood by scientists.

Drawing on its use in computer science education, teaching using black boxes does not mean skipping over or ignoring parts of a mechanism, but characterizing them. This characterization should include an input/output description without any details on its internal function (Statter & Armoni, 2017). Such a description focuses on the functionality of the black box and its contribution to the mechanism. For example, we can describe a "regulation" black box in the gene-to-trait mechanism. The input to this black box is signals from the internal or external environment, and the outputs are inhibitors/activators of the gene-to-trait mechanism. A different "regulation" box may act on different sections of the mechanism (i.e., gene expression, translation or protein function). In each section, the internal mechanism is different but the conceptual idea of modulating the mechanism, as a result of environmental signals, is the same.

Black boxes are effective scaffolds in computer science education for moving toward a deeper conceptual understanding (Ben-David Kolikant & Haberman, 2001). This is because by using black boxes in teaching, novices can first understand the functionality of a process and its contribution to the whole mechanism, before coping with the complicated details of the process itself. In the field of biology, a study was conducted with middle-school students who learned biochemistry by first describing a black box and then learning about the mechanism inside the black box. The authors reported that these students asked deeper questions and acquired a more thorough understanding than the control group (Olsher & Dreyfus, 1999). These studies are in line with several related findings in science teaching: first, that centering on the functionality of processes prior to teaching the process itself can establish a deeper understanding of the latter (Liu & Hmelo-Silver, 2009); second, that describing a process as a black box is, in essence, acknowledging a knowledge gap. Such acknowledgement has been shown to be an important step for learning how to fill this gap (Linn et al., 2004). Lastly, it has been claimed that after learning a mechanism and forgetting its details, the relearning of the mechanism is much faster due to "cognitive traces" in the shape of multiple black boxes between central entities (Keil, 2019). Building on this claim, we can assume that having these black boxes prior to actually learning about the details of the mechanism may facilitate learning.

If we wish to use these black-box scaffolds, we should first identify them along the progression. The first milestone in the aforedescribed progression of mechanistic understanding in genetics is acknowledging that there is a causal connection between genes and traits and that this causal connection may be detailed, meaning acknowledging that there is a black box between genes and traits (Fig. 5.1, right). Genes–black box–traits is not a mechanistic explanation but it is a phenomenal model that describes an indirect relationship mediated by a mechanism. One can use this phenomenal model to make predictions (such as in Mendelian genetics) while still recognizing that this is not a full explanation. Hence, the black box may also act as a placeholder and scaffold the progress toward a more detailed mechanistic explanation. In addition, understanding the limits of reasoning using this black box is important in terms of recognizing the limits of this phenomenal model and avoiding conflation with other, more detailed models, as described by Gericke and

Hagberg (2010). For the same reason, acknowledging that there is a black box that links genes to traits might also inhibit views of genetic determinism.

The second milestone in the progression is opening the black box and revealing the mechanism that mediates the causal relationship, i.e., unpacking the mediating entities and activities. As previously mentioned, not all entities and activities should be unpacked. The use of black boxes at this stage may assist in building a whole mechanism without the risk of forming an erroneous direct connection between indirect entities. For example, moving forward from genes–black box–traits to genes–black box–proteins–traits may allow focusing on the central role of proteins without risking the formation of a direct link between genes and proteins, and without the need for a detailed explanation of transcription and translation. In this example, the input for the black box may be a DNA sequence and the output a specific protein structure.

The move from the second mechanistic milestone to a more complex understanding of regulatory mechanisms may be facilitated by introducing a "regulatory black box"—a recognition that sensing mechanisms can regulate each step in the gene-to-trait mechanism without providing the details of these regulatory mechanisms. This should provide the ability to think about regulation by the internal/external environment in terms of conceptual ideas, without the need for a detailed explanation (Fig. 5.1, right). Later on in the progression, since the placeholders already exist, students may more easily understand the details of a specific regulatory mechanism, such as regulation of gene expression, including transcription factors or chromatin remodeling.

Use of these black boxes may facilitate the move between milestones in the progression scale of mechanistic reasoning in genetics, and they may act as placeholders, inhibiting conceptions of genetic determinism.

5.3 Remaining Issues

The black box scaffolds suggested in this chapter are based on findings and theories from science education but are nonetheless theoretical. Understanding the affordances and constraints of the use of black boxes as scaffolds in genetics teaching warrants further empirical testing. Such testing should first analyze how students grasp the gap between the input and output (the black box) and how we should help them understand this gap. This is because if students do not perceive this input–output as a gap, or they perceive it as a gap that cannot be explained (even in the future), this black box cannot act as a scaffold. One possible indication that students do perceive this black box as one that can be opened is their ability to ask questions about how it may function or raise assumptions regarding its possible content (similar to what was done in Olsher & Dreyfus, 1999). These questions and assumptions lay the groundwork for understanding what knowledge is needed to open the black box. Research should also characterize the difficulties students may encounter when reasoning in biology using a black box. Some of these difficulties have been characterized in the field of computer science, but not in biology (Statter & Armoni, 2017).

5.4 Implications for Teaching

The current LP in genetics is wide, targeting more than gene-to-trait mechanisms. It includes many other important ideas, such as mechanisms and probabilities in sexual reproduction (Duncan et al., 2009). This LP was empirically tested and revised accordingly (Shea & Duncan, 2013; Todd & Kenyon, 2015). In the revised version, the LP includes several constructs that are relevant to the discussion in this chapter (see Table 5.1): genes code for proteins (construct B), proteins do the work of cells (construct C1), proteins as mediators between genes and traits (construct C2), and environmental factors interact with genetic mechanisms (construct H).

This description of the constructs from the genetic LP was adapted from Todd and Romine (2018, p. 7). Each construct (B, C1, C2, H) is a description of a "big idea" in genetics and the levels correspond to the levels of understanding this idea. The progression occurs along these levels. Although construct B deals with the connection between genes and proteins, the first progression level describes genes as instructions for the development of traits. This is essentially a black box description which scaffolds the movement from students' prior knowledge about a link between genes and traits toward a more mechanistic description of how genes affect the appearance and development of an organism. The higher levels focus on the mechanisms by which genes code for proteins and do not deal with how proteins affect

Table 5.1 Condensed description of several constructs from the genetic LP which are discussed in the implications

Construct	Level 1	Level 2	Level 3	Level 4	Level 5	Level 6
B—Genes code for proteins	Genes noninformational	Genes are instructions (at the body level)	Genes code for cell entities	Genes code for proteins	Genes translated into proteins	----
C1—Proteins do work of cell	Cells perform function	Proteins are good for your body	Proteins do work of cell	Protein function depends on structure	Protein function and structure depends on amino acids in the protein	----
C2—Proteins connect genes and traits	Change to genes changes traits	Change to genes changes cells	Change to genes changes proteins	Change to genes changes proteins to change traits	Change to genes changes amino acids in proteins	Change to genes changes protein function to change traits
H—The environment interacts with genetic information	Environment can affect organisms	Environment can affect traits or functions	Environment can affect our cells, organs or tissues	Environment can change or mutate things inside cells	Environment can change type and amount of proteins that influence cell function	Environment can change genes which change proteins or gene expression

traits, meaning that only the black box between genes and proteins is opened (explaining how genes code for proteins), but not the one between proteins and traits (no explanation of how protein function affects traits). Construct C2, which deals with proteins as mediators between genes and traits, also begins by establishing a causal relationship between a change in genes and a change in traits. It is only at higher levels that the construct further describes the change in genes as affecting protein structure and consequently, its function. Again, a black box is established between genes and traits, then proteins are described as mediators and only the black box between genes and proteins (and not proteins and traits) is opened at the higher levels.

The construct that opens the black box between proteins and traits is construct C1, dealing with how protein function may lead to traits. As already suggested, the centrality of proteins as mediators arrives mainly via an understanding of their contribution to the formation of traits (namely their function) and less via knowledge about genes as coding for proteins. Thus, construct C1 is essential for gaining an understanding of constructs B and C2. In addition, the relevance of construct C1 to genetics is understood only when higher levels are reached in constructs B and C2. And indeed, such contingencies were empirically detected (Shea & Duncan, 2013). Hence it is recommended not to teach all constructs in parallel but to begin with lower levels in B and C2 which describe the black box between genes and traits (genes affect traits via a mechanism that is not taught for now) and adding C1 before moving to higher levels in these constructs. In addition, the connection between these constructs should be made explicit.

Another idea mentioned in the LP, which is relevant for the discussion here, is the construct dealing with environmental factors that interact with genetic mechanisms (construct H). According to the recommendations here, this construct should be elaborated to include the idea of sensing the environment and should be tightly connected to regulation mechanisms, such as regulation of gene expression (which is suggested in a completely different construct).

In conclusion, this chapter identifies three milestones along the development of mechanistic understanding and mechanistic reasoning in genetics, and suggests black boxes which may be used as scaffolds for supporting this development. These black boxes seem to already exist, in some parts of the current genetics LP, but not explicitly or consistently.

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Chapter 6
How Might Authentic Scientific Experiences Promote an Understanding of Genetics in High School?

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6.1 The Problem

Research in genetics is dynamic and complex, and knowledge and practices are constantly being updated. There is a discrepancy between the dynamic way in which genetics knowledge is gained and practiced, and the way in which it is taught in schools; high-school students rarely have access to current genetic research, and this may limit their understanding of genetics. The first section of this chapter focuses on this problem and mainly on the fact that genetics is often taught in an expository and oversimplified manner, which is seldom updated.

The second section suggests how practicing authentic scientific experiences in high schools can mitigate these problems. It defines what authentic scientific experiences are, and reviews the different ways in which they have been practiced in genetics teaching in high schools. Due to paucity of relevant published research, in this review we make no attempt to synthesize or perform a meta-analysis of research on authentic experiences in genetics; rather, we seek to promote awareness of this approach to genetics teaching. We go on to discuss the remaining issues, mainly the difficulties in practicing authentic scientific experiences in genetics teaching in high schools.

6.2 Current Knowledge About the Problem

Understanding genetic concepts and their relations to various biological phenomena has implications for personal and public life. These concepts appear on the news, and direct-to-consumer genetic services (e.g., genetic testing, ancestry for humans

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and animals) are promoted and available to all (Redfield, 2012; Stern & Kampourakis, 2017). Studies have shown that better scientific knowledge may lead to a higher level of critical thinking about the options with which one is confronted, such as whether to perform genetic tests during pregnancy (Siani & Ben-Zvi Assaraf, 2015). Therefore, understanding genetics is crucial for future citizens.

However, beyond understanding the content knowledge, a comprehensive understanding of genetics includes its applications for society and in current scientific research (Boerwinkel et al., 2017; McElhinny et al., 2014). Students should be informed about how genetics knowledge is gained, the current status of genetic research, the technological advances, and its biomedical applications and social relevance (Kampourakis et al., 2014). While students do not need to learn in detail the comprehensive scientific knowledge and methods used by geneticists, it is believed that the students should understand their actual potential, their current limitations and the uncertainty of the respective conclusions (Stern & Kampourakis, 2017).

One obstacle to developing a comprehensive understanding of genetics is the discordance between the way genetics knowledge is gained and the way in which it is usually taught. In the biological sciences, the approach of integrating research areas has emerged: concepts and methods at the molecular level, functional genomics and bioinformatics are integrated to offer opportunities to investigate fundamental questions. In contrast, biology textbooks tend to retain their traditional subject matter selection and sequence. Most of them include chapters designed as separate units and lack integration activities and cross-references between the chapters. Thus, genetics "topics" are often isolated from other biological contents and from each other (e.g., meiosis is typically isolated from heredity, and they are both isolated from evolution). As a result, students often have fragmentary knowledge and lack a coherent idea of genetics. This fragmentation also contributes to the abstract nature of genetics (reviewed in Knippels, 2002; Knippels et al., 2005).

Moreover, although attempts are constantly being made to improve learning materials and textbooks, research in genetics is advancing rapidly, and keeping up with the latest findings can be challenging. As a result, textbooks are not always updated with the most current knowledge (Lombard & Weiss, 2018; Stern & Kampourakis, 2017). Although not all current knowledge should be taught in schools, lagging behind current research may lead to teaching outdated knowledge that is detached from current understanding, lacks the ability to explain the natural world, and may inhibit students' connected understanding of scientific ideas and the world (Lombard & Weiss, 2018).

In addition, academic knowledge goes through a process of transposition, meant to suit pedagogical needs. This process is necessary to some extent, but may also lead to oversimplification of knowledge. For example, Mendelian genetics is often reduced to a deterministic model, where heredity is reduced to dominant/recessive/codominant alleles, eyes are either blue or brown, and practice involves the use of Punnett squares (Lombard & Weiss, 2018). This common presentation of genetics in schools teaches students that genes "control" or "code for" individual characteristics, which is a misrepresentation of the current scientific knowledge about the effects of genetic material. This may lead to students' (and some teachers')

explanations of the origin of traits as the direct products of genes, rather than as the outcome of the interaction of genetic, environmental, and behavioral factors with molecular networks, or a result of evolutionary and developmental processes (Kampourakis et al., 2014, 2016). This common reduction not only leads to failure in conveying modern genetics accurately, but it also gives students false impressions about real-world issues such as the inheritance of diseases or concept of race (Donovan, 2014; Dougherty et al., 2011). Despite these problems, most teachers still rely on textbooks for teaching and usually do not question their validity (Lombard & Weiss, 2018; Stern & Kampourakis, 2017).

In accordance with this reliance on textbooks and curricula and instruction that are testing-oriented, genetics knowledge is often communicated to the students as factual information in an expository manner that does not reflect the complexity, reliance on evidence, uncertainty, and limitations in the way in which this knowledge was gained (Yarden, 2009). Exposure to these attributes is crucial for students to develop an understanding of the tentative nature of some knowledge claims in genetics, to evaluate current and outdated ideas, and to develop genetic literacy (Cartier & Stewart, 2000; Lombard & Weiss, 2018). Engaging students in tasks that reflect the attributes of authentic science can give them the opportunity to develop a contextualized and deep understanding of scientific knowledge, and in turn apply it meaningfully in real-world settings (Chinn & Malhotra, 2002; Edelson, 1998; Machluf et al., 2017).

Giving students access to the recent tools, practices and knowledge in genetics, and to the process of scientific research, may compensate for these pitfalls and enhance genetics learning. Using examples, the next section outlines how high-school students' understanding of genetics may be enhanced by exposing them to authentic scientific tools and the accompanying practices, i.e., by engaging them in authentic scientific experiences.

6.2.1 Authentic Scientific Experiences as a Suggested Solution

What is Authentic Science?

The need to refrain from teaching science in a factual expository manner and to create authentic science learning experiences has been extensively discussed in the literature (e.g, Chiappetta, 2008; Chinn & Malhotra, 2002; Edelson, 1998; Yarden et al., 2009). Buxton (2006) suggested three perspectives of authentic science education: (1) canonical—science education is as similar as possible to the way science is practiced by scientists; (2) youth-centered—students use science and technology to solve problems that are of interest to them; and (3) contextual—chosen aspects of the canonical and youth-centered perspectives are combined, in terms of both questions asked and methods used. Here we refer to authenticity as interpreted in the canonical perspective. Canonical authentic science is aligned with the Western scientific canon and with the US National Science Education Standards (National

Research Council, 1996). In the new US framework and standards for K–12 Science Education (National Research Council, 2012; NGSS Lead States, 2013), it is reflected by the scientific practices: "the major practices that scientists employ as they investigate and build models and theories about the world" (National Research Council, 2012, p. 30). The notion of teaching science in a way that resembles scientific research is also prevalent in policy documents worldwide (e.g., European Commission, 2011; House of Commons—Science and Technology Committee, 2011) and has been a common goal among educational reformers for several centuries, often referred to as "inquiry" (reviewed in Chiappetta, 2008; Edelson, 1998). Nevertheless, the term "scientific inquiry" has been interpreted differently by educators, presenting a broad array of meanings and strategies (National Research Council, 2012; Surr et al., 2016). According to Chinn and Malhotra (2002), there is a continuum ranging from authentic scientific research as carried out by scientists to simple inquiry tasks—confirmatory "cookbook" experiments carried out in schools. Along this continuum, while inquiry learning in some schools is closer to authentic science, most inquiry tasks commonly used in schools are too simplistic and are not similar enough to authentic research. They do not reflect the core attributes of authentic science, along with its epistemology, reasoning, and complexity (Chinn & Malhotra, 2002). Today, many classroom inquiry tasks still focus on simple inquiry (Peffer et al., 2015), thus delivering scientific knowledge expositorily, and the need for more authenticity remains. But what is considered authentic practice in the field of genetics learning?

The Characteristics of Authentic Science

For the purpose of this chapter, we draw from Edelson (1998) who claimed that authentic scientific practices go beyond scientific knowledge, tools and techniques, which sometimes become the focus when translating scientific practices into educational settings. He suggested several key features of scientific practices, which fall into three categories: (1) *tools and techniques*—the practice of science includes a set of tools and techniques that have been developed and refined throughout the history of the field and allow scientists to ask and investigate a range of questions. Sharing these practices across a community of scientists facilitates communication, by establishing a shared context; (2) *attitudes*—scientific practice involves uncertainty and commitment. Accordingly, techniques and results are subject to continual reexamination, and the scientists are committed to the question that they are attempting to resolve; and (3) *social interaction*—sharing results, concerns and questions with the community. This communication requires identification of evidence to support scientific reasoning and argumentation (Edelson, 1998). Accordingly, here we adopted not only the use of tools and techniques that are used by geneticists, but also "the interest of engaging students in posing questions, designing their own paths to solve them, collecting evidence, evaluating claims against evidence, building arguments in a dialogic setting" (Yarden et al., 2009).

The Applications and Contributions of Authentic Science Experiences to Genetics Learning

Applications and benefits of authentic scientific experiences for genetics learning are demonstrated through the few examples of such experiences that have been implemented in high schools. While several authentic science activities in genetics have been described for the college undergraduate level (e.g., Elwess et al., 2017; Hester et al., 2018), these are scarce for the high-school level. For this chapter, we sought research published between the years 2000 and 2020 that complied with the following criteria: (1) implementation at the high-school level; (2) carried out in the school itself (and not, for example, in research laboratories or museums) using available apparatuses, thus affording accessibility to distant communities; (3) designed with the explicit aim to create an authentic scientific experience; and (4) representing the sophisticated concept of authenticity described above. For each example, the key features of scientific practices suggested by Edelson (1998) are stated in parentheses, to demonstrate how authenticity was manifested. Five examples that represent different types of authentic scientific practices were selected: (1) hands-on inquiry, (2) student–teacher–scientist partnership (STSP), (3) design-based learning (DBL), (4) web-based environment, and (5) learning with adapted primary literature (APL). These examples also represent different research contexts in which genetic concepts are rooted, relating genetics to other subjects in biology (evolution, plant science, biotechnology, bioinformatics, and developmental biology, respectively).

I. **Hands-on inquiry—remote laboratory evolution.** Hands-on science involves practical and concrete activities in which students carry out experiments (Triona & Klahr, 2007). To afford high-school students access to cutting-edge technologies, Dahan et al. (2019) developed a program consisting of a remote laboratory evolution experiment. Students investigated the emergence of antibiotic-resistant bacteria in real time, while using a cutting-edge liquid-handling robotic platform that users can instruct remotely using a standard internet connection and Google Sheets. The robotic system itself was in an active research laboratory in the US, while the students (150 students, 10th and 11th grades) participated simultaneously from their schools in Israel or in the US. Students also experienced sequencing and bioinformatics *(tools and techniques)*.

Throughout the program, the teachers guided their students, with the support of the scientist who runs the laboratory accommodating the robotic system. First, students conducted introductory hands-on bacterial growth and antibiotic-resistance experiments in their schools. The authentic inquiry project itself included a 10-day laboratory evolution experiment aimed at investigating which antibiotic regimens would result in multidrug resistance in *Escherichia coli*. Each group of students designed drug regimens for their assigned bacterial cultures. They uploaded their selections to Google Sheets, thus operating the robot that applied the selected antibiotics to the cultures. An absorbance-reader monitored the optical densities and the results were posted daily on the program's website, allowing the students to make

informed decisions for the next day—whether to pursue their original regimen or change it. After 10 days, the growth of each culture was compared to that of the ancestral population. Chosen genes of the resistant bacteria were sequenced and the students were guided in identifying the mutations via sequence alignments. Students discussed, with their teachers and the scientist, how these mutations could be linked to different mechanisms of antibiotic resistance. Authenticity was also manifested in the dynamic, complex, exploratory and open-ended nature of the investigation. The students analyzed results involving multiple factors, they constantly had to make decisions in real time, and there were no expected outcomes (*attitudes*). They communicated their ideas and results within and between their groups, and with the teacher and the scientist (*social interaction*).

Pre- and post-program questionnaires assessed students' understanding of evolution and antibiotic resistance. The students provided detailed explanations for the evolutionary phenomena, with reference to the mechanisms underlying them, and used genetic terminology relating to mutations and the genome. Still, many students expressed the wrong idea that mutations never disappear (Dorfman et al., in preparation). Nevertheless, these findings indicated that experiencing authentic practices may have assisted students in linking changes in the phenotype to changes in the genotype and may have promoted students' understanding of genetics principles and their relations to biological phenomena such as evolution.

II. **STSP—students' contribution to characterizing gene function in *Arabidopsis thaliana.*** STSP is a strategy that allows students and teachers to engage in actual scientific research, while benefiting scientists by providing additional resources for data collection (Houseal et al., 2014). In The Partnership for Research and Education in Plants (PREP), a program developed by Dolan et al. (2008), plant scientists and teachers guided high-school students in investigating gene function in *Arabidopsis thaliana*. Scientists have been trying to determine the function of all of the genes in *Arabidopsis thaliana* by disabling genes, but they do not always see changes in the phenotype. One suggested way to reveal hidden phenotypes was to put the plants under different environmental stresses (Somerville & Dangl, 2000), which requires many hours of work. The partnership with high schools therefore gave the scientists the necessary extra help in collecting data that would otherwise be difficult to obtain.

The scientists provided wild-type seeds and seeds in which the gene that they were studying had been disabled. For a period of 8 weeks, using online distant communication tools, the scientist guided the students together with the teacher in the process of designing and conducting their own original investigations. These investigations were aimed at studying the phenotypic differences between wild-type and mutant plants growing under different environmental stresses, thus helping to characterize the functions of the disabled genes (*tools and techniques*). These functions were unknown to the students and to the scientists. Hence, a unique facet of authenticity was exhibited: students took part in producing data of interest to the scientific community, and some of these data were included in scientific publications (*attitudes, social interaction*). Students were also exposed to messy and inconclusive

data and to the complexity of "real-world" genetics: they were challenged with the idea that a plant may look like the wild-type although it has a disabled gene, and that some phenotypes are the results of interactions between genes and environment (Dolan et al., 2008). A comparison between pre- and post-tests (covering genetics, plant biology, scientific inquiry, and experimental design) showed increased knowledge among students who participated in the program (n = 595). Over the years, PREP has involved over 15,000 high-school students and 30 scientists. Simpler versions of PREP (e.g., that involve known phenotypes or in which genotype is the sole independent variable) were later developed as an instructional scaffold to engage "average or low achievers" in authentic scientific experiences (Dolan, personal communication, November 21, 2019).

III. **DBL—genetic engineering project.** In DBL, students learn content while designing an object or prototype (Ellefson et al., 2008). The design process involves iterative exploration and refinement processes (Bell et al., 2013), which may allow students to learn genetic concepts, while realizing the way this knowledge is applied in genetic research. Aimed at teaching gene expression, Ellefson et al. (2008) created an 8-week DBL unit in which students designed and created genetically engineered *E. coli* bacteria to meet a need in their own lives (e.g., a tanning lotion containing bacteria that turn blue when the skin is about to burn). Through a series of investigations, discussions, and design modifications, students learned about the molecular processes and structures involved in gene expression, and how these are dependent upon different environmental variables. First, students tested the influence of environmental factors on bacterial growth and discussed how the environment and DNA interact, including possible changes to the DNA that can be passed on to the next generation. Then, the students considered how they could get the bacteria to exhibit new traits. Students introduced new genes via plasmids, which caused the bacteria to express new traits, and then tested the success of their bacterial transformations, desired trait expression and whether the traits were carried into the next generation (*tools and techniques*). Since many of the students failed on their first try, they had to discuss possible reasons for their failure and try again, testing different approaches (*attitudes*). This required them to learn about the physical structure of the DNA, transcription and translation. In addition to in-group discussions, students presented their ideas and received feedback from their teachers and peers during mini-symposia, simulating the way in which scientists and engineers get feedback (*social interaction*).

During the first year of implementation, over 500 urban and rural high-school students participated in the unit. Increased performance between pre-unit and post-unit questionnaires (n = 89), containing short-answer essay questions from the gene-expression database of the National Assessment of Educational Progress (NAEP), indicated that the students had gained a good understanding of genetics and gene expression during this unit and displayed a good ability to transfer what they had learned to new situations.

IV. **Web-based environment—bioinformatics tools.** Web-based environments are gaining popularity in science education, as they allow for integrating various sources of information, such as text, static and dynamic visualizations, and scientific web-based tools and databases. Students can work at their own pace and receive immediate feedback, and teachers can receive information regarding their students' progress (Petra et al., 2016). Web-based technologies are also an integral tool for genetic research today, enabling scientists to search a wide array of databases (e.g., genes, proteins, nucleic acid sequences) and access analytical and modeling tools (Waight & Abd-El-Khalick, 2011). Therefore, these tools are good candidates for creating authentic scientific experiences for students in the field of genetics. Despite the desire to use the technology in the same way as scientists, these tools need to be adapted to high-school cognitive level and knowledge (Edelson & Reiser, 2006). While web-based research simulations in genetics have been found effective in expanding and refining genetics knowledge (e.g., Gelbart et al., 2009; Waight & Abd-El-Khalick, 2007), use of state-of-the-art simulations requires keeping pace with the actual bioinformatics tools used by scientists, which are constantly being updated. Therefore, web-based learning environments were designed in which students could approach real-world problems while acquiring and applying modern scientific practices and using the actual bioinformatics tools, not adapted or simulated ones. For example, Machluf and Yarden (2013) designed a web-based environment in which students used diverse bioinformatics tools and databases that are fundamental and widely used by scientists—Entrez, BlastN, BlastP, ClustalW, ORF Finder, Primer3Plus, PROSITE, and Jmol (*tools and techniques*). These tools are freely available on the web and the databases are ordinarily updated. Thus, in addition to the technology itself, students are exposed to key practices of the scientific community, such as sharing resources, and the dynamic nature of scientific knowledge. The activities in the learning environment focus on authentic investigations aimed at improving human life quality and expectancy.

Throughout the inquiry process, which was embedded in questions and assignments, the students experience different scientific practices. They are required to coordinate different types of knowledge from genetics and biotechnology, to reason scientifically, to make decisions following a strategic plan, and to evaluate and justify them (*attitudes, social interaction*). To evaluate the activity's contribution, forty-four 11th-grade students who went through a 5-hour-long bioinformatics experience were asked to define, schematically draw and explain genetic terms which were central to the 11th-grade curriculum but were not the focus of the activities, such as gene, open-reading frame, coding sequence, exon, and promoter. Then, students encountered a research problem that differed from those appearing in their textbooks or in the learning environment. They were asked to design a study using any of the methodologies and techniques with which they were familiar (biological techniques and/or bioinformatics tools), justify it, and make predictions. Following

the bioinformatics activity, increases were observed in average grades, and students' designs were based mainly on a bioinformatics approach. This indicated that the students had acquired genetics content knowledge and perceived bioinformatics knowledge as useful, and that they could apply it in various research contexts. It might also signify adoption of authentic scientific practices (Machluf & Yarden, 2013; Machluf et al., 2017).

V. **Learning with APL.** APL is a unique text genre developed to enable the use of primary scientific literature (PSL) to learn science in high school, while exposing students to authentic scientific language and discourse practices. The adaptation process maintains the canonical structure of the PSL and the original results, but suits them to the cognitive level of high-school students (Yarden, 2009; Yarden et al., 2015). Use of APL gives high-school students access to an inseparable part of scientific research—communicating knowledge—while reading and writing in the discipline's unique language (*social interaction*). Some consider experiencing these aspects of science as a form of inquiry (Phillips & Norris, 2009). Learning through APL may be a way to experience the uncertainty of scientific discoveries and the fact that scientific results are subject to continual examination and refinement (*attitudes*) (Yarden et al., 2015). To introduce the process of research and the way knowledge is produced, a curriculum unit presenting developmental genetics through several APL articles (*tools and techniques*) was developed and implemented by Brill and Yarden (2003).

Students in the 11th and 12th grades who learned using the developmental genetics APL curriculum (n = 69) and a control group who studied the common genetics curriculum (n = 33) were asked to raise questions regarding the materials they had learned. The APL group raised more higher-order questions and more unique questions. This suggests that higher-level and more diverse thinking about genetics may be elicited by the nature of research papers in which the reader is exposed to the entire research process (Brill & Yarden, 2003).

Additional Advantages—Affective Gains

In the above cases, in addition to cognitive gains, affective gains were reported. Students enjoyed the activities and found them interesting and relevant, which made them more engaged. Students attributed their engagement and interest to the contemporary nature of the contents, and to feeling "like real scientists"—involved with real scientific practices and processes, solving real problems, sometimes with unknown outcomes. Students also felt ownership of their projects. Often students stressed the contrast between the authenticity of the activity or genre and the traditional curricula (Dahan et al., 2019; Dolan et al., 2008; Ellefson et al., 2008; Machluf et al., 2017). Note, however, that some students felt that this type of learning was too demanding (Dahan et al., 2019). These affective gains contribute to genetics learning because emotions influence learning—they influence cognitive processes, information processing, storage and retrieval. Memory is affected by the emotions one

experiences during learning (reviewed in Kim & Pekrun, 2014). In addition, genetics education aims to equip students with knowledge that they will be able to apply as citizens. Developing a personal interest in science and helping learners identify with science as an endeavor are important for reaching this goal (National Research Council, 2012). Thus, positive affect is an important outcome of authentic scientific experiences related to genetics.

Summary

The projects presented here demonstrate how different types of authentic scientific experiences can promote an understanding of genetics and of the relations between genotype and phenotype, and how these relations are exhibited in actual complex biological phenomena, as well as higher-level question asking, which goes beyond declarative knowledge. Although different in character, these projects had several characteristics in common, which may have contributed to their success.

First, the authentic genetics practices were adapted to balance authenticity with students' scientific knowledge, capabilities and needs, and to reduce their complexity. The practices used by trained scientists are typically complicated for non-experts, and this might create a cognitive load that could prevent efficient learning. Adapting the practices, while retaining their key elements, may provide the necessary scaffold for students. Adaptation of the tools should be accompanied by adaptation of the tasks that structure the learning and the interactions between students and with teachers (Edelson & Reiser, 2006). From a cultural perspective, one should also consider that technologies which were designed in response to the values and needs of one culture are not always suitable for the purposes of another (Waight & Abd-El-Khalick, 2018). The content knowledge itself—in this case genetics knowledge—should be adapted to the institutional, social and cognitive constraints of high school, so that it can be understood (Chevallard, 2007 as cited in Lombard & Weiss, 2018). In the examples described above, the content-knowledge adaptations were performed while attending to the most accurate and up-to-date genetics knowledge.

Second, genetic concepts were not taught explicitly as the topic of focus, but rather as part of a wider task and subject. These tasks were also related to authentic challenges confronting current scientific research. Thus, four main benefits were achieved.

1. Genetics was put into context and was taught in close association with other topics (e.g., evolution, plant science, human body, and developmental biology). Students were required to link knowledge from different scientific topics, to recall prior content knowledge, and to reason scientifically. The separation between genetic concepts, from each other and from other biological concepts, which is common in curricula and textbooks, is a source of students' difficulties in understanding genetics. Making the connection between the topics and helping students relate genetics tasks with concrete biological phenomena may reduce the abstract nature of genetics and thus reduce students' difficulties in

understanding genetics (Knippels, 2002; Knippels et al., 2005). This is in line with learning theories that see understanding as evolving while students are engaged in thinking and inquiry in contexts that make sense to them, rather than evolving from fragmented knowledge resulting from complex ideas being broken down into smaller parts (reviewed in Zohar & Nemet, 2002).

2. The genetic concepts that students learned reflected current and accurate genetics knowledge. Despite the need for adaptation to students' cognitive level and prior knowledge, engaging in authentic challenges required current and accurate genetics knowledge. Students confronted complex concepts that go beyond the oversimplified ideas commonly discussed in class (Dougherty et al., 2011; Lombard & Weiss, 2018). Understanding only the oversimplified ideas may limit the impact on helping students understand the genetics-related issues they will encounter as citizens. Learning about complex concepts is necessary because most of the genetics information to which citizens are exposed through the media deals with complex diseases and traits. Mutations, gene regulation and non-Mendelian patterns of inheritance should therefore be taught to students and find their way into curricula and standards (Dougherty et al., 2011; Lanie et al., 2004). Although the call for changes in genetics curricula and standards has been acknowledged (e.g., Dougherty et al., 2011; McElhinny et al., 2014; Redfield, 2012), these changes take time. Engagement in authentic experiences may provide a way to teach students these concepts and to explore high-school students' abilities to understand complex ideas.
3. Using the context of current scientific challenges enabled connecting curricular genetics content knowledge with issues faced by scientific research and society. Making the connection between content knowledge and its applications, while delivering genetic concepts through authentic experiences, promoted students' understanding of these concepts in various grade levels (e.g., Shuda et al., 2016), as evidenced by the examples presented above. Encountering genetics applications and their relevance to society is necessary for a comprehensive understanding of genetics (Boerwinkel et al., 2017; McElhinny et al., 2014). This, in turn, is necessary for students as citizens when they need to make informed decisions on genetics-related issues. For this, they need to have both the content knowledge and the understanding of its applicability, limitations and relevance to their lives (Stern & Kampourakis, 2017).
4. Students were given the responsibility to make decisions, while they were exposed to the complex data and the dynamic, uncertain nature of genetics investigations, thereby developing students' understanding of the limitations of genetic technologies and research. This may help students better evaluate claims that rely on conclusions from research and the power of some genetic technologies that are discussed in the media (Stern & Kampourakis, 2017). It may also improve students' understanding of scientists' work, as well as their attitudes toward science and scientists (e.g., Houseal et al., 2014). Being involved in decision-making through the inquiry process, especially an open-ended and dynamic process, also encourages the development of a sense of agency and

ownership, and students' identification with science and its endeavor (Kapon, 2016; Sadeh & Zion, 2009). These attitudes toward science, and in this case genetics, are important in giving students the desire to understand the science concepts required to make informed decisions (Fortus, 2014). In the examples presented above, the complex dynamic inquiry process was described by students as a meaningful experience, in contrast to the simple, predictable inquiry usually encountered in school. Similar descriptions have been reported in other studies regarding dynamic inquiry and authentic scientific experiences (e.g., Kapon, 2016; Sadeh & Zion, 2009). In some cases, these were reported about a decade after the experience took place, where the participants remembered the content knowledge or concepts acquired during the process (Dorfman et al., 2020). This was attributed to the affective aspects together with the flexibility, judgment, and critical thinking that are expressed in situations involving change and uncertainty (Zion et al., 2004), and indicates that such an experience indeed contributes to science literacy in adulthood.

Finally, these activities were developed in collaborations between science educators (researchers and teachers) and life-science researchers. This will be further discussed in the next section.

6.3 Remaining Issues

6.3.1 The Challenges in Implementing Authentic Experiences in Schools

Engaging students in authentic practices in genetics is challenging. Current genetic research requires access to knowledge, supplies and equipment that are not commonly accessible to high schools. However, technological advances in recent years, such as the internet and free web tools, are changing this. Still, students are rarely exposed to these practices, and through them to the way genetics knowledge is gained, applied and communicated by scientists (Dahan et al., 2019; Dolan et al., 2008; Redfield, 2012). The authentic experiences described above have overcome some of the barriers to implementing authentic practices in schools, such as limited resources and safety concerns (Chinn & Malhotra, 2002; Hossain et al., 2016; Peffer et al., 2015). Nevertheless, there are more profound obstacles to applying authentic scientific practices and experiences, especially if we wish to make them sustainable. Here, we will discuss two issues that should be considered in attempting to overcome these obstacles—the involvement of scientists in the project and the tension between authentic science and school science.

6.3.2 *Life Scientists' Involvement in Authentic Scientific Experiences for High-School Students*

When it comes to designing the implementation of authentic scientific experiences for high-school students, several researchers stress the importance of involving scientists in the relevant content areas in the process (e.g., Edelson, 1998; Waight & Abd-El-Khalick, 2007). In the design stage, scientists can contribute by sharing their first-hand knowledge of the content and of the practices of their expertise. In addition to the planning phase, implementing authentic practices in class and helping students handle their complexity can be challenging, as some teachers may have limited first-hand experience with scientific practices, or with incorporating such practices into their instruction (Edelson & Reiser, 2006). In addition, some experienced teachers' knowledge of the techniques used for genetics-related research may be outdated, because of the rapid progress in the field (Ellefson et al., 2008). Relying on their experience and profound understanding of the content knowledge, scientists can assist students in task structuring, provide explanations while using scientific terminology, and offer methodological suggestions. They can assist students in questioning hypotheses or interpretations of the data, and give them feedback on their progress (Peker & Dolan, 2014). The examples presented above had varied degrees of scientist involvement. While web-based tools and APL require limited scientist involvement, usually in the design stage, hands-on inquiry, DBL and STSP demand a great deal of the scientist's time, effort and resources.

Some universities and research institutes encourage scientists' engagement with the public, and more and more granting agencies are requiring scientists to participate in K–12 outreach (Peker & Dolan, 2014). Still, many universities discourage these efforts, instead requiring their scientists to focus on their main area of scientific research (Ecklund et al., 2012). Scaling up the projects or making them sustainable requires more effort to attract scientists to take part in science education initiatives. Another possible solution might be to reduce the scientists' role by relying more on APL or web-based tools.

6.3.3 *The Tension Between Authentic Science and School Science*

Leading authentic inquiry tasks require teachers' mastery of authentic science practices, as well as their willingness to enact a model of instruction that attends to issues such as taking inquiry outside, and providing the time and resources to engage in problem posing and problem solving, and attends to student preferences (Buxton, 2006; Chinn & Malhotra, 2002). Teachers sometimes struggle with making the transition from traditionally structured and controlled classrooms to less ordered experiences that rely on discussions with students. Students, too, may be unfamiliar with the nontraditional roles that they are required to adopt, and sometimes hold to the

established teacher and student roles (Grady et al., 2010). These changes also require support from the working environment. The organizational culture and structures must conform with these ways of working, and the collaboration should be consciously created in the teachers' working environment. Unfortunately, in many cases, school administrators or other members of the school community do not support these kinds of approaches, which makes it difficult for teachers to continue such a model of teaching (Buxton, 2006; Kunnari & Ilomäki, 2016). This may be one of the reasons that sometimes, while researchers try to introduce authentic tools such as educational technologies, teachers remain aligned with the realities of K–12 science classrooms, using structured problems and prescribed procedures (Waight & Abd-El-Khalick, 2011).

Kapon et al. (2018) noted tension between common school science and authentic science, as these often have different goals. For example, having the time to engage in the practice of doing science conflicts with the pressure to cover the required number of topics during the school year (Kapon et al., 2018). Furthermore, whereas authentic science aspires to constant progress and innovation, schools are self-stabilizing systems, which need extensive time to adopt innovations and to integrate advanced technologies and practices into science classes (Waight & Abd-El-Khalick, 2011).

Calls to adapt genetics curricula to modern genetics practices and to current approaches to science education are already being heard (e.g., Redfield, 2012). Further effort should be made to incorporate authentic genetics experiences in schools, to increase teachers' understanding of current genetic research, and to involve genetics researchers in those efforts.

6.4 Implications for Teaching

The information presented in this chapter demonstrated the contribution of authentic scientific experiences to genetics understanding of high-school students, along with the difficulties in implementing them. It also demonstrated that there is a variety of ways to carry out such experiences. This may ease teachers' ability and willingness to lead such experiences in their classes. For example, a teacher who wishes to bring authentic experiences in genetics into her biology class has many ways of doing so, according to the needs of her class—the students' previous knowledge, cognitive abilities, interests, and factors such as available time, number of students and accessible equipment. The teacher can also incorporate the activities in different parts of the curriculum, thus relating them not only to various aspects of genetics but to different topics learned in biology class. This may assist in creating a more concrete and coherent picture of the content knowledge for the students (reviewed in Knippels, 2002; Knippels et al., 2005).

As of now, most of the authentic experiences in genetics were reported at the undergraduate level, where they are more prevalent (e.g., Elwess et al., 2017; Hester et al., 2018). This chapter implies that such approaches can be suitable and beneficial for high-school students and thus, may encourage teachers and decision makers to incorporate them in genetics teaching earlier than in college. Teachers can adapt existing undergraduate activities to the level of their own class. While planning or adapting activities, it should be noticed that authenticity would be manifested not only in using authentic tools and techniques, but rather in relating to the more comprehensive view of scientific practices presented above. Teachers should notice that they (a) reflect on the complexity, uncertainty and dynamics of scientific research; (b) create appropriate scaffoldings that would allow their students to understand the complexity that lies in authentic experiences; (c) create appropriate context for the genetics content knowledge by introducing it as part of a wider task and topic, preferably relating to issues faced by the scientific community and the society at large; and (d) give their students the responsibility of decision making.

The science education community may assist teachers in adapting existing programs, or creating their own, through partnerships, teacher professional-development courses, and creating connections between them and geneticists.

6.4.1 Providing More Evidence for the Benefits of Authentic Inquiry to Genetics Learning

Critics of inquiry-based learning, in which students are asked to discover the fundamental and well-known principles of science by modeling the investigatory activities of professional researchers, argue that it is highly time-consuming and if there are conceptual gains, they are too little to compensate for the loss of time (Kirschner et al., 2006). However, when examining the entire spectrum of learning, including nonconceptual aspects, the benefits of authentic scientific experiences are more salient. There are learning gains that cannot be achieved in a formal classroom setting, such as the development of agency, ownership, interest, and passion with regard to science (Kapon, 2016). Nevertheless, although we presented several examples showing the benefits of this approach when it comes to genetics learning, more evidence is still needed. As already noted, there are only a few published papers regarding the benefits of authentic scientific experiences to genetics learning, and we realize that some of the evidence presented here may not be strong enough. Therefore, more research into the applications and implications of authentic science experiences in genetics education is needed. Further research may allow us to overcome some of the aforementioned challenges, and to study the best practices for harnessing the full potential of authentic scientific experiences in enhancing students' understanding of genetics.

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Part III
Reflecting Upon the Relationship Between Genetics Learning and Related Conceptions and Beliefs

Chapter 7
Is Belief in Genetic Determinism Similar Across Countries and Traits?

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7.1 The Problem

Genetic determinism—the ascription of more causal power to genes in the formation of traits than scientific knowledge supports—is thought to be widespread in many cultures and countries (Nelkin & Lindee, 2004). Belief in genetic determinism (BGD[1]) is an important educational problem, not only because it is at odds with scientific knowledge, but also because it may be linked to societal problems such as prejudicial assumptions and stereotypes contributing to unsound social decision-making (Keller, 2005). For example, BGD has been associated with ascribing

[1] The acronym BGD, which was coined by Keller (2005), will be used in this chapter.

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excessive weight to genetic testing in decisions concerning employment or insurance, and with fostering intolerant attitudes toward certain groups of people based on categorizations of gender, race, sexual orientation, etc. Genetic explanations have the potential to be used in nature-based rationales for the unequal treatment of marginalized members of society. These rationales involve a naturalistic fallacy, the conviction that what is 'natural' (in this case, genetically predetermined) is fundamentally good or right (Nelkin & Lindee, 2004). The problems associated with BGD motivate further exploration of how common it is, who is most at risk, and how it might be mitigated through genetics education (Jimenez-Aleixandre, 2014). Ascribing excessive power to genes in the formation of traits is not a normative biological perspective and it is therefore an important issue to address in genetics education, as previously argued (e.g., Gericke & El-Hani, 2018).

Genetic determinism is linked to essentialist reasoning, which can be understood as the view that every entity, including biological traits, contains an immutable underlying essence that predicts similarities between members of a group (Gelman, 2003). Genetic determinism can be regarded as the biological component of essentialism (Keller, 2005), but it is generally considered to be a lay concept deserving independent and focused attention. As Dar-Nimrod and Heine (2011) argue, essentialist thinking can be reinforced by a superficial understanding of genetics, in which genes take the role of concrete placeholders for essentialist ideas. Such an understanding of genetics tends to inaccurately attribute an overactive, primary, or even exclusive determining power to the gene. However, recent developments in genomics and epigenetics have reinforced the notion of gene action as probabilistic and mutually interdependent with the environment, but this is not taught in many classrooms (Gericke et al., 2014, see also Gericke's Chap. 2 on epigenetics in this volume). Indeed, studies have shown that students leaving education at both high school and university levels often hold a genetic deterministic conception (e.g., Lewis & Kattmann, 2004; Jamieson & Radick, 2017), and it is therefore important for educators to know how widespread this conception is.

In this chapter, we examine whether BGD is a widespread problem by investigating its occurrence in university students from three different countries, and we explore the prevalence of BGD across different kinds of traits (biological and social).

7.2 Current Knowledge About the Problem

This section explores the historical roots of genetic determinism and then discusses how the concept can be defined and measured.

7.2.1 The Roots of Genetic Determinism

The philosophical idea of determinism dates back at least to the ancient Greeks, among whom we find the idea that everything that happens, including human actions, is completely determined by previous events or entities. This idea is related to a sort of fatalistic thinking found in a variety of religious worldviews, for instance, in Christian and Hindu cultures (Young et al., 2011). This way of thinking entails the belief that there is only one possible, predictable future.

There is a philosophical tension between deterministic ideas and other views on human actions, such as those emphasizing free will. This tension relates to the current implications of deterministic thinking for societal problems: e.g., is social inequality determined by biological factors such that it is natural and inevitable, or can it be changed by human actions? This question hinges upon a philosophical discussion of 'nature' and 'nurture' as determinants of individual differences in physical and behavioral traits.

The empiricist philosopher John Locke expressed the idea that humans acquire all or almost all of their behavioral traits from nurture, claiming that the human mind is a *tabula rasa*, and that mental functions and behaviors develop solely from environmental influences. In contrast, the idea that human minds develop from nature rather than nurture (found, e.g., among rationalists such as Descartes) corresponds to the 'essentialist' claim that the mind is endowed from birth with certain ideas or knowledge (Doyle, 2011). Under the influence of increasing knowledge about heredity and evolution, the 'nature versus nurture' debate at the end of the nineteenth century focused on the influences of heredity and environment on social advancement (Allen, 1984).

While it is correct to assume that a number of organismal traits, including human behaviors, can be explained by appealing (at least in part) to biological factors (Resnik & Vorhaus, 2006), the excessive attribution of causal powers to these factors is often considered a misuse of biological explanations (Allen, 1984). Despite the advancement of genetics during the twentieth and twenty-first centuries, a 'straw man' model portraying genes as active and causal agents for traits has become common in some social discourses, influencing the minds of laypeople (e.g. Keller, 2000; Nelkin & Lindee, 2004); this can be seen, for instance, in expressions such as "a gene for intelligence" or "a car's DNA." At the same time, interactionist models for the relationship between genes and traits are nowadays considered more scientifically correct in most cases, where environmental and epigenetic factors play an increasingly important role. Genetic and environmental factors often interact, and genes show different levels of expression and penetrance depending on the influence of those factors (Sarkar, 1998). Indeed, a general shift has recently occurred in the scientific community from a more deterministic to a more probabilistic and complex understanding of the relationship between genes and traits, which recognizes that gene action and function are embedded in multiple hierarchical levels in which complex networks of interactions among components are the norm (Meyer et al., 2013). Thus, the degree to which genetic deterministic accounts are accepted

for biological patterns has declined in the scientific community, generally speaking, while it is argued that they are still common among laypeople.

7.2.2 How to Define and Measure Genetic Determinism

Genetic determinism is not a scientific construct with a clear definition; rather, in the literature, it is defined as a 'lay theory' developed by 'everyday' citizens as a way of making sense of genes and their functions. In psychological research, genetic determinism has been defined casually as "the biological component of essentialism" (Keller, 2005, p. 686).

As a consequence, there is no single definition of genetic determinism and, indeed, Condit (2011, p. 619) concluded in a review of the literature that "most articles that employ the term 'genetic determinism' do not give an explicit definition of the term, let alone a formal definition." However, she identified three key definitional components in her review: (1) the idea that genetics entails a closed future; (2) the pitting of genes against other particular causal agents (e.g., personal agency, social structure, culture, personal behaviors, or 'the environment'); (3) the *quantity* of influence assigned to genes in the formation of traits, i.e., an excessive belief in the 'power of genes' (Condit, 2011). In the science education literature, most of the definitions adhere to some of these three components. For example, genetic determinism has been described as if genes determine characters in a fixed manner, such that outcomes are minimally affected by changes in the environment (Kampourakis, 2017; Smith & Gericke, 2015; Stern & Kampourakis, 2017). Jamieson and Radick (2017, p. 1265) defined genetic determinism as an "attitude towards genes as overriding 'super causes' in the making of bodies and minds."

Our study uses the definition of genetic determinism provided by Gericke et al. (2017, p. 1223): "the attribution of the formation of traits to genes, where genes are ascribed more causal power than what scientific consensus suggests." This definition is operationalized by comparing respondents' attributions of genes to traits with scientific heritability scores representing the normative scientific measure for those traits. This definition focuses on the third component mentioned by Condit (2011), which enables developing measures and identifying the occurrence (or magnitude) of genetic determinist beliefs among groups of people—the aim of our study. Indeed, Kitcher (2001, p. 285) discussed the importance of quantitatively discerning the influence assigned to genes: "we might thus see genetic determination as a matter of degree."

Moreover, because genetic determinism is a lay theory, we treat it here as a belief, i.e., a state of mind in which a person thinks something is true, with or without any convincing reasons for it being the case with factual certainty (Wyer & Albarracín, 2005). This belief concerns the excessive attribution of causal powers to genes (Gericke et al., 2017) in comparison to the values estimated from heritability

studies.[2] To define genetic determinism in this manner is important, because a more nuanced understanding becomes necessary when one recognizes that we cannot deny that genetic factors are somehow involved, typically in rather complex ways, in the development of human traits, including behaviors (Turkheimer, 2011). We need to distinguish, then, between an 'excessive' genetic explanation, which we dub 'deterministic', from a 'sound' genetic explanation. We attempt to capture this distinction by differentiating between an explanation of trait formation where genes are ascribed more causal power than what scientific knowledge suggests, and an explanation where genes are ascribed the level of attribution suggested by scientific studies. In the case of complex traits (such as cognitive or behavioral ones), heritability scores tend to be relatively low, typically allowing lower levels of attributed causal or explanatory power to genetic factors than BGD entails. It seems, therefore, that this distinction can be operationalized to investigate degrees of BGD related to specific traits, as we did in a previous study in which respondents' answers that ascribed higher influence to genes than heritability studies support were referred to as 'overattribution' (which were interpreted as BGD; Gericke et al., 2017; see also Tornabene et al., 2020). Willoughby et al. (2019) applied the same approach as Gericke et al. (2017), showing its applicability in further research. Without any normative point of reference, in this case the scientific knowledge from heritability studies, it is difficult to establish whether BGD is a widespread phenomenon in societies worldwide, and therefore how relevant it is as an educational problem, as is often suggested in the literature (e.g., Nelkin & Lindee, 2004).

7.2.3 Is Genetic Determinism a Universal Belief?

In this section, we discuss the issue of whether genetic determinism is a widespread belief (i.e., if it can be found in different countries and cultures), and is dependent on the types of traits examined.

As already noted, genetic determinism can be viewed as the biological component of essentialist beliefs (Keller, 2005). Such beliefs have been researched more extensively than BGD and can be found, according to Dar-Nimrod and Heine (2011), in many cultures and countries. Essentialist reasoning is a general human tendency, and there is evidence of its presence among children and adults in an array of diverse cultures, including, for instance, underprivileged neighborhoods in Brazil (Sousa et al., 2002), herdsmen in Mongolia (Gil-White, 2001), children in Madagascar (Astuti et al., 2004), and children and adults in the United States

[2] We are aware of the limitations of heritability scores (e.g. Lynch & Bourrat, 2017), as well their dependence on context and ascription to the population level. Nevertheless, we still think heritability scores can offer relevant information for contrasting the estimated ratio of phenotypic variation in a population that one can assume to be explainable genetically from a scientific standpoint with the lay ascription of genes' causal power in trait formation, even though lay people could be thinking in individual rather than populational terms.

(Gelman, 2003). The evidence for essentialism seems broad enough to support the claim that the construct is a good candidate for a functional universal concept in humans, although cultures may vary in the degree to which these essentialist biases are present (Norenzayan & Heine, 2005). These studies lead to the notion that the closely related concept of genetic determinism may also be widespread in various cultural contexts. However, considering that BGD is regarded as a lay theory, it is worth noting that such theories often exhibit cultural differences (Andreychik & Gill, 2014). For example, people from Asia are more likely to see dispositions residing within groups, whereas Westerners tend to see dispositions as residing in individuals (Menon et al., 1999). When it comes to genetic determinism in general and the measurement of overattribution in particular, we could not find any prior cross-cultural studies, reinforcing the contribution of the present study. Norenzayan and Heine (2005) suggested a three-culture or triangulation strategy in such cross-cultural comparisons, and we employed this research design here by including data from three countries from three different continents: Europe, North America, and South America.

A second important aspect of the possible universalism of BGD is whether it is stable over different kinds of traits. The very nature of the cognitive and affective structure of BGD is uncertain, due to the few empirical studies available. Tygart (2000) suggested that genetic attribution depends on trait type, claiming the importance of investigating a large diversity of traits. Similarly, Morin-Chassé (2014) found that people convey perceptions of genetic attribution from one behavioral trait to another, but not to biological traits, indicating that they perceive different kinds of traits as separate entities. Condit et al. (2009) also found that laypeople incorporate two sets of public discourse—one that describes genetic causation and another that describes behavioral causation. In the same vein, a large study with Brazilian university students, using the same instrument as the present study and including a set of 17 different traits, found evidence for two dimensions or belief systems among the respondents: beliefs concerning social traits and those concerning biological traits (Gericke et al., 2017). These findings of two dimensions were later confirmed by a study of Swiss high school students (using a slightly different set of traits; Stern et al., 2020, p. 238) and an American sample (using the same set of traits; Tornabene et al., 2020). However, when Willoughby et al. (2019) repeated the study of Gericke et al. (2017) using yet another set of traits, in a sample of laypeople in the United States, four dimensions were found: physical traits, psychiatric traits, psychological attributes, and lifestyle attributes. Hence, from these few studies we can conclude that there is a high likelihood of different populations (e.g., university students, citizens) understanding various traits in different ways, but we still cannot say whether this is the same over various countries and cultures.

We also need to dissect the question further, by investigating whether these different ways of perceiving traits are also linked to varied levels of genetic overattribution and, thus, BGD, depending on the character of the trait. Gericke et al. (2017) reported that bipolar disorder, schizophrenia, alcoholism and, to a lesser degree, intelligence, severe depression, attention deficit hyperactivity disorder (ADHD), and violent behavior scored lower for genetic deterministic beliefs among the

participants of the study, when compared to heritability scores from the literature, whereas only two traits—related to the biological component (diabetes and breast cancer)—scored higher, indicating genetic overattribution. Hence, there was, among the Brazilian students involved in the study, a tendency to attribute less power to genes for social and mental traits, compared to biological traits. Furthermore, the overall conclusion from this study was that BGD was not a widespread phenomenon among the participants.

Similar results were reported by Willoughby et al. (2019), who found that North American laypeople seem to perceive the attribution of traits to genes well in line with the scientific view of heritability studies, as they obtained a correlation between lay estimates and published heritability scores of 0.77 when including all 21 traits, and overattribution could only be found for two traits: sexual orientation and breast cancer. Hence, based on these previous studies, we might not expect high levels of genetic overattribution in the cross-sectional study reported here, which included university students from three countries. However, given that thinking about genetic contributions to traits is likely to be influenced by culture and education, cross-country comparisons of student beliefs might reveal differences as well.

The aim of the presently described study was therefore twofold: (1) to investigate whether BGD is a general problem by investigating the possible occurrence of genetic overattribution in three different countries, Brazil, Sweden and the United States, and (2) to examine whether such beliefs generalize across different kinds of traits or are more trait-specific.

7.2.4 Methods

Instrument We analyzed data from Public Understanding and Attitudes towards Genetics and Genomics (PUGGS) subscale 2, the *Table of Traits* (for additional details, see Carver et al., 2017). This subscale consists of 17 items designed to measure BGD, and we therefore refer to them here as the BGD items (see column 1 in Table 7.1). Each item in this subscale consists of a distinct human trait (e.g., bipolar disorder) to which respondents attribute some amount of genetic or environmental contribution (see column 2 in Table 7.1) on a 5-point Likert-type scale (i.e., "only environmental contribution", "mostly environmental contribution", "both environmental and genetic contributions", "mostly genetic contribution", "only genetic contribution"). Gericke et al. (2017) coded these categories from 1 to 5, with 1 being the lowest genetic endorsement and 5, the highest. Although this coding scheme provides a measure of respondents' genetic attribution for each trait, it does not provide a readily interpretable measure of genetic *overattribution* as a single construct (i.e., BGD). That is, a score could indicate normative attribution, overattribution, or underattribution, depending upon the actual or normative level of genetic attribution for the trait in question. Heritability estimates from the published literature have been used in prior studies to classify the PUGGS traits along a continuum of genetic influence (see Table 7.1, column 3).

Table 7.1 Classifications for each trait in the Table of Traits based on heritability scores

Item	Trait	Heritability scores[a]	Normative answer[a, b]	Response indicating BGD[c]
01	Height	0.69–0.84	Mostly genetic	Only genetic
02	Bipolar disorder	0.8–0.89	Mostly genetic	Only genetic
03	Diabetes type 2	0.31	Mostly environ	Both, mostly or only genetic
04	Color blindness	0.85	Mostly genetic	Only genetic
05	Schizophrenia	0.81–0.85	Mostly genetic	Only genetic
06	Alcoholism	0.48–0.6	Both	Mostly or only genetic
07	Breast cancer	0.3	Mostly environ	Both, mostly or only genetic
08	Interest in fashion	Not available	Only environ	Mostly environ, both, mostly or only genetic
09	Addiction to gambling	0.35–0.6	Mostly environ, both	Mostly genetic, only genetic
10	Political beliefs	Not available	Only environ	Mostly environ, both, mostly or only genetic
11	Intelligence in adults	0.2–0.8	Mostly environ, both, mostly genetic	Only genetic
12	Severe depression	0.2–0.65	Mostly environ, both	Mostly or only genetic
13	ADHD	0.54–0.98	Both, mostly or only genetic	*Not productive for measurement of BGD*
14	Asthma	0.53–0.92	Both, mostly or only genetic	*Not productive for measurement of BGD*
15	Violent behavior	0.29–0.56	Mostly environ, both	Mostly or only genetic
16	Religious beliefs	Not available	Only environ	Mostly environ, both, mostly or only genetic
17	Blood group (ABO)	1	Only genetic	*Not productive for measurement of BGD*

BGD, belief in genetic determinism

[a]Information gathered from Gericke et al. (2017)

[b]Based on the heritability scores in column 3

[c]During analysis, all responses matching these classifications were coded as "1", indicating the presence of BGD for that trait (all other responses were coded as "0", indicating the lack of BGD)

Measures Derived from the PUGGS To use the Table of Traits for a more direct measurement of the intended construct of BGD, we modified the original PUGGS coding scheme such that responses reflecting genetic overattribution relative to the heritability estimate were coded as "1" and all others were coded as "0". We determined what response categories should be coded as 1 or 0 for each trait by first establishing which response(s) could be considered a correct or normative level of genetic attribution based on published heritability scores. The normative levels of genetic attribution are shown in Table 7.1, column 4. All responses attributing a role of genetics that was above this normative level were considered to be genetic overattribution, i.e., indicating BGD, and coded as 1. The responses that were considered genetic overattribution for each trait are shown in Table 7.1, column 5. All responses attributing a role for genetics that was either consistent with or below this normative level were coded 0. For example, the heritability score reported in the literature for

the trait of color blindness is about 0.85, and we therefore set the normative level of genetic attribution to "mostly genetic". A respondent would be correctly attributing the role of genetics for this trait if they selected "mostly genetic" but would be overattributing the role of genetics for this trait if they selected "only genetic contribution". In the event of this latter selection, the respondent would be given a score of 1, indicating the presence of BGD for this trait because they expressed more genetic attribution than the heritability score supports. All other selections would be coded as 0, indicating lack of BGD for this trait.

It is important to note that three of the traits (ADHD, asthma, blood group) are limited in their ability to measure BGD because they have very high heritability scores. Consequently, none of the responses on the BGD rating scale would represent genetic *over*attribution. In other words, it would be impossible for a respondent to be classified as endorsing BGD using these traits/items. Therefore, these items were removed from subsequent analyses because they were not productive for measurement. These items could be used for a study of genetic *under*attribution, but this was not the focus of the current study.

In addition to collecting responses from the PUGGS subscales, we gathered participant background data: age group (> or < 21 years of age), gender, year in college, major, knowledge of someone with a genetic disease, and the influence of religion (i.e., "In general, to what extent are your opinions and decisions influenced by your religion?"). These background variables were used in our analyses of differences across countries. See Carver et al. (2017) for details on these items.

Sampling and Data Collection Our participant sample included university undergraduate students from Brazil, Sweden, and the United States. We used a targeted sampling strategy, seeking a participant sample with basic exposure to normative genetics content (i.e., high-school education, and varying exposure to more advanced topics in genomics). The study was conducted according to Brazilian, Swedish, and North American guidelines for ethical conduct involving humans. All participants provided written informed consent, and the questionnaire was anonymous. The instrument was administered in either online or paper format. The Brazilian participant sample was drawn from several classes of first-year Brazilian undergraduates enrolled in an Interdisciplinary Bachelor Program at a large public university in northeast Brazil in February and March 2015. The Swedish sample was drawn from the first-year classes of several undergraduate courses at a mid-sized university in central Sweden between February and April 2019. The United States participant sample was drawn from two semesters (Fall 2016, Fall 2017) of an introductory biology course at a large, public, research-oriented university in the northeastern United States. The demographic and background variables for these samples are shown in Table 7.2. Note that in our comparisons, we controlled for background variables (see below).

In this study, we conducted an exploratory investigation, in which descriptive statistics for each item were used to identify possible genetic overattribution for individual items/traits.

Table 7.2 Demographic and background variables for each country

	Brazil	Sweden	United States
Sample size	441	355	402
Targeted courses	Several intro-level undergraduate courses in a range of subjects	Several intro-level undergraduate courses in a range of subjects	One intro-level undergraduate course in biology
University size	Large	Medium-size	Large
Typical time to survey completion	20–25 min	10–15 min	10–15 min
Percent female	50	55	57
Percent ≥21 years of age	67	41	92
Percent ≥year 2	89	86	51
Percent bio majors	22	21	62
Percent respondents who know someone with a genetic disease	29	26	48
Percent respondents somewhat or greatly influenced by religion	45	17	18

7.2.5 Results

Overall, participant response patterns were remarkably consistent across the three countries (Fig. 7.1). Most items in all countries had no or very few responses indicative of trait-level genetic overattribution, as indicated by less than 1% of respondents showing BGD (coded as 1). However, five items—'breast cancer', 'type 2 diabetes', 'interest in fashion', 'political beliefs' and 'religious beliefs'—had a relatively high percentage of responses suggesting genetic overattribution. This was especially evident for the trait 'breast cancer'. Although all countries displayed elevated levels of genetic overattribution for these five items, the magnitudes differed across countries. Specifically, controlling for all background variables (including prior experience with genetic disease and self-reported impact of religion), Brazilian respondents were approximately 2.5 times more likely than United States respondents to endorse genetic overattribution for 'diabetes'.[3] Conversely, Swedish and United States respondents were about 3 times more likely than Brazilian respondents to endorse genetic overattribution for 'interest in fashion'.[4] Finally, Swedish respondents were 6 times more likely than Brazilian respondents, and 2 times more likely than United States respondents, to endorse genetic overattribution for

[3] Brazil vs. United States (US): B = 0.16, $p < 0.001$, df = 1127; log odds ratio = 2.54.

[4] Sweden vs. Brazil: B = 1.08, $p < 0.001$, df = 1128, log odds ratio = 2.95; US vs. Brazil: B = 1.03, $p < 0.01$, df = 1134, log odds ratio = 2.79.

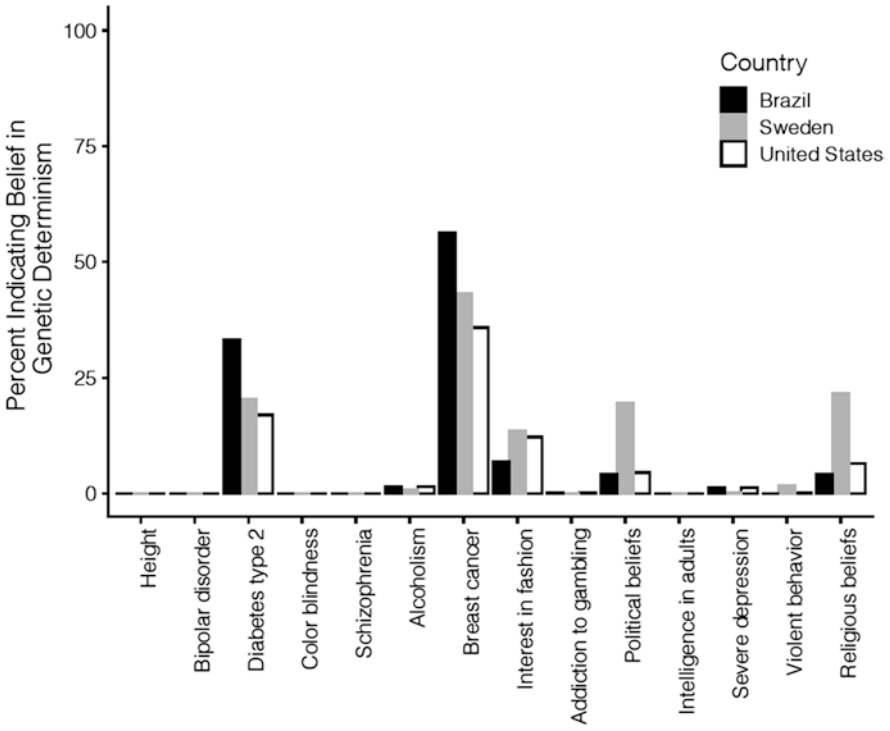

Fig. 7.1 Percentage of respondents endorsing genetic overattribution for each trait in each country

'political beliefs'[5] and 'religious beliefs'.[6] In summary, trait-level genetic overattribution was low in all three countries; only one of the 14 traits investigated had a majority of participants endorsing genetic overattribution (i.e., breast cancer), whereas low, significant differences in genetic overattribution in some traits were found across countries.

In the United States and Brazil, background variables (i.e., gender, age, major, year in school, impact of religion, experience with genetic condition) did not contribute to patterns of genetic overattribution for any of the traits analyzed. However, in Sweden, there was evidence of aspects of religion, gender, and education (major) being associated with genetic overattribution for selected traits. Controlling for all other background variables, those respondents who self-reported an impact of religion on their opinions and decisions were 3 times more likely to endorse genetic overattribution for 'religious beliefs'.[7] Non-biology majors were over 4 times more

[5] Sweden vs. Brazil: B = 1.88, $p < 0.001$, df = 1130, log odds ratio = 6.58; Sweden vs. US: B = 0.96, $p < 0.01$, df = 1130, log odds ratio = 2.6.

[6] Sweden vs. Brazil: B = 2.22, $p < 0.001$, df = 1133, log odds ratio = 9.2; Sweden vs. US: B = 1.20, $p < 0.001$, df = 1133, log odds ratio = 3.31.

[7] B = 1.26, $p < 0.01$, df = 331, log odds ratio = 3.54.

likely than biology majors to endorse genetic overattribution for 'political beliefs'.[8] Females were twice as likely as males to endorse genetic overattribution for 'breast cancer'.[9] In summary, background variables differentially contributed to endorsements of genetic overattribution, with the Swedish sample displaying significant effects, in contrast to Brazil and the United States, as can be seen in the calculations shown in footnotes 3–9.

7.3 Remaining Issues

7.3.1 Genetic Overattribution Across Countries

Using raw data suitable for describing genetic overattribution, we reported an overall low incidence across countries, with the same nine traits showing almost no genetic overattribution in any of the countries studied. While some traits—the same five in each country—showed elevated rates of genetic overattribution, rarely did a majority of respondents endorse a deterministic response. To conclude, this large study in three countries from three different continents did not support the idea that genetic determinism is a general and widespread belief. This general finding of low overall genetic overattribution was also reported by Gericke et al. (2017) in the same sample of Brazilian undergraduates (using a distinct analytical approach), as well as by Willoughby et al. (2019) for laypeople in the United States. However, it is at odds with other prior literature (e.g., Dar-Nimrod & Heine, 2011; Keller, 2005; Nelkin & Lindee, 2004). Several authors have previously reported BGD to be a widespread phenomenon in common discourse (Keller, 2000; Nelkin & Lindee, 2004), in the media (Condit et al., 2001), and in school textbooks (Gericke et al., 2014). Furthermore, essentialism, of which genetic determinism can be considered the biological component, has been commonly reported to be a bias in many countries (e.g., Astuti et al., 2004; Gelman, 2003; Gil-White, 2001; Sousa et al., 2002). The low prevalence of genetic overattribution across countries in our study is surprising, both because of the plentiful literature suggesting otherwise, and because of the diverse cultures and educational systems in the United States, Brazil, and Sweden. However, considering that two studies using the same approach (Gericke et al., 2017; Willoughby et al., 2019), based on the Table of Traits from the PUGGS, generated the same results, the results of this study might be expected. The consistent results in all three studies further suggest that genetic determinism—when defined as overattribution (i.e., ascribing more causal power to genes in the formation of traits than available scientific knowledge endorses)—may not be a general problem spread over the world.

[8] Biology majors vs. all other field categories: B = 1.53–2.75, $p < 0.001$, df = 328, log odds ratios = 4.61–15.54.

[9] B = 0.88, $p < 0.001$, df = 328, log odds ratio = 2.4.

It is important to emphasize that our results do not address the issue of whether genetic determinism is linked to social categorization or prejudiced attitudes, as found by Keller (2005). Hence, among the participants of this study, there might exist smaller groups showing BGD, and those groups might be more prejudiced against social groups based on gender, race, or sexual orientation, but this was not studied here. Based on these results, one might be tempted to claim that if genetic determinism were the major cause of prejudiced attitudes, then this would not be a major problem in society. We think, however, that it is more likely that the interpretation suggested by Condit (2011) applies, namely, that people who already have prejudiced attitudes tend to endorse genetic deterministic explanations to support their already existing biases.

Additional questions raised by the findings of this study include: In what ways is genetic determinism related to essentialism? Can genetic determinism be explained as the biological component of essentialism? As outlined previously, studies have found essentialism to be a widespread bias (e.g., Dar-Nimrod & Heine, 2011; Gelman, 2003), but the same conclusion cannot be drawn for genetic determinism based on our study. Perhaps there are other predictors that more strongly support essentialist ideas than genetic determinism.

7.3.2 Genetic Overattribution Across Different Traits

Of the five traits that showed evidence of genetic overattribution, two were human diseases: 'breast cancer' and 'type 2 diabetes.' The other disease traits in the BGD item set (e.g., 'schizophrenia', 'bipolar disorder', 'alcoholism', 'depression') did not show evidence of genetic overattribution. Hence, it is difficult to discern any clear tendency or difference between different kinds of traits, such as biological and social, as previously discussed by Gericke et al. (2017). Research has shown that individuals tend to overestimate genetic contributions to diseases perceived as biological (e.g., 'cancer' and 'diabetes'), but not to those perceived as psychological (e.g., 'schizophrenia' and 'bipolar disorder') (Condit, 2010). The three other traits in which we found evidence of genetic overattribution—'political beliefs', 'religious beliefs', and 'interest in fashion'—are social traits that might be stereotypes of certain groups. The stereotype may be perceived as part of the 'essence' of an individual and the social group to which that individual is assumed to belong. In this way, essences might be naively used for categorization, just as one might naively use genes: both essences and genes may be considered natural, immutable, homogeneous within groups, discrete across groups, and fundamental aspects of the individuals to which they belong (Dar-Nimrod & Heine, 2011). In these terms, social stereotyping may be linked to deterministic thinking (Dar-Nimrod & Heine, 2011) and may explain the patterns of genetic overattribution for certain socially relevant traits. Given this, it is also possible that social traits and disease traits might elicit deterministic conceptions for different reasons, which could explain why these two trait types differed in their patterns across countries: Brazil had the highest rate of

genetic overattribution for the two disease traits and Sweden had the highest rate of genetic attribution for the three psychological/social traits. Similarly, Willoughby et al. (2019), in their study of laypeople in the United States, found overattribution for 'breast cancer', but not for 'diabetes' or 'political beliefs'. However, they did not include the traits 'religious beliefs' and 'interest in fashion' but did include 'sexual orientation', for which they found overattribution. Hence, there are some interesting and consistent results across studies, such as the genetic overattribution for breast cancer. But it seems difficult to generalize depending on the character of specific traits, which in turn calls for more studies of this topic.

When individuals' essences or genes are used to explain group differences, especially for the purpose of justifying one's position in society, deterministic concepts become motivated by social factors (e.g., Haslam et al., 2006; Keller, 2005). As a result, genetic determinism may be drawn out by some contexts and in some respondents for whom those social factors are salient, but not others. For example, it is notable in our results for the Swedish sample that the background variables that predicted genetic overattribution for 'religious beliefs' and 'breast cancer' (self-reported impact of religion and gender, respectively) were both relevant to the trait itself. These traits, therefore, may have drawn out personal and identity-focused reasoning about the role of genes vs. environment in phenotype development. It is possible that the BGD traits that failed to elicit genetic overattribution in this study would be more likely to do so if they were situated in more explicit social contexts. For example, the traits could be associated with particular groups for which there are well-known stereotypes (religious vs. non-religious individuals, males vs. females, among others) (see, e.g., Donovan, 2017). Suhay and Jayaratne (2012) found that genes were used to causally explain *group* differences regarding race, class, and sexual orientation, but this strong genetic attribution was not observed when participants were asked to explain *individual* traits. In light of these concerns, in the following section we discuss what implications these findings might have for the teaching of genetics.

7.4 Implications for Teaching

Although there seems to be some inconsistency in the literature regarding how widespread BGD is within populations (Gericke et al., 2017; Willoughby et al., 2019), it appears to be pervasive in at least some contexts (e.g., Nelkin & Lindee, 2004). Our study of undergraduates from Brazil, Sweden, and the United States suggests that genetic overattribution in relation to traits is rather limited. Indeed, only one trait (breast cancer) in one country (Brazil) was characterized by genetic overattribution by most of the students. Nevertheless, some significant differences in the magnitude of genetic overattribution were identified across countries (see Fig. 7.1), making the issue of how to address them in teaching an important consideration.

Students may enter their biology courses with a variety of normative and non-normative ideas about how the environment and genetics impact the form and function of traits. Teaching aimed at generating normative conceptualizations of the relative roles of genes and the environment should be designed with diverse misconceptions in mind. Genetics education research on student conceptions has repeatedly shown that students have difficulties distinguishing between genotype and phenotype (e.g., Haskel-Ittah & Yarden, 2017; Lewis & Kattmann, 2004), a confusion that is aligned with a genetically deterministic understanding in which the mediating effect of gene expression is ignored. Investigations of students' understanding of the genotype–phenotype relationship have found multiple underlying and partially overlapping models of the gene and its function (summarized by Gericke & Smith, 2014). Moreover, explanations considering molecular processes and gene expression are only found among the most advanced students in high school and university education. According to these studies, genetic deterministic understandings may be considered a 'knowledge problem'.

Conversely, in the socio-psychological literature (see Introduction), genetic determinism is viewed as an ideology, a value, or a belief system (e.g., Haslam et al., 2006; Nelkin & Lindee, 2004; Norenzayan & Heine, 2005). The PUGGS instrument attempts to relate these research traditions to each other. In the study by Gericke et al. (2017), the relationship between knowledge of genetics (considering contributions of genes and environment to trait formation, and modern genetics and genomics) and the BGD items from the Table of Traits was researched, but no meaningful correlations were found; nevertheless, other studies have indicated that increased levels of genetic knowledge might refute essentialist thinking (Donovan et al., 2020) and genetic determinism (Jamieson & Radick, 2017). Here, we sought to broaden our usage of the PUGGS by comparing the BGD items across countries using coding aligned with genetic overattribution (see also Tornabene et al., 2020). Our results indicate that some traits may not elicit genetic overattribution among the vast majority of learners, while other traits may do so differentially (e.g., breast cancer), with some respondents more likely than others to have deterministic conceptions triggered. This is a novel finding, which to our knowledge has not been previously addressed in genetics education research. Therefore, one major implication of this study is that educators must be careful when selecting the traits they include in their instruction, and consider the possibility that students with different background characteristics may think differently about them.

The human traits currently utilized in the BGD item set (Table of Traits) were selected from textbooks (Carver et al., 2017) and thus represent common examples used in many classrooms. However, as shown in our study, individual students do not think about each of these traits in the same way. Our recommendation is therefore to direct more attention to (1) the various social contexts to which the traits used in instruction are related, and (2) how students' background variables may influence their interpretations of and responses to the instructional material, making it important for the teacher to create a common ground of understanding in the classroom (e.g., through classroom discussions using dialogic teaching). In line with both recommendations, other scholars in socio-psychological research have

suggested that a diversity of background variables may trigger genetic deterministic conceptions in various societal contexts and may be more predictive of genetic deterministic conceptions than genetics knowledge (e.g., Nelkin & Lindee, 2004). Therefore, whether teaching and increasing levels of knowledge act to reduce BGD (in cases where it occurs) remains an open question. Hence, it is important as a teacher to be aware that successful teaching may lead to an improved conceptual understanding in genetics but will not necessarily reduce deterministic beliefs. Efforts to increase student knowledge should be combined with discussions of socio-scientific issues related to genetics, including ethical and political dimensions, if the aim of teaching is to foster less genetic deterministic beliefs among students.

Another important consideration for instructors regarding the diverse misconceptions of their students is that genetics learning challenges may include both over-*and* underemphasis of genes as causal factors. Therefore, it is important to both avoid overstating the causal power of genes, given the complex nature of development, and clearly establish that they are involved in the causal processes leading to most traits, and in some cases to a significant degree. As educators work to address genetic determinism among their students, any intervention should be carefully designed to offer a balanced view of the relative contributions of genes and environment. Our goal as genetics educators should be to replace excessive reliance on genetic causal power in explaining trait formation with sound genetic explanations in which one ascribes the proper level of causal power to genes, as sanctioned by accepted scientific studies, but not a denial of genetic explanations *in toto*. If we agree as a community that genetic determinism is a poor outcome of genetics education, an underappreciation of the causal role played by genes in trait development is equally poor. In our efforts to develop a more complex understanding of trait formation, we must be careful not to inadvertently endorse the equally limited view of environmental or cultural determinism. Such a misstep would reinforce the problematic nature–nurture dichotomy, instead of promoting a more sophisticated understanding of gene–environment interactions.

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Chapter 8
Why Does Multiple and Interactive Causation Render Comprehension of Genetics Phenomena Difficult and What Could Genetics Educators Do About It?

Marcus Hammann, Tim Heemann, and Johannes C. S. Zang

8.1 The Problem

Resulting from multifactorial and interactive causation, complex genetic phenomena are difficult to understand, even for scientists. Nevertheless, research in gene–environment interactions has contributed to significant advances in understanding diseases (Jackson, 2014; McAllister et al., 2017; Ritz et al., 2017), psychological traits and disorders (Barlow, 2018; Dick, 2011), human behavior (Rutter, 2006), and development (Lovely et al., 2017). The term gene–environment interplay refers to the fact that genetic and environmental causation are rarely separate or direct (Rutter, 2006). The formation of traits generally involves multiple genes (polygenic), multiple environmental factors, and the complex interplay between genes and environment at the different levels of biological organization (Champagne, 2018; Kampourakis, 2017; Moore, 2015). The media, however, sometimes misrepresent this complexity, for example, by claiming the discovery of a single gene for a complex trait such as alcohol use disorder (Dar-Nimrod & Heine, 2011). Complex traits are rarely caused by single genes. Rather, genetic networks interact with environmental factors and increase the risk for developing a complex trait (Buchanan et al., 2009; Kendler, 2005).

Although the topic is complex, there is consensus that the interplay between genes and environment is a core idea of genetic literacy (Boerwinkel et al., 2017). Fifty-seven experts participating in a delphi study brought to light the genetics knowledge that is relevant for laypeople in the twenty-first century. Nine knowledge categories of genetic literacy emerged. One of them addressed understanding

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multiple and interactive causation of genetic phenomena: “Multiple genes and multiple environmental factors interact in the development of most traits.” Some experts even argued that gene–environment interaction was the most relevant category of all (Boerwinkel et al., 2017). Furthermore, a learning progression for modern genetics education for grades 5–10 emphasizes the importance of understanding the interplay between genes and the environment (Duncan et al., 2009). In general, learning progressions describe possible pathways of students’ deepening understanding in a domain. They allow educators to coordinate curricula, instruction and assessment so that the students can revisit the same concept several times (cumulative learning).

The general pathway for understanding the complex interplay between genes and the environment leads from the organismal level to the molecular level. In grades 5–6, genetic educators are recommended to address the interplay between genes and the environment at the level of the phenotype. In particular, the students should understand that the environment can affect human characteristics such that organisms end up looking or behaving differently, even though they are related (Duncan et al., 2009). In grades 7–8, students should deepen their understanding of the interplay between genes and environment to the level of gene products. In particular, teachers are recommended to teach that the environment can influence cell functioning through changes at the protein level. In grades 9–10, students should become familiar with the interplay between genes and environment at the level of genes and gene regulation. More specifically, they should learn that environmental factors can cause mutations in genes, or alter gene expression.

There are multiple barriers to effectively teaching about the interplay between genes and the environment at the high-school level: curricula focus on Mendelian genetics which directly links single genes and simple traits (McElhinny et al., 2014), educational standards inadequately address the impact of the environment on genes and their products (Dougherty et al., 2011), high-school textbooks provide only limited discussions on genetic and environmental influences on multifactorial diseases (Hicks et al., 2014) and omit the impact of the environment on gene expression altogether (Aivelo & Uitto, 2015; Martínez-Gracia et al., 2006), trait-formation tasks in high-school textbooks hardly ever address the role of the environment in trait formation (Heemann & Hammann, 2020), internet websites fail to address gene–environment interactions (Cheng et al., 2008), and media portrayals emphasize genetic influences and diminish environmental ones (Horwitz, 2005).

Furthermore, researchers have argued from their own teaching experience that most students, paradoxically, know intuitively that the environment influences complex traits, such as body height, but they do not know that the environment also affects the expression of many monogenic disorders because they are not taught this knowledge (Dougherty, 2009). This lack of knowledge is hypothesized to reinforce the students’ belief that genes alone determine traits.

Development of this monocausal understanding of the linkage between genes and traits might be further supported by the architecture of cognitive processes that

directly link causes and effects to structure information when the individual is confronted with complexity. There is a rich body of literature suggesting that students struggle to comprehend distributed, interactive, time-delayed or nonlinear patterns of causality. Instead, the student's mind favors simplistic, linear relationships, and it tends to directly link causes and effects (Chi et al., 2012; Grotzer, 2012). These cognitive default patterns help deal with tasks in environments that are overwhelmingly rich in information. However, they might present an obstacle when dealing with genetic phenomena, because students favor simplistic and linear instead of complex and multicausal relationships. Hence, while current teaching practice fails to provide knowledge about different sources of variation and instead tends to provide monocausal (Mendelian) models of trait formation, these aspects are met by a cognitive setup that is eager to prefer simplistic over complex explanations.

Recently, behavioral geneticists have described a psychological obstacle to acknowledging gene–environment interactions (Barlow, 2018). Authors have argued that genetic literacy involves the understanding that all complex psychological traits are the result of multifaceted gene–environment interactions. Individuals, however, may not be willing to acknowledge the fact that genes contribute to the formation of traits, because genetic explanations for traits and behaviors often go hand in hand with racialized genetic attributions, which increase social inequalities and heighten prejudice. When people encounter the argument that genes are relevant for a behavior or trait, their essentialist biases are activated, in particular the beliefs that the behavior or trait is immutable and determined, that the outcome of the behavior or trait is natural and good (naturalistic fallacy), and that groups which share the genetic basis for the trait or behavior are homogeneous and discrete (Dar-Nimrod & Heine, 2011). For example, genetic attributions are often associated with the belief that differences between groups cannot be changed because they are genetic. Even more importantly, genetic attributions for perceived racial and ethnic differences are associated with prejudice and discrimination (Dar-Nimrod & Heine, 2011; Jayaratne et al., 2006, 2009). Because people intentionally want to avoid prejudiced thinking, they may deny scientific evidence supporting the role of genes (and gene–environment interactions) in the formation of traits. For health outcomes, in contrast, acknowledging the importance of genes (and gene–environment interactions) seems less problematic. In particular, health assessment, evaluation and promotion models stress the fact that individuals vary in their inherited predisposition to certain health conditions, but can control their exposure to environmental risks through their own behavior (e.g., Doyle et al., 2019).

Research-based information about high-school students' intuitive causal attributions of genetic phenomena is lacking. Without this knowledge, genetics educators cannot inform the development of effective instruction that will prevent inadequate gene-only and environment-only (monocausal) attributions of human characteristics. According to attribution theory (Weiner, 1986, 1995), a causal attribution is defined as an individual's perception of the cause of a behavior or event. Causal learning theory hypothesizes that children possess causal knowledge in the form of

causal maps, which allow them to solve causal inverse problems and make assumptions about the causal structure of the world (Gopnik et al., 2004). Furthermore, the causal knowledge acquired by children is hypothesized to act as a constraint on later causal inferences. According to constructivist learning theories, learning is most effective when teachers provide students with the opportunity to relate new knowledge to what they already know. Thus, knowledge about high-school students' intuitive causal attributions of genetic phenomena is important, so that genetics instructors can relate to it and reconstruct it if necessary. For understanding multiple and interactive causation, gene-only and environment-only (monocausal) attributions of human characteristics are deemed problematic (see Fig. 8.1). In this study, therefore, we seek to

- characterize individuals' causal attributions of human characteristics and
- discuss the implications of the findings for genetics instruction.

Type of attribution		Abbreviation
genes only		**G-only**
environment only		**E-only**
genes and environment, but non-interactive		**GIE**
gene-environment-interaction		**GxE**

Fig. 8.1 Laypeople's causal attributions in studies positing two major causal categories

8.2 Current Knowledge About the Problem

This section is divided into two parts: the first reports on empirical findings on laypeople's and teachers' causal attributions of human traits and behaviors; the second reports on findings from a pilot study conducted by the first and second authors of this paper to investigate high-school students' causal attributions of human behavioral and psychological traits. Whereas the information in the first section is based on a broad literature base, the second section is based on a single study because we found no research on high-school students' causal attributions of human traits.

8.2.1 Laypeople's Causal Attributions of Genetic Phenomena

Studies focusing on laypeople's and teachers' causal attributions of human characteristics can be broadly divided into two groups: studies based on the *nature–nurture* debate positing two primary causal categories (e.g., Carver et al., 2017; Crosswaite & Asbury, 2018; Singer et al., 1998), and those based on both the *nature–nurture* debate and the *determinism–free will* debate, positing three primary causal categories (Condit et al., 2004; Jayaratne et al., 2009). The nature–nurture debate focuses on the influence of genetic (nature) and environmental (nurture) factors. Accordingly, individuals' causal attributions of human characteristics are assumed to be genetic and/or environmental. Some studies in this group make finer distinctions by dividing environmental factors into the physical environment (e.g., air quality), social environment (e.g., access to education) and behavior (e.g., physical activity) (e.g. Parrott et al., 2003). The *determinism–free will* debate focuses on the question of whether individuals can control human characteristics by free will and individual decisions, or whether these characteristics are determined by something like natural laws. Accordingly, people are assumed to attribute human characteristics to genetic factors, environmental factors and/or personal decision. Jayaratne et al. (2009) speak of personal choice (rather than personal decision), and they define personal choice as "how much someone chooses to be one way or another" (p. 26). To date, little is known about how these primary causal categories relate to each other, although there is evidence to suggest that people perceive genes and personal choice as negatively correlated (Jayaratne et al., 2009).

Studies positing two primary causal categories have provided four major insights. First, laypeople and teachers assign substantial roles to both genetic and environmental factors when asked to assess their relative roles (Condit, 2011; Condit et al., 2006; Crosswaite & Asbury, 2018; Parrott et al., 2003; Walker & Plomin, 2005). Thus, laypeople rarely attribute human characteristics to genes alone or to the environment alone (compare Condit, 2011 and G-only and E-only causal attributions in Fig. 8.1). Second, the relative influence of the different factors in individuals' causal attributions varies according to the trait. In general, individuals attribute greater genetic influence to features of the body than to features of the mind, and greater

environmental influence to features of the mind than to features of the body (mind/body split; see Condit, 2011). For example, individuals assigned 71% of the influence over height to genes, 41% over weight, 31% over lung cancer, 26% over talents, and 40% over mental abilities (Parrott et al., 2003). Furthermore, individuals assigned 12% of the influence over height to the physical environment, 16% over weight, 23% over lung cancer, 18% over talents, and 17% over mental abilities (Parrott et al., 2003). Third, although laypeople intuitively acknowledge both nature *and* nurture (rather than nature *versus* nurture) in their causal attributions, they rarely describe gene–environment interactions (compare Condit et al., 2009 and GxE causal attribution in Fig. 8.1). Instead, genes and the environment are often believed to act separately and independently, for health outcomes as well (two-track model of causal attribution, compare Condit, 2011 and G | E causal attribution in Fig. 8.1). Fourth, the correspondence of laypeoples' estimates of genetic attributions with published heritability estimates for 21 human traits was large (r = 0.77), even though causal attributions were intuitive rather than based on knowledge of genetics (Willoughby et al., 2019).

Personal decision emerged as a third primary causal factor for human characteristics in other studies with laypeople (Condit et al., 2004; Jayaratne et al., 2009; see also Condit, 2011). In particular, people held the belief that individuals make decisions and are thus responsible for their own positions in life. Attributing the development of alcohol use disorder to both genes and personal decision, one person said: "You have to make a choice to be an alcoholic" (Condit et al., 2004, p. 261). Analyzing how a diverse sample of Americans attributed human characteristics to genes, the environment and personal decision, Jayaratne et al. (2009) reported a significantly negative correlation between genetic and personal-decision attributions. Furthermore, while most respondents attributed human characteristics to all three primary causal factors with their relative weights differing from trait to trait, ethnicity seemed to influence causal attribution patterns for several behavioral traits.

8.2.2 High-School Students' Causal Attributions of Behavioral and Psychological Traits

The aim of this study was to explore high-school students' causal attributions of behavioral and psychological traits; 242 high-school students (16–17 years old, 154 female) participated. Approximately half of the sample (45%, n = 109) was in grade 11 and possessed basic knowledge of genetics; the other half (55%, n =133) was in grade 12 and possessed more advanced knowledge of genetics because they had completed a semester-long genetics course. The curricula for both groups of students focused on: the genetic model (i.e., patterns of inheritance between parents and offspring), the meiotic model (i.e., the passage of genes from parents to offspring through sperm and egg), and the molecular model (i.e., how genes are translated into proteins that bring about physical traits) (Stewart et al., 2005). An analysis

of the textbooks used for these students revealed that the model of trait formation conveyed to the students was predominantly gene-centered, rather than dual-focused on both genes and the environment, or on gene–environment interactions (Heemann & Hammann, 2020). For example, only 5% of the integrative learning tasks (n = 2) focused on the role of the environment in trait formation. The authors of the study defined integrative tasks as tasks addressing the relationships between genes, proteins and traits.

We asked the students to respond to two scales focusing on behavioral and psychological traits, the belief-in-genetic-determinism (BGD) scale (Keller, 2005) and the biological-basis-of-genetic-determinism (BB) scale (Bastian & Haslam, 2006). For both instruments, we used 4-point Likert scales (agree, rather agree, rather don't agree, and don't agree). The BGD scale (Keller, 2005) consists of 18 items, which can be classified into three item types. One item type is formed by statements about the influence of genes on traits, for example, "In my opinion, alcoholism is caused primarily by genetic factors." Another item type addresses genes as a cause for similarities and differences between people, for example, "I think that differences between men and women in behavior and personality are largely determined by genetic predisposition." A third item type focuses on the impact of genes on a person's fate, for example, "The fate of each person lies in his or her genes." The items in the scale address the following traits: character (A1), alcoholism (A2), behavior (A1, A3, A6, A11, A17), personality (A3, A10), personal traits (A4, A8), homosexuality (A5), talents (A7), abilities (A8, A16, A18), differences between humans with different skin color (A9), fate (A12), intelligence (A13), intellectual abilities (A14), characteristics (A15), and traits (A18). The BB scale (Bastian & Haslam, 2006) consists of eight items, which can be classified into two item types. One item type is formed by statements about the influence of genes on traits, for example, "The kind of person someone is can be largely attributed to their genetic inheritance." The other item type consists of statements about the influence of a person's biology (or biological makeup) on traits, for example, "Whether someone is one kind of person or another is determined by their biological makeup." Items in the scale address the following traits: the kind of person someone is (B1, B3, B4), a person's traits (B2, B8), different types of people (B5), a person's attributes (B6) and a person's basic qualities (B7).

Two weeks after the survey, we conducted interviews with seven students from the same sample. The interviews were semi-structured and focused mainly on the students' ratings of the items of the BGD scale (Keller, 2005). More specifically, we asked the students to elaborate on their ratings of the items in the questionnaire and provide insights into the reasons for their ratings. All students were asked to choose items from the questionnaire and explain why they agreed or disagreed with the genetic attribution of the trait stated in the item. The interviewer encouraged the participants to further elaborate on their answers and give examples. At the end of the interviews, the students were asked whether they knew the terms "polygenetic inheritance" and "gene–environment interaction." The interviews lasted approximately 20 min. They were audiotaped and transcribed verbatim with MAXQDA. Case descriptions were used to summarize transcripts. Qualitative content analysis

focused on the three primary causal categories (genes, environment and personal decision) to characterize students' causal attributions.

8.2.3 Quantitative Findings for the BGD and BB Scales

Analysis of scale means for the BGD scale ($M = 2.29$, $SD = 0.34$) and the BB scale ($M = 2.11$, $SD = 0.47$) showed that the students tended to reject genetic attributions of human behavioral and psychological traits (see Table 8.1). Means were significantly below the theoretical scale midpoint (M = 2.5) for both the BGD scale [$t(241) = -9.28$, $p < 0.001$] and the BB scale [$t(241) = -12.66$, $p < 0.001$]. Correlation analysis showed a substantial and significant positive relationship between students' ratings on the BGD scale and BB scale ($r = 0.61$, $p < 0.001$). Neither grade nor biological sex had a significant influence on student's total scores on the BGD or BB scale. Furthermore, we found no sex-specific differences in student's ratings of BB scale items. For one item of the BGD scale, however, male students tended to agree more strongly ($M = 2.27$, $SD = 0.78$) than female students ($M = 2.09$, $SD = 0.78$): "I am convinced that the analysis of the genetic predispositions of an embryo allows good predictions as to which characteristic and abilities the child will develop" [$t(240) = 2.22$, $p < 0.05$, $d = 0.3$]. According to Cohen's convention, however, effect size was small and biological sex unequally distributed in the present sample. Therefore, we regard this as an interesting but preliminary indication of sex-specific differential item functioning. Further analyses did not reveal grade-specific rating differences for items of the BGD or BB scales.

8.2.4 Qualitative Findings for High-School Students' Lack of Endorsement of Items in the BGD Scale

General Characterization of Students' Causal Attributions The interviews provided insights into high-school students' lack of endorsement of genetic attributions for human behavioral and psychological traits. Interviews yielded three major insights. First, all seven students did not endorse most of the genetic attributions in the questionnaire. Rather, when elaborating on the causation of human traits and

Table 8.1 High-school students' ratings of items of the BGD and BB scales

Scale	n (items)	M	SD	r_{it}	α	Sample item
BGD	18	2.29	0.34	0.17–0.50	0.72	In my opinion, alcoholism is caused primarily by genetic factors. (A2)
BB	8	2.11	0.47	0.36–0.49	0.75	The kind of person someone is can be largely attributed to their genetic inheritance. (B1)

α = Cronbach's α, r_{it} = Item-total correlation

behaviors, they referred to a person's *education and upbringing*, the *social and cultural environment*, *personal decisions* and *adverse life experiences*. Second, the relative weight of nongenetic and genetic factors in high-school students' causal attributions varied from trait to trait and from student to student. For some traits, students argued that nongenetic factors are the only cause, for example when students attributed human behavior to the social environment or to personal decision alone. Other students focused on genetic and nongenetic factors in their causal attributions, but relegated a secondary role to the genetic factors. Such students put forth the views that genetic factors and nongenetic factors contribute separately and independently (G | E causal attribution in Fig. 8.1) and that nongenetic factors are more important than genetic ones. The students hardly ever endorsed genes-only (monocausal) attributions. Furthermore, students tended to use stronger genetic attributions for features of the body and stronger environmental attributions for features of the mind. When commenting on their causal attributions, high-school students mainly relied on two sources of knowledge, both related to experience: personal and that of a close person. Third, few students described the regulation of gene activity. Furthermore, the term gene–environment interaction was unfamiliar to them.

The Role of Genes in High-School Students' Causal Attributions Students rarely attributed traits to genes alone. Rather the students predominantly argued that human behavioral and psychological traits are caused by either genetic and nongenetic factors, or nongenetic factors alone. Furthermore, students distinguished between features of the body and of the mind. Generally, students tended to use stronger genetic attribution for the former and stronger environmental attributions for the latter. The students, for example, elaborated on the following item from the BGD scale: "I believe that many differences between humans of different skin color can be attributed to differences in genetic predispositions" (Keller, 2005). Three students distinguished between features of the body (e.g., skin color, hair texture, motor skills), which they attributed to genes, and psychological features (e.g., intelligence, temperament, behaviors), which they attributed to cultural influences and upbringing. We also observed the mind/body split at another point in the interviews, when students were asked about the role of genes. Five of the seven students explicitly distinguished between features of the body and features of the mind. One student, for example, stated: "Well, the behavior and so, I think it's rather not fixed by the genes…But things like the outer appearance are strongly bound to the genes, I guess" (Student 4).

The Role of Nongenetic Factors in High-School Students' Causal Attributions All seven high-school students made frequent reference to nongenetic factors when they argued against the genetic attribution for behavioral and psychological traits in the items of the BGD scale. In particular, the students referred to a person's *education and upbringing*, the *social and cultural environment*, *personal decisions* and *adverse life experiences*. One student, for example, commented on item A9 of the BGD scale ("I believe that many differences between humans of different skin color can be attributed to differences in genetic predispositions"). The

student disagreed with the statement and argued that differences in people's behaviors result from cultural and environmental influences rather than from genetic differences:

> No, I wouldn't say so. Simply because people have different skin colors, they don't behave differently. I think there are vast cultural differences, at least to some degree. Whether the person is from Africa or from Europe. I think the people are from very different cultures; they have a very different past and such things and they grow up in different environments. And perhaps that's how their behavior is formed and not because of their genes. (Student 1)

The same student commented on the role of genes in response to item A2 of the BGD scale ("In my opinion, alcoholism is caused primarily by genetic factors"). The student disagreed with attributing alcoholism primarily to genes and argued instead for the roles of personal decision, behavior, upbringing and social environment:

> For example alcoholism. I don't think that it depends on genes. I met a kid not very long ago, whose father drinks a lot, but he doesn't drink at all. Or he drinks very little. OK, perhaps that's how it goes: when it's the genes, then the kid would also drink a lot, kind of. But I think, it's rather that he sees his father and then he thinks: "No, I'd rather not drink" or something like that. And that's why it depends on the behavior and not on the genes. Like how he is raised, and what he gets to see in his environment, and how he kind of lives his life in the family, and then it's not in the genes but in the upbringing, I believe. (Student 1)

Like the first quote from the same student, this quote illustrates the view that nongenetic factors alone influence the formation of the trait (E-only causal attribution in Fig. 8.1). We observed monocausal environmental attributions in six of the seven students that we interviewed.

Furthermore, we observed considerable between-student variation in their causal attributions. Like student 1, four other students commented on item A2 which attributes alcoholism primarily to genes. Therefore, we report on students' responses to this item to illustrate between-student variation. Like student 1, the students disagreed with the view of primarily attributing genes to alcoholism and stressed the role of nongenetic factors. Student 5 and student 2 attributed alcoholism to personal decision, but differed in whether or not genes have some role in the formation of the trait. Student 3 attributed alcoholism to adverse life experiences alone. Student 4 likewise attributed alcoholism to adverse life experiences, but also mentioned the role of genes. The following quotes further illustrate these differences.

The first quote is from student 5 who described his personal decision not to drink to support his argument against the genetic attribution of alcoholism:

> And item A2 says that the consumption of alcohol is caused by genetic factors. That's definitely not the case because…I want to give a personal example now. Can I describe a personal example? Well, my parents drink a lot and my grandparents drank a lot, too. And I, for example, I have made the decision not to drink at all. I don't drink anything! And I haven't drunk in the past. And that was my personal decision. That's not related to anything genetic…I don't believe that addictions are determined by genes. That's my personal decision. It's what people can decide themselves. (Student 5)

The quote illustrates the view that alcoholism is attributable to personal decision alone (monocausal attribution). Student 2 held a similar view about the role of personal decision. In contrast to student 5, however, student 2 held the belief that genes are involved, although she did not believe that genes were the main cause of alcoholism. Rather, student 2 argued that genes may contribute to making a person more susceptible to alcohol. However, she attributed alcoholism primarily to personal decision, the social environment and the way in which people live their lives:

> That's not true. Because I believe alcoholism and things like drug use, for example, are not caused by one's genetic makeup, or by genetic factors, but people are personally responsible. Perhaps it's also related to the people around you, and the environment, but that's not related to the genes you have, and it's simply how you live your life. It can happen when you live in an environment where people drink a lot of alcohol. But that's not really related to genes. Of course, there are people who are more susceptible to alcohol, who react more strongly to alcohol, and that's in the genes, somehow, but it's not true that alcoholism is caused by that. (Student 2)

Student 3 attributed alcoholism to adverse life experiences (monocausal attribution), but did not mention the role of genes in the formation of the trait:

> I disagree [with A2] because I think that it is personal susceptibility again, somehow. When people receive a blow of fate or something like that they slip into alcoholism. (Student 3)

Similarly, student 4 attributed alcoholism to adverse life experiences. In contrast to student 3, however, this student held the view that the environment is more important than genes:

> As I said, I rather disagree [with the item] because that has to do with this social [thing] again and with behavior, as I said before. Alcoholism…I think it's possible to find genetic factors for alcoholism so that somebody is more inclined to drink. But, I think, usually alcoholism comes from problems, when people try to solve them with alcohol. (Student 4)

Gene Regulation and Gene–Environment Interaction In the interviews, the students often drew on everyday knowledge and personal experience, as well as experience of a person who is close to them, when they commented on the items in the questionnaire. Molecular mechanistic reasoning about the formation of traits was rare. One student, however, used her knowledge about gene regulation to reject the view that health outcomes are determined by genes. She argued that genes can be switched on and off so that possession of a gene is not a predictor of the disease:

> Because, in biology we have learned that genes can be kind of switched on and switched off… and therefore I thought, if a person has a gene [related to the disease] …and because it is present it must not necessarily lead to the onset of the disease. (Student 2)

Although students acknowledged genetic and nongenetic causation, they did not generally address gene–environment interactions. Rather, most students put forth the views that genetic factors and nongenetic factors contribute separately and independently (G | E causal attribution in Fig. 8.1). One student, however, reasoned about the interplay between genes and the environment in the development of breast cancer although he did not use the term "interaction." Talking about the role of

genes in hereditary diseases, the student distinguished between diseases for which the possession of one gene is sufficient for the onset of the condition and others, for which the possession of a gene is only sufficient in combination with further environmental or behavioral factors:

> Because, for some diseases it's enough to have this gene, but for some other [diseases] you must also make some mistakes, I would say or something…like for example smoking in the case of breast cancer…Well, that one gene alone is not enough, but in combination with some other factors it causes the disease. (Student 4)

Although the student did not highlight gene–environment interactions as a general principle, he speculated about interactions between genetic and environmental factors for specific diseases like breast cancer. He did not expand on mechanistic details, however, such as the impact of mutagenic substances on gene regulation, for instance.

At the end of the interviews, we probed the students' understanding of gene–gene and gene–environment interactions by asking them to define "polygenesis" and "gene–environment interaction." Only one student said that he knew the terms, but further questioning revealed a relatively superficial ad-hoc explanation of the terms. When other students attempted to define the terms after we had mentioned them, they used rather vague definitions, as illustrated by the following two statements:

> Actually, it's the way I have explained it, it depends a little bit on the environment and a little bit on the genes. (Student 7)
>
> Gene–environment-interaction?…I think that it is the very thing I have been talking about all the time. Well this "one has genes," but there is also the "environment"…an individual emerges from genes and the environment together. Well a kind of mishmash, I would say. (Student 5)

8.2.5 Summary

We report on quantitative findings (n = 242) and qualitative findings (n = 7) for high-school students' causal attributions of human behavioral and psychological traits. The students tended to reject genetic attributions for behavioral and psychological traits when asked to rate items of the BDG scale (Keller, 2005) and BB scale (Bastian & Haslam, 2006). Both scale means were clearly below the theoretical scale midpoint. Furthermore, when interviewed, students tended to argue from experience and attributed behavioral and psychological traits primarily to education, upbringing, social environment, cultural environment, personal decision and adverse life experiences. In general, the students tended to consider these nongenetic factors to be more important than genes in the formation of behavioral and psychological traits. This qualitative finding explains the lack of endorsement of the BGD scale items observed in the quantitative part of the study. Furthermore, students tended to express the view that nongenetic factors alone cause behavioral and psychological traits. Environmental attributions (E-only in Fig. 8.1) are indicative

of the students' tendency to favor simple patterns of causality over interactive causation. The same is true for monocausal attributions referring to personal decision alone. We observed monocausal attributions by six of the seven students interviewed. Furthermore, interview data revealed that knowledge of gene–environment interaction was lacking; students tended to view the influence of genes and the environment as independent and separate (G | E in Fig. 8.1), and they tended to view the impact of the environment on traits as purely phenotypic. In contrast, there is ample evidence from behavioral geneticists that the formation of complex traits involves multiple genes, multiple environmental factors, and the complex interplay between genes and environment at the different levels of biological organization.

8.3 Remaining Issues

At present, high-school students' causal attributions of human characteristics are an under-researched aspect of biology education research. In particular, little is known about why high-school students rely on monocausal attributions. Motivated by the intention to prevent genetic determinism, researchers have devoted more attention to genetic attributions than to nongenetic ones (environment, personal decision). Research designs for investigating students' causal attributions seem most productive when they leave room for three major causal categories (genes, environment and personal decision) rather than two (genes and environment). Furthermore, such research designs seem most informative when students are encouraged to comment on their causal attributions, and when their ability to reason mechanistically about the interplay between genes and environment is assessed in conjunction with their causal attributions. Interactions between teaching materials, teaching–learning strategies and students' causal attributions of genetic phenomena can be expected, but research-based information about this aspect is lacking. Normative reflections on students' tendency to causally attribute traits to personal decision are presently lacking as well. Such reflections are beyond the scope of this contribution, also because the philosophical debate of free will versus fate has not yet reached the genetics education literature.

8.4 Implications for Teaching

We offer interview findings which show that students argued from experience that complex traits are influenced by the environment. In particular, the students differentiated between different types of environmental influences and considered them to be more important than genetic influences. Genetics educators should build on these intuitions when they attempt to change the present situation of high-school curricula, teaching standards and high-school textbooks emphasizing genetic influences over environmental ones. Genetics educators today are suggesting

frameworks for an integrated understanding of trait formation (e.g. Condit et al., 2009; Haskel-Ittah & Yarden, 2017; Heemann & Hammann, 2020; Pavlova & Kreher, 2013). Such frameworks should include the fundamental insight from behavioral genetics that individual differences have genetic and environmental sources. Students' intuitive causal attributions of complex traits show that this core idea of genetic literacy can be linked with students' own experience.

Our proposal to give genes and the environment equal emphasis builds on Dougherty (2009) who addresses the problem that complex traits are rarely addressed in genetics education, so that students are ill-prepared to participate in public discourse on genetic issues in the twenty-first century. In particular, Dougherty (2009) argues that many students doubt that genetic causation plays a role in the formation of complex traits (e.g., personality, addiction, cardiovascular disease). Dougherty (2009) thus argues that complex traits need to be addressed at the very beginning of genetics instruction, before immersing students in the genetics of rare monogenic traits, to build a conceptual base where multifactorial causation is the norm. We also build on Jamieson and Radick (2017) who suggest a genetics curriculum in which gene–environment interaction is the main message. Those authors suggest introducing students to the concepts of internal and external environments and their impact on gene expression at the beginning of the genetics curriculum. Similarly, Todd et al. (2017) consider gene–environment interactions to be an important concept. They suggest that high-school students should understand that the environment can change the type and amount of proteins that influence cell functions (progression level 5), and that environment can change genes which change proteins, or change gene expression of proteins (progression level 6). Using cancerous cells, the coloring of Siamese cats and the freezing of tree frogs as contexts for teaching gene–environment interactions, they report on significant learning gains for high-school students in a 23-week course (Todd et al., 2017).

Findings from this study foreground the need to prevent monocausal explanations (E-only and G-only explanations in Fig. 8.1) and to foster the students' ability to reason mechanistically about gene–environment interactions. In the interviews that we conducted, most students understood the environment to be social and cultural. This made it easy for them to describe the *phenotypic* impact of the environment on traits. No student, however, described the *molecular* impact, at the level of proteins and genes. Hence, students were not aware of the fact that environmental factors "get under the skin," and they did not reason mechanistically about the impact of the environment at the molecular level (see Haskel-Ittah & Yarden, 2017, 2018 for molecular mechanistic reasoning in genetics). Thus, as a main educational implication, genetics educators need to teach about the complex interplay between genes and environment at the different levels of biological organization.

To link bodies and environments, genetics educators need to enact the molecular imperative (Darling et al., 2016). This term is used to redefine psychological traits and behaviors in terms of their molecular components and describe them in the language of molecular biology. Essentially, bodies can be seen as environments in and of themselves to study the interplay between an individual's physical and chemical exposure to the external environment and its impact on the internal

environment. Furthermore, bodies can be seen as the materialization of social experiences to study the interplay between social phenomena, such as discrimination, and the molecular level. Therefore, for high-school education, the challenge lies in interconnecting the different levels of biological organization (see also Duncan & Reiser, 2007), for the interplay between genes and the environment as well. For example, biology educators can enact the molecular imperative by teaching that, in stressful situations, the adrenal glands produce the hormone cortisol, which passes through the plasma membrane of target cells and forms a hormone–receptor complex. This complex then moves into the nucleus and acts as a transcription factor binding to specific genes and activating their transcription. This eventually leads to proteins which aid in elevating glucose levels in the blood, helping the organism meet the demands of the stressful situation.

In the interviews, some students were not aware of the fact that complex human traits have a genetic basis and attributed traits to the environment alone. In such cases, environmental attributions were associated with the belief that genes do not contribute to the formation of the traits. Furthermore, the students tended to hold the view that genes and the environment have separate and independent impacts. In general, such beliefs result from lack of knowledge of the different types of gene–environment interplay and the inability to reason mechanistically about them at the different levels of biological organization. Frameworks for an integrated understanding of genetics should therefore address genetics from the perspective of interaction, providing not only one, but several sources of variation while emphasizing the range of causal relationships between these different factors.

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Chapter 9
How are High-School Students' Teleological and Essentialist Conceptions Expressed in the Context of Genetics and What Can Teachers Do to Address Them?

Florian Stern, Kostas Kampourakis, Marine Delaval, and Andreas Müller

9.1 The Problem

Genetics has long been reported to be a challenging topic to learn and teach, and researchers in genetics education have documented a wide array of genetics-related misconceptions. A recent literature review suggests that these misconceptions can be related to the nature of the genetic material, the role of genes, and the nature or potential of genetic technologies. Most interestingly, there are multiple origins for such misconceptions, such as the inherent difficulty of the topic, the content of formal teaching, the content of the textbooks, the media, and intuitive thinking, such as genetic essentialism (Stern & Kampourakis, 2017). Among these, we decided to focus on the latter and investigate how intuitive conceptions are expressed in the context of genetics. To achieve this, we analyzed students' teleological and essentialist conceptions, which have both been shown to be obstacles to understanding genetics and inheritance (e.g. Heine, 2017; Ware & Gelman, 2014), as well as to

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understanding evolution (Gelman & Rhodes, 2012; Kampourakis, 2014; Kelemen, 2012).

The main argument of this chapter is that in order to teach genetics effectively, it is necessary to explicitly address students' preconceptions. As design teleology and psychological essentialism have been found to be persistent obstacles in understanding biological concepts, especially evolution, we explored the possible expression of these conceptions in the context of genetics. To this end, we defined "genetic teleology" and redefined "genetic essentialism," and developed a questionnaire to measure these conceptions (Stern et al., 2020). In this chapter, we review the relevant previous research, define the constructs of "genetic teleology" and "genetic essentialism," and present results from our study with 396 high-school students.

9.2 Current Knowledge About the Problem

Our knowledge and understanding of the world is formulated in terms of concepts that are mental representations of the world. Scientific concepts provide systematic representations that enable explanations of, and predictions about, phenomena (Nersessian, 2008, p.186). Concepts should be distinguished from conceptions, the latter being the different meanings associated to particular concepts. From our early childhood, we experientially formulate conceptions of the world, which are described as preconceptions. As we grow up, we often assimilate knowledge that further modifies our preconceptions, occasionally turning them into more complex (but often incorrect) conceptions. Thus, new knowledge (e.g., acquired through schooling) does not guarantee a correct understanding of concepts. Preconceptions must be appropriately challenged so that the people who hold them understand that they are incorrect or insufficient. This is what conceptual change is about: the change of conceptions as a result of conceptual conflict toward more accurate concepts (Vosniadou, 2012).

Achieving conceptual change is not an easy task, and it becomes particularly difficult when preconceptions stem from deeply rooted intuitions. These may be strongly held, may often persist into adulthood (Bloom & Weisberg, 2007), and in some cases, are never completely overridden, even by expert knowledge (e.g., Goldberg & Thompson-Schill, 2009; Kelemen et al., 2013). Of course, not all intuitions hinder the learning of scientific concepts; some of them even lead young children to grasp sophisticated correlational and causal patterns (Keil, 2011). However, some intuitions can generate persistent conceptions that may turn into conceptual obstacles: conceptions that are strongly held and resistant to change, and impede the understanding of scientific concepts. Two such intuitions are psychological essentialism and design teleology.

9.2.1 Genetic Teleology

Teleology is defined as the tendency to believe that an organism's traits exist to fulfill a goal. Design teleology is the belief that such characters have been designed by some external entity to fulfill that goal (Lennox & Kampourakis, 2013). Other conceptualizations of teleology exist; for example, some authors have suggested distinguishing three levels of teleology in explanations for a phenomenon or process: "basic function-based"; "basic need-based"; "elaborated need-based." (Kelemen, 2012).

A great deal of research suggests that people tend to intuitively provide teleological explanations of this kind for the characters of organisms from a very early age. One body of research suggests that children provide teleological explanations for both organisms and artifacts, but in a discriminative manner, as they are able to perceive the differences between them (e.g., the thorns of a rose exist to protect it, whereas the barbs of barbed wire exist to protect something that is valuable to humans) (Keil, 1992, 1994, 1995). Another body of research suggests that children provide teleological explanations in a non-discriminative manner for organisms, artifacts and non-living natural objects (e.g., pets exist for loving; clocks exist for telling time; clouds exist for raining) (Kelemen, 1999a, b, c). Recent research suggests that 7- to 8-year-old children exhibit discriminative teleology (Kampourakis et al., 2012b), but a shift from non-discriminative to discriminative teleology may also be possible at as early as 5 years of age (Kampourakis et al., 2012a). Whatever the answer, there is general agreement that children tend to intuitively provide teleological explanations for the characters of organisms. These may rely on underlying artifact thinking, because children seem to perceive the parts of organisms in the same way as the parts of human-made artifacts. Teleology has thus been shown to be a conceptual obstacle to understanding evolution (Kampourakis, 2014; Kampourakis & Zogza, 2008, 2009; Kelemen, 2012).

It is important to note that teleology in general is not illegitimate in biology. Explanations based on natural selection exhibit a robust form of teleology (Lennox, 1993), and it is legitimate to state that something exists for a purpose because it was selected in order to fulfill it: this can be described as selective or selection teleology (Lennox & Kampourakis, 2013). Rather, what is illegitimate is design teleology, the idea that something exists for a purpose because it was intentionally designed to fulfill it. This idea entails that entities have the features that they need for the roles or functions they were intended to fulfill; this is the case for artifacts but not for organisms. Therefore, what is rejected in biology is not the idea of teleology in general, but the idea of intended uses that are the outcome of design, which we describe as design teleology. In this sense, we can distinguish between three types of teleology, which are presented in Table 9.1 (Kampourakis, 2020).

Given these considerations, one might assume that the notions of design and purpose could make students intuitively think that there exist "genes for" traits, in the sense that genes exist for an intended use or purpose. Previous research has shown how this can be possible. For instance, a study found that undergraduates

Table 9.1 The main features of design and selection teleology

Types of teleology	Consequence etiology	Assumption of design
Design teleology (external)	Something exists because of its consequences that contribute to the fulfillment of an external agent's intention to achieve a goal	Yes (it is explicit as there is reference to the intentions of an external agent)
Design teleology (internal)	Something exists because of its consequences that fulfill the intentions/needs of its possessor	Yes (it is implicit as there is reference to the intentions/ needs of the organism itself)
Selection teleology	Something exists because of its consequences that contribute to the well-being of its possessor, and it is thus favored by natural selection	No

From Kampourakis (2020)

Table 9.2 The main features of genetic teleology

Types of teleology	Consequence etiology	Assumption of design
Design teleology (external)	A particular gene exists because of its consequences that contribute to the fulfillment of an external agent's intention to achieve a goal	Humans have opposable thumbs. Thus, genes associated with opposable thumbs have been designed for several roles such as holding objects.
Design teleology (internal)	A particular gene exists because of its consequences that fulfill the intentions/needs of its possessor	Humans have opposable thumbs. Thus, genes associated with opposable thumbs have appeared for satisfying several needs such as holding objects.
Selection teleology	A particular gene exists because of its consequences that contribute to the well-being of its possessor, and it is thus favored by natural selection	Humans have opposable thumbs. Thus, genes associated with opposable thumbs have appeared by chance and were selected for several effects such as holding objects.

Kampourakis (2020); example from Stern et al. (2020)

used purpose-based reasoning to explain properties that have particular consequences. Although they were generally inclined to think of physical properties as inherited and stable, they explained those properties that performed some function or were useful in a particular habitat differently. That study concluded that essentialist and teleological conceptions about inheritance are contrasting: traits with some function are either more heritable and less modifiable (essentialist stance) or less heritable and more modifiable (purpose-based stance) (Ware & Gelman, 2014).

In the present study, we wanted to investigate whether students think that genes are designed/needed for a particular use or are selected for it. We therefore defined "genetic teleology" as the conception that genes exist for some purpose. The three different types of genetic teleology, with examples, are presented in Table 9.2.

9.2.2 Genetic Essentialism

Essentialism refers to the belief that an entity has an essence, in other words, it has underlying characteristics that make it what it is (Wilkins, 2013). Psychological essentialism is defined as the intuition that organisms have fixed essences (Gelman, 2003). Several research findings support this conclusion. First, children seem to believe that organisms belonging to the same taxonomic group share some underlying, non-visible properties, and they rely on those properties to draw inferences about the characteristics of organisms (Gelman & Markman, 1986, 1987). Second, children tend to think that the group to which an organism belongs does not change, despite possible changes in its appearance (Keil, 1989, pp. 197–215). Third, children seem to consider internal, invisible features and properties to be more important that external ones (Gelman & Wellman, 1991). Fourth, children believe that organisms can undergo radical, developmental changes with no change in their identity (Rosengren et al., 1991). Thus, children seem to believe that organisms are characterized by underlying, distinctive "essences" that make them what they are. These "essences" are unchangeable and so they characterize organisms despite any superficial changes they may undergo. Essentialism has been studied as a conceptual obstacle to understanding evolution (Gelman & Rhodes, 2012; Kampourakis, 2014; Shtulman, 2006; Shtulman & Schulz, 2008).

Interestingly, genes seem to be perceived as perfect placeholders for essences (as expressed by the popular sentences: "it is in your genes", or "it is in your DNA"), and several studies have therefore analyzed the potential links between essentialism and genetics. Genetic essentialism has been suggested to be the propensity to draw inferences from one's characteristics based on one's perceived genes. There exists more fine-grained conceptualizations of genetic essentialism, and one of them supports its consisting of four dimensions: it may lead people to believe that genetically influenced traits are immutable, that genes are the main cause for a given trait or behavior, that groups sharing a genetic character are homogeneous, and that traits or behavior are more natural if they are genetically determined (Dar-Nimrod & Heine, 2011). It has also been found that people tend to implicitly (and thus unconsciously) associate genes with fate concepts (Gould & Heine, 2012). Overall, genes seem to be perceived as our essences, which determine who we are and how we behave (Cheung et al., 2014; Heine, 2017).

A problem with the aforementioned conceptualization is that two of the four dimensions of genetic essentialism can actually be considered dimensions of genetic determinism. The difference between the two is subtle but important. Genetic essentialism is the idea that genes are fixed entities, which are transferred unchanged across generations and are the essence of what we are by specifying characteristics from which their existence can be inferred. Genetic determinism is the idea that genes invariably determine characteristics, so that the outcomes are just a little, or not at all, affected by changes in the environment or by the different environments in which individuals live (Kampourakis, 2017, p. 6). Genetic determinism thus focuses on how the characteristics are caused, considering the environment as

unimportant, whereas essentialism focuses more on the properties of the genes themselves and the implications of those properties. Given these considerations, we decided to focus only on two dimensions of essentialism, which are clearly distinct from genetic determinism and which are presented in Tables 9.3 and 9.4. The fixity of genes is about whether all, some or no genes can change to something else. A strong essentialist view would be that genes are fixed and cannot change, whereas the correct view is that no genes are fixed and that all genes can undergo changes. The group-specificity of genes is about whether all, some or a few genes found in a group are specific to it. A strong essentialist view is that all genes are group-specific, that is, found in particular groups only, but not in others. The correct view is that only a few genes can be considered group-specific.

9.2.3 Measuring Students' Genetic Teleological and Genetic Essentialist Conceptions

It should be noted that many of the studies cited above included children and adults. There have also been studies of teleological and essentialist conceptions focusing only on adults, showing that they may exhibit the same biases as children when it comes to teleological explanations (e.g., Kelemen & Rosset, 2009), as well as a coherent essentialist reasoning (Meyer et al., 2013). Based on this research, when students are presented with information about the role of genes and environments in relation to simple or even complex traits, psychological essentialism and/or design teleology may favor the accommodation of explanations according to which single

Table 9.3 The main features of genetic essentialism – immutability

Types of genetic essentialism (Fixity of genes)	Example
All genes are fixed	A woman with breast cancer has a tumor in this organ. We assume that in a given family no woman has a breast cancer. Therefore, there are only genes associated with a 'normal breast'. A descendant in this family will have unaffected breasts, because the genes associated with a 'normal breast' always remain fixed.
Some genes are fixed	A woman with breast cancer has a tumor in this organ. We assume that in a given family no woman has a breast cancer. Therefore, there are only genes associated with a 'normal breast'. A descendant in this family will have unaffected breasts, because the genes associated with a 'normal breast' are fixed, even though others may change.
No genes are fixed	A woman with breast cancer has a tumor in this organ. We assume that in a given family no woman has a breast cancer. Therefore, there are only genes associated with a 'normal breast'. A descendant in this family may have breast cancer, if the genes associated with a 'normal breast' change into genes associated with breast cancer.

Example from Stern et al. (2020)

Table 9.4 The main features of genetic essentialism – homogeneity

Types of genetic essentialism (Homogeneity of groups based on Genes)	Example
All genes are group-specific	If we analyze the genes of chimpanzees, we will identify genes specific to them.
Some genes are group-specific	If we analyze the genes of chimpanzees, we will identify many genes different from ours.
Few genes are group-specific	If we analyze the genes of chimpanzees, we will identify few genes different from ours.

Example from Stern et al. (2020)

Table 9.5 Summary of the investigated constructs

Construct	Subconstruct
Genetic teleology (GT)	explanations about past outcomes
	predictions of future outcomes
Genetic essentialism (GE)	homogeneity
	fixity

genes determine these traits. This is because single genes are considered "essences" that are, by definition, unique, hidden and fixed (genetic essentialism) and that exist for a purpose or intended use, namely to determine those traits (genetic teleology).

Several instruments have been designed to measure students' general knowledge and understanding of genetics at the undergraduate level, such as the Genetics Concept Assessment (Smith et al., 2008), the Genetics Literacy Assessment instrument (Bowling et al., 2008), and the Public Understanding of Genetics and Genomics questionnaire (Carver et al., 2017). In addition, a test of genetics understanding at high-school level was developed (Tsui & Treagust, 2010). Finally, in relation to our research, another questionnaire addressing teleological, essentialist and anthropocentric thinking to explain biological phenomena has been published (Coley & Tanner, 2015). We therefore developed a new questionnaire (Stern et al., 2020), with the aim of measuring high-school students' teleological and essentialist conceptions in the context of genetics (and this is why we respectively refer to them as genetic teleological and genetic essentialist conceptions). To the best of our knowledge, this is the first instrument designed to investigate these specific conceptions.

Based on the development of the genetic essentialism and teleology (GET) questionnaire (Stern et al., 2020), and on theoretical considerations about teleology (Kampourakis, 2020) and essentialism (Heine, 2017; Heine et al., 2017), we considered the constructs and subconstructs detailed in Table 9.5. The subconstructs for genetic essentialism are two of those that have been previously investigated in research: homogeneity of populations as far as their genes are concerned and fixity of genes across generations. These are two distinct and very different dimensions of essentialism. The subconstructs for genetic teleology are also distinct, and they differ in whether they are explanations for past outcomes or predictions of future outcomes.

In the following, we present our use of the GET questionnaire to understand whether high-school students exhibit genetic teleological and genetic essentialist conceptions.

9.2.4 Genetic Teleology and Genetic Essentialism in High-School Students

9.2.4.1 Method

Our study included 396 students, 15–18 years of age (M = 16.5, SD = 0.90), 232 females and 164 males, from nine different high-schools in Geneva, Switzerland, plus one school in France, in a neighbourhood near Geneva. According to the International Standard Classification of Education of UNESCO, these schools belong to ISCED level 34 called "upper secondary general education" (Unesco, 2012; Office fédéral de la statistique, 2015). Our study was conducted in French. A summary of the sample characteristics can be found in Table 9.6.

We developed 10 genetic teleological (GT) items about several human traits, and these were divided into two subcategories. In the first category, there were five items about genes associated with traits that are the result of *past* evolutionary processes: opposable thumbs (GT1), big brain (GT2), bipedalism (GT3), communication (GT4), and sociability (GT5). In the second category, there were five more items about genes associated with traits that could be the result of hypothetical *future* evolutionary processes: cellulose digestion (GT6), antibody production (GT7), protection from ultraviolet radiation (GT8), protection from high heat (GT9), and protection from radiation (GT10). To explain the origin of these traits or abilities, students could choose between three answers, which were assumed to represent the following conceptions: (1) design-based teleology; (2) need-based teleology; (3) natural teleology. Natural teleology is a kind of teleology which is based on natural selection and does not utilize design arguments. Therefore, these three answers were associated with *strong*, *moderate* and *weak* design intuition, respectively, and we considered the third answer to be correct, as natural selection is what best explains adaptations.

Finally, we developed 10 genetic essentialism (GE) items about several human traits, which were also divided into two subcategories. In the first category, there were five items about the *homogeneity* of genes among Europeans when compared

Table 9.6 Summary of the sample characteristics. Entries are number of students

Gender	Male	164
	Female	232
Age	15 year	67
	16 year	107
	17 year	180
	18 year	42

to the following groups: Neanderthals (GE1), Chinese people (GE2), chimpanzees (GE3), Eskimos (GE4), and baboons (GE5). In this case, students could choose between three answers, which were assumed to represent the following conceptions: (1) existence of group-specific genes; (2) large genetic differences between the compared groups; (3) small genetic differences between the compared groups. In the second category, there were five more items about the *fixity* of genes in several conditions: daltonism (GE6), breast cancer (GE7), dwarfism (GE8), diabetes (GE9), and Alzheimer's disease (GE10). In this case, students could choose between three answers, which were assumed to represent the following conceptions: (1) all genes are fixed entities; (2) some genes are fixed entities; some others are changeable; (3) all genes are changeable entities. In both cases, answers (1), (2) and (3) were respectively associated with *strong*, *moderate* and *weak* genetic essentialist intuition, and we considered the third answer to be the correct one.

The development and validation of all of these items through interviews and several pilot studies are extensively described in Stern et al. (2020). Eventually, the 10 genetic teleology and 10 genetic essentialism items were included in the GET questionnaire. The list of all genetic teleology and genetic essentialism items used can be found in Stern et al. (2020).

All calculations and graphs were computed with the R software. For each subconstruct (genetic teleology-*past*, genetic teleology-*future*, genetic essentialism-*homogeneity*, genetic essentialism-*fixity*), percentages of students' answers were displayed through histograms.

9.2.4.2 Results

According to Fig. 9.1a, the overall rate of design teleological and need-based teleological misconceptions for past processes decreased from students aged 15 years (77%) to students aged 18 years (26%). Furthermore, across the age groups, the rate of need-based teleological conceptions was on average twice higher (between 21% and 45%) than that of design teleological conceptions (between 5% and 32%). According to Fig. 9.1b, the overall rate of design teleological and need-based teleological misconceptions for future processes decreased from students aged 15 years (67%) to students aged 18 years (21%). Furthermore, across the age groups, the rate of need-based teleological conceptions was overall similar (between 13% and 32%) to that of design teleological conceptions (between 7% and 35%). Accordingly, we also see that the amount of responses in favor of natural selection increased with age, by a factor of 5–7 from students aged 15 years (10% and 14% for *past* and *future* processes, respectively) to students aged 18 years (69% and 72% for *past* and *future* processes, respectively).

According to Fig. 9.2a, the overall rate of misconceptions (psychological and moderate essentialist conceptions considered together) related to the homogeneity of genes was overall rather stable across the different ages (between 33% and 36%). Furthermore, across the age groups, the rate of psychological essentialist conceptions was on average slightly higher (between 18% and 22%) than that of moderate

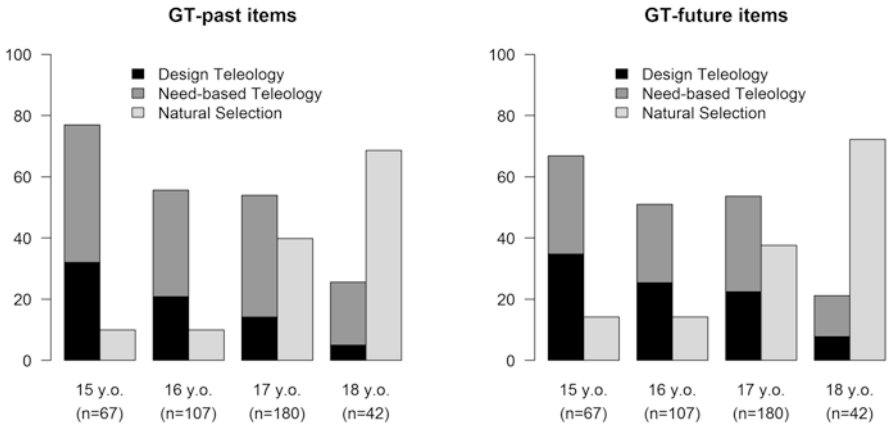

Fig. 9.1 Average of students' answers, in percent, to: (**a**) genetic teleology-*past* items; and (**b**) genetic teleology-*future* items. The misconceptions "design teleology" and "need-based teleology" (in black and dark gray, respectively) are stacked and are contrasted with the correct one, "natural selection" (light gray)

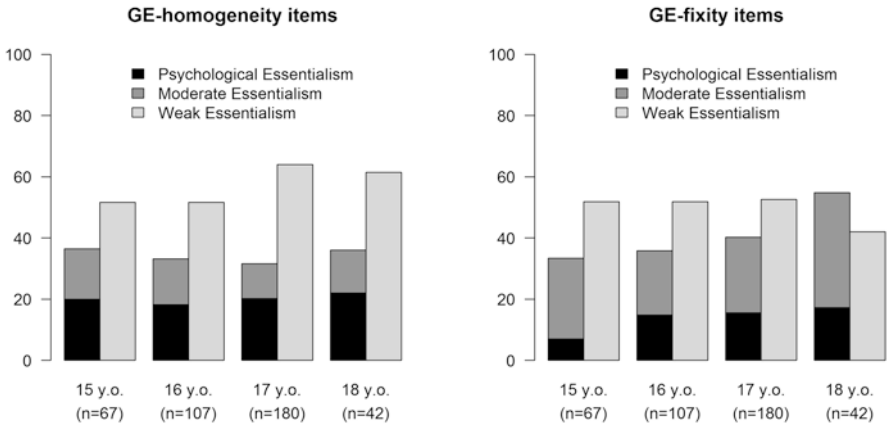

Fig. 9.2 Average of students' answers, in percent, to: (**a**) genetic essentialism-*homogeneity* items; and (**b**) genetic essentialism-*fixity* items. The misconceptions "psychological essentialism" and "moderate essentialism" (in black and dark gray, respectively) are stacked and are contrasted with the correct one, "weak essentialism" (light gray)

essentialist conceptions (between 11% and 14%). According to Fig. 9.2b, the overall rate of misconceptions (psychological and moderate essentialist conceptions considered together) related to the fixity of genes increased from students aged 15 years (33%) to students aged 18 years (55%). Furthermore, across the different age groups, the rate of moderate essentialist conceptions was on average higher (between 21% and 38%) than that of psychological essentialist conceptions (between 7% and 17%). Accordingly, we also see that the amount of responses

related to weak essentialism was overall rather stable across the age groups (between 52% and 64%, and between 42% and 53% for *homogeneity* and *fixity* items, respectively).

9.2.4.3 Discussion

The major result from our analysis was that, genetic teleological and genetic essentialist misconceptions are common among high-school students, as these were found for all investigated constructs and among all age groups.

In sum, the genetic teleological conceptions seem to be addressed in high-school education, whereas this does not seem to be the case for the genetic essentialist conceptions. For now, we can only assume that formal teaching at the high-school level includes topics relevant to natural selection, because we see that the genetic teleological conceptions decrease with age. Accordingly, as the conceptions of group-homogeneity and fixity of genes do not increase with age, we can assume that they are not explicitly addressed during formal teaching. Unfortunately, as the curriculum is flexible, and teachers are free to select what and how to teach, there is no systematic knowledge about what the students participating in our study were taught. To better understand these results, it would therefore be necessary to observe teachers and document if and how they address or do not address these conceptions.

Our interpretation of the results is constrained by some limitations. First, some of the sample sizes could have been larger for some ages (N = 67 students aged 15 years and N = 42 students aged 18 years) to obtain more accurate results. Second, the study was conducted in French, therefore our results should be cross-validated in a non-French speaking sample. Overall, we gathered evidence, in the context of genetics, for the persistence of essentialist conceptions at high-school level, in contrast to teleological ones. Students' conceptions and their change could be investigated more systematically with a longitudinal study following the same cohort of students through high-school education, or through an intervention study including pre- tests and post-tests.

9.3 Remaining Issues

Even though our findings do not support generalizations, genetics teaching could benefit from addressing students' essentialist conceptions. Assuming that teaching and learning about natural selection helps to address students' teleological conceptions, as our results seem to indicate, the question is what can be done about essentialism? The two dimensions that we investigated in our study, homogeneity and fixity, could be addressed in various ways during the teaching of biology. This would require two major changes: in the way biology is taught and in the way textbooks address these issues.

9.4 Implications for Teaching

Beginning with fixity, a major aim of genetics education would be to discuss molecular mechanisms in more detail, so that students could become better aware of the various phenomena that can bring about changes in DNA sequences. The mutation rate in humans is estimated at $1.4–2.3 \times 10^{-8}$ mutations per nucleotide per generation (Sun et al., 2012). Details notwithstanding, and given that the human genome is estimated to consist of 6×10^9 (six billion) nucleotides, these data suggest that any individual is likely to carry at least 72 new mutations ($1.2 \times 10^{-8} \times 6 \times 10^9 = 72$). These mutations are of course rare, but it is still possible to cause important phenotypic changes. One characteristic example is achondroplasia, a form of short-limb dwarfism in humans. Whereas in more than 95% of the cases the disease is due to the same single-nucleotide mutation in the *FGFR3* (fibroblast growth factor receptor 3) gene on chromosome 4, more than 80% of these cases are due to new mutations, most often in the father (Horton et al., 2007). Another example is human cancers, most of which are caused by two to eight sequential mutations that occur over the course of 20–30 years in approximately 140 genes. These genes affect several signaling pathways that regulate three important cellular processes: cell-fate determination, cell survival, and genome maintenance. The pathways affected in different tumors are similar, but the mutations in each individual tumor are different (Vogelstein et al., 2013). These are important findings that should be properly included and discussed in high-school education to address the essentialist view that genes are always transmitted unchanged across generations or during one's lifetime.

Turning to homogeneity, teachers would have to emphasize the idea that populations consist of individuals that differ from one another and that exhibit significant phenotypic and genetic variation, which in turn can have significant ecological effects (Bolnick et al., 2011). Therefore, it is important that students become familiar with this variation from a very early age. Teachers should thus refrain from referring to exemplars, e.g., the bear, the oak tree, and instead make reference to populations of bears, oak trees, etc. Furthermore, they should emphasize the phenomena that bring about this variation. Genetic variation is, of course, the result of the mutations discussed above. However, mutations do not only bring about disease; they also -and most significantly- result in new genetic variation in the population. Because of this, and also because of the complexities of development, new phenotypic variation is also possible. In fact, development actually has two distinct and complementary aspects: robustness and plasticity. Developmental robustness is the capacity of individuals of the same species to exhibit the general characteristics of their species irrespective of the environment in which they live, thus resulting in consistency of phenotype in different environments. This is the feature that confers the tendency to think in essentialist terms about species, as we prioritize the common features of individuals belonging to a same species. However, there is also developmental plasticity: the capacity of individuals of the same species with the same genotype to exhibit phenotypic variation, and thus to produce different phenotypes during development in response to local environmental conditions (Bateson & Glucksmann, 2011, pp. 4–5, 8).

These are important phenomena that should be explicitly discussed in high-school genetics teaching as they might help address students' essentialist conceptions. How this can be done effectively is an open question that requires further research. This knowledge should be properly reframed for inclusion in schools. Most importantly, teachers should be aware that this knowledge is not to be presented to students only for its own sake, but also as a tool to address their essentialist conceptions. However, this requires that teachers possess both the required content knowledge and the respective pedagogical content knowledge.

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Chapter 10
How Can We Make Genetics Education More Humane?

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10.1 The Problem

Throughout history, racist policies have been justified through "genetic essentialism" (Jackson & Depew, 2017). Psychologists (Dar-Nimrod & Heine, 2011) define genetic essentialism as the belief that racial groups have different genes that cause them to differ cognitively and/or behaviorally. Consequently, genetic essentialists believe that complex traits are little influenced by the social environment (Dar-Nimrod & Heine, 2011). They therefore claim that efforts to redress racial inequality are futile because it is caused by genetic differences that are immutable (Lynch et al., 2018). This makes genetic essentialists prone to the "naturalistic fallacy", which is the belief that racial disparities are natural and need not be eliminated (Lynch et al., 2018).

Institutional racism in the United States (US) and in Europe was built upon an essentialist world view, and the inequality created through racism is still augmented by belief in genetic essentialism today (Jackson Jr. & Depew, 2017; Morning, 2011; Omi & Winant, 1994). For example, a genetic essentialist world view is apparent in the Nazi justification of the Holocaust and the segregationist justification of Jim Crow segregation (Jackson & Depew, 2017). At least one-fifth of non-black Americans still believe that economic disparities between races are caused by genetic differences between races (Morning et al., 2019). Unsurprisingly, then, belief in genetic essentialism still predicts opposition to racially ameliorative policies in white (Byrd & Ray, 2015) and non-white adults in the US (Soylu Yalcinkaya et al., 2017).

Yet, genetic essentialism could not be a more genetically flawed world view (Ereshefsky, 2010; Jackson & Depew, 2017; Mayr, 1982). Twenty-first century

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genomics research (Graves, 2015; Rosenberg et al., 2018) and mid-twentieth century population genetics (Jackson & Depew, 2017) have both revealed that the genetic essentialist world view overestimates the amount of genetic differentiation between human groups, as well as the amount of cognitive and behavioral variation attributable to genes (see Table 10.2). Despite the danger of genetic essentialism and its scientific flaws, genetics education does little, if anything, to challenge it (Donovan, 2015b; Jamieson & Radick, 2013; Stern & Kampourakis, 2017). Worse than that, it may perpetuate it (Donovan, 2014, 2016, 2017; Donovan et al., 2019b).

In this chapter, we review theory and research that describe how genetics education can both perpetuate and prevent the development of genetic essentialism of race[1]. We advance the argument that genetics educators can help prevent the development of genetic essentialism by offering youth a more humane genetics curriculum; one that helps students understand the flaws and consequences of justifying prejudice with genes.

10.2 Current Knowledge About the Problem

Genetic essentialism is one manifestation of a social-cognitive bias termed psychological essentialism (Dar-Nimrod & Heine, 2011). Psychological essentialism is a bias that varies between individuals on a continuum (Haslam et al., 2000). In Western, Educated, Industrialized, Rich, Democracies—the so-called WEIRD societies (Henrich et al., 2010)–genetic essentialism is widespread due to the prevalence of the gene concept in technoscientific culture (Nelkin & Lindee, 2004). Although there is cross-cultural variation in the proportion of people who exhibit essentialism (Diesendruck et al., 2013), it is prevalent in both WEIRD and non-WEIRD societies at a non-zero percentage (Henrich et al., 2010).

Psychological essentialism develops early in humans (Cimpian & Salomon, 2014; Gelman, 2004), and its onset is associated with the development of interethnic bias in children and adolescents (Bigler & Liben, 2007; Diesendruck & Menahem, 2015; Pauker et al., n.d.). Studies show that essentialist beliefs about race increase as children progress through school (Pauker et al., 2010). As children mature in US schools, their belief in essentialism becomes a stronger predictor of their tendency to stereotype (Pauker et al., 2010). Several studies have found that essentialism interacts with factors in school environments to influence the development of racial stereotyping (Bigler & Liben, 2007). Genetics education is one such factor.

[1] Those interested in the interplay of genetics education and genetic essentialism of gender are referred to Donovan, et al. (2019).

10.2.1 The Role of Genetics Education in the Development of Genetic Essentialism

Genetic essentialism theory (Dar-Nimrod & Heine, 2011) contends that exposure to genetic information affects belief in genetic essentialism through its impact on how individuals perceive the relationship between genes and traits. Information that leads learners to believe that there is a specific, proximate, stable and immutable relationship between a gene and a trait tends to increase belief in genetic essentialism through a mechanism based in causal reasoning (Lynch et al., 2018). Information that leads learners to believe that individuals of the same group are genetically uniform and that different groups are genetically discrete increases belief in genetic essentialism through a mechanism based in social categorization (Lynch et al., 2018). We now unpack how the genetics curriculum affects the belief in genetic essentialism through these two mechanisms.

Causal Reasoning Genetics education is designed to affect causal reasoning about genes. However, there can also be unintended effects on the belief in genetic essentialism. For instance, Mendelian genetics education has been criticized for leading students to develop a model of inheritance in which human traits are determined by genes (Jamieson & Radick, 2013; Lawson & Thompson, 1988; Shaw et al., 2008; Venville et al., 2005) with no molecular mechanism to separate gene and trait (Duncan et al., 2016; Duncan et al., 2009). This "gene for" model implies a *specific* causal relationship between gene and trait, because the gene is believed to be the only cause of the trait (Lynch et al., 2018). Since students learn that genes are located within the nuclei of cells, and since they rarely learn how the environment moderates gene expression (Jamieson & Radick, 2013; Stern & Kampourakis, 2017), students are at risk of believing that genes are a more *proximate* and *stable* cause of trait variation than any social/environmental factors operating outside of the body (Lynch et al., 2018). Proximity and stability beliefs can then make students believe that human traits are *immutable* and *predetermined*, thus reinforcing their belief in genetic essentialism (Lynch et al., 2018). These changes may then lead individuals to adjust their belief in gene-determined causes for novel human traits through a process called genetic interpolation (Morin-Chassé, 2014).

Social Categorization Genetics education can also affect belief in genetic essentialism through its impacts on social categorization. It is estimated that 90% of biology textbooks discuss racial differences in genetic disease prevalence (Morning, 2008; Willinsky, 2020). Students usually learn that sickle-cell anemia is common among African-Americans and that cystic fibrosis is common among "Caucasians" (Donovan, 2015b; Morning, 2008). In other words, they often learn that there is a "gene for" sickle-cell anemia in "black" people and a "gene for" cystic fibrosis in "white" people. Since the Mendelian curriculum rarely explains the prevalence of both diseases in other ethnic or racial groups (Donovan, 2015b), the curriculum also implies that racial groups are genetically *discrete* and that individuals of the same

group are genetically *uniform*. This, in turn, implies that each race has a different genetic essence that is *stable* across contexts, *immutable* over time, and unaffected by the environment. Because of these implications, students may believe that any trait variation between social groups is best explained by *discrete* genetic differences between groups and the genetic *uniformity* within any single group, thereby increasing their belief in genetic essentialism.

Evidence for Hypotheses Mounting evidence from studies in different countries suggests that an early emphasis in the biology curriculum on Mendelian genetics—where environmental factors are ignored—contributes to the development of belief in genetic essentialism through this causal reasoning mechanism (Clément & Castéra, 2013; dos Santos et al., 2012; Jamieson & Radick, 2013; Parrott & Smith, 2014; Stern & Kampourakis, 2017). For example, Dougherty et al. (2011) showed that the genetics standards in virtually every US state omit concepts related to genetic complexity, the importance of the environment to phenotypic variation, and differential gene expression. Deterministic gene concepts have also been documented in Brazilian, French, and British textbooks (Clément & Castéra, 2013; dos Santos et al., 2012; Jamieson & Radick, 2013). Studies suggest that such textbook representations of the gene could influence belief in genetic essentialism. For example, experiments have shown that the blueprint metaphor for DNA used in textbooks can cause elevated levels of genetic essentialism in adults who read these texts (Parrott & Smith, 2014). Randomized control trials (RCTs) in samples of undergraduates have also found that reading about population genetics can significantly increase the belief that genes determine complex traits in humans (Keller, 2005). Lynch et al. (2008) also demonstrated that when adults (N = 104) read science texts that include "gene for" language, their belief in genetic essentialism grows.

Table 10.1 reports the findings of three RCTs that are consistent with this social categorization hypothesis. All three were carried out in US biology classrooms. Each of these studies demonstrate that when middle-school and/or high-school biology students learn from a curriculum describing the prevalence of monogenic disorders in different racial groups, it can cause students to believe significantly more in genetic essentialism (Donovan, 2014, 2016, 2017). Furthermore, in the third study from Table 10.1, Donovan (2017) demonstrated that students who learned about genetic diseases with racial terminology (compared to those who did not) increasingly perceived more *discreteness* between racial categories and exhibited greater growth in their belief in genetic essentialism over a 3-month period. On the basis of these findings, Donovan (2017) predicted that, in any given year in the US, roughly 10,594 7th–9th-grade students at racially diverse and majority white schools are at risk of developing greater belief in genetic essentialism of race, because of their exposure to information about racial differences in genetic disease prevalence. If this prediction is accurate, then the genetics curriculum is a risk factor for the development of genetic essentialism of race when it impacts beliefs implicated in social categorization, such as discreteness beliefs. If these mechanisms are correct, then can we reverse them and reduce belief in genetic essentialism?

Table 10.1 Experimental studies demonstrating a cause–effect relationship between the genetics curriculum and genetic essentialism

Study	Design	Sample	Findings
1. Donovan (2014)	Double-blinded RCT: Students read a textbook passage on human genetic diseases. In one condition, the passage included racial terminology (the treatment) and in the other condition, the passage did not include racial terminology (the control).	N = 43 8th graders	Statistically significant effects ($p <$ 0.05) were observed on two genetic essentialism instruments: (i) the race conception scale (RCS) (Cohen's $d =$ 0.47) and (ii) the genetically based racism instrument (GBRI) (Cohen's $d = 0.56$). Students in the racial condition exhibited greater belief in genetic essentialism than students in the nonracial condition.
2. Donovan (2016)	Double-blinded RCT: This study was a direct replication of that in Donovan (2014), using the same design and materials as in the study above.	N = 86 9th graders	Statistically significant effects ($p <$ 0.01) were observed on one genetic essentialism instrument: (i) the GBRI ($d = 0.46$). Students in the racial condition exhibited greater belief in genetic essentialism than students in the nonracial condition.
3. Donovan (2017)	Double-blinded RCT: This study was a conceptual replication of the studies in Donovan (2014) and (2016). Individual students were randomly assigned to learn from either: (i) four text-based lessons discussing racial differences in skeletal structure and the prevalence of genetic disease (racial condition); or (ii) an identical curriculum lacking racial terminology (nonracial condition).	N = 135, 7th–9th graders	Compared to the nonracial condition, students in the racial condition grew more ($p< 0.05$) in: (i) perception of genetic variation between races ($d =$ 0.41); (ii) the GBRI ($d = 0.42$); (iii) disinterest in cross-racial socialization (odds ratio = 1.76); (iv) opposition to policies that reduce racial inequality ($d = 0.37$).

10.2.2 The Potential Role of Genetics Education in the Prevention of Genetic Essentialism

Many understandings from genomic science show that genetic essentialism is a flawed worldview. These ideas are outlined in Table 10.2. Could teaching students these ideas reduce their belief in genetic essentialism?

Causal Reasoning Theoretically, such an outcome is probable. Developing the understanding that the social/external environment interacts with genes to effect complex trait variation means developing a mental model of inheritance in which genes have less *specificity* and *proximity*. Developing this knowledge should then lead students to believe that the relationship between genes and traits is *unstable,* because the effect of the gene is no longer constant across different environments. Since people perceive environmental factors as more changeable than genes

Table 10.2 Understandings from genomic science that refute genetic essentialism

Idea	Description	Supportive scientific evidence
1	Most forms of human variation are explained by multifactorial models that include polygenic and social-environmental factors.	Most forms of human variation are not discrete, and they are not explained by a Mendelian model of inheritance (Dougherty, 2009). In nature, even Mendel's peas do not exhibit the discrete forms of variation that are discussed in most biology textbooks (Radick, 2015). In fact, most of the traits that are described as monogenic in biology textbooks are not actually well explained by a biallelic/monogenic model at all (Myths of Human Genetics: Introduction, n.d.). Rather, human variations, especially complex traits, are best explained by multifactorial models of inheritance where variation in a trait is explained by a combination of environmental effects, gene-by-environment interactions, and polygenic effects (Duncan & Keller, 2011; Keller, 2014; MacMahon, 1968). This means that complex human traits are malleable and responsive to the environment (Devlin et al., 1997; Flynn, 1999; Turkheimer et al., 2003). Thus, genes do not have a stable impact on complex traits. Their impact is contingent on the environment.
2	Polygenic inheritance does not equal genetic determinism.	This means that traits like intelligence or educational attainment are influenced by hundreds or thousands of alleles (not just two alleles), each of which has only a tiny effect on the trait in question (23andMe Research Team et al., 2018). For example, in a recent study of 1.1 million people, 1271 alleles were found to be associated with educational attainment. Together, the 1271 alleles explained only 11–13% of the variation in educational attainment (23andMe Research Team et al., 2018). Therefore, while there is clearly some polygenic inheritance associated with educational attainment, it is not completely explained by genes alone. Thus, human traits are not genetically determined, rather they are genetically underdetermined.
3	It is a distortion of the limits of genetic knowledge to claim that racial disparities are simply the result of polygenic variation.	Polygenic contributions to group-level differences in complex traits are predicted to be small, possibly spurious, and dependent on the environment, according to population genetics theory (Rosenberg et al., 2018). In other words, differences between populations can be caused entirely by environmental factors, even when trait differences among individuals within a population are completely inherited (Feldman & Lewontin, 1975). For example, the heritability of skin color among white New Yorkers is high. However, if you compare the skin color of white New Yorkers spending the winter in Florida to those who stay in New York, the average group difference in color has no genetic basis (Feldman & Lewontin, 1975). Similarly, many studies demonstrate that racial disparities are the result of modifiable social factors, such as segregation (Reardon et al., 2019) and discriminatory beliefs and attitudes (Canning et al., 2019; Leslie et al., 2015; Storage et al., 2016).

(continued)

Table 10.2 (continued)

Idea	Description	Supportive scientific evidence
4	People within a group are not uniform, nor are human groups genetically discrete.	Human groups exhibit low levels of genetic differentiation because there is proportionally more genetic variation within human populations (95.7%) than between them (4.3%) (Graves, 2015; Rosenberg et al., 2002). Although these proportions of between and within group variation continue to be studied, a consistent pattern exists across studies in that the vast majority of human variation is found within any single ancestry group (Relethford, 2002).

(Lynch et al., 2018), students who understand multifactorial models of inheritance should believe that complex traits are *malleable* and not genetically *predetermined*. Less belief in the *proximity*, *stability*, *immutability*, and *determinative* power of genes should make genes a poor explanation for social inequalities.

Social Categorization In addition, constructing the understanding that most genetic variation is found within any single ancestry group and that only a small extra amount of genetic variation occurs between ancestry groups (4.3%; Rosenberg et al., 2002) should help students understand that beliefs in, for example, *discreteness* and *uniformity* are flawed. Decreasing these beliefs could, in turn, reduce belief in genetic essentialism.

Evidence for Hypotheses With regards to causal reasoning, Jamieson and Radick (2017) used a non-randomized comparative design in which undergraduates (N = 56) learned genetics from a standard Mendelian curriculum or from a multifactorial curriculum that emphasized the first two ideas in Table 10.2. Students completed pre and post surveys about their endorsement of genetic essentialism. Although students did not differ significantly in their belief in genetic essentialism before treatment, afterwards, the students who received the multifactorial intervention had significantly lower belief in genetic essentialism than those who received the Mendelian curriculum (Cohen's d = 0.6). This finding tentatively suggests that belief in genetic essentialism can be reduced by influencing causal reasoning through the teaching of multifactorial genetics.

A handful of studies have also explored whether genetics education can decrease belief in genetic essentialism by affecting beliefs related to social categorization. In three different RCTs, Donovan et al. (2019a) demonstrated that teaching students about genetic variation within and between US census races can significantly reduce belief in genetic essentialism of race by changing how learners perceive the discreteness of racial categories. In their first study, they randomized 8th- and 9th-grade students (N = 166) into separate classrooms to learn for an entire week about the topics of either: (i) human genetic variation; or (ii) climate variation. They used a climate variation curriculum for the comparison because it controlled for ideologically motivated reasoning and the cognitive difficulty of reasoning about variation. Both treatments used identical instructional frameworks and differed only in

Table 10.3 Conceptual differences between treatments in Donovan et al. (2019a)

	Core ideas taught
Human Variation	1. Scientists do not agree on whether race is biologically real. 2. 99.9% of the DNA between any two humans is identical. When geneticists look at the variable portion of human DNA (0.1%) they find that: (i) 95.7% of differences are between people of the same race (ii) 4.3% of differences are between people of different races 3. Skin color changes continuously as one moves away from the equator. But there is more variation in skin color across races than within races. 4. When people construct arguments about the superiority of one race over another, they tend to overestimate the amount of genetic difference between races.
Climate Variation	1. Scientists agree that the climate is changing. 2. Weather and the climate are different concepts. 3. When scientists support claims about climate change they use data on climate variation and not weather variation. For example, if we look over the last 100 years: (iii) We can see that daily and monthly temperatures and precipitation change – this weather variation occurs within a climate zone and cannot be used to evaluate claims about climate change (iv) But[1], when we look across large land areas and periods of time greater than 30 years, we see a continuous increase in the average temperature and precipitation across the US – this climate variation data can be used to evaluate claims about climate change 4. When people construct arguments about climate change not being real, they tend to incorrectly use evidence about the weather to evaluate claims about climate.

content objectives. Table 10.3 shows the conceptual differences between their interventions.

In the first RCT, Donovan et al. (2019a) demonstrated that students who learned about human genetic variation (compared to the control) had significantly reduced scores on a composite measure of racial bias. This measure included items assessing belief in genetic essentialism of race and belief in racial stereotypes. They then replicated these findings in two more RCTs. One was conducted with adults (N = 176) and another with biology students (N = 721, 9th–12th graders). Importantly, they also demonstrated that learning about human genetic variation caused reductions in the perception that same-race people are uniform *and* reductions in the perception that different-race people are discrete. The reductions in genetic essentialism caused by the intervention were *only* transmitted by changes in how students perceived the discreteness of racial categories. Donovan et al. (2019a) argued that when students develop the understanding that racial groups are genetically alike (but not identical) in their variable DNA, it leads to a reduction in their belief in genetic essentialism by reducing belief in racial discreteness.

Summary Several studies demonstrate that belief in genetic essentialism can be increased or decreased depending on the content learned by students. Underlying this proposition are two distinct mechanisms based in *six beliefs*. One is based in

causal reasoning and has to do with beliefs about how *proximate*, *stable*, *immutable*, and *determinative* the relationship is between genes and traits. The other is based in social categorization and has to do with beliefs about the biological *uniformity* of a group and the biological *discreteness* of different groups. Content in the genetics curriculum that strengthens these six beliefs may increase belief in genetic essentialism. Content that weakens these beliefs may reduce it.

10.3 Remaining Issues

If these hypotheses are correct, then they raise many unanswered questions for researchers interested in the interplay of genetics education and the development of genetic essentialism, such as: How does genetics knowledge moderate the relationship between genetics education and genetic essentialism? What role do teachers play in this process? What sociocultural factors enable or impede the relationship between genetics education and belief in genetic essentialism? Below we describe some untested hypotheses that speak to these questions.

10.3.1 The Genetics Knowledge Hypothesis

Genetics is difficult to learn because it requires students to reason across different ontological levels of biological organization to understand how sequence variation in DNA is encoded in proteins, thereby affecting tissue and organ formation, and ultimately population-level trait variation (Duncan, 2007; Duncan & Reiser, 2007; Duncan et al., 2009). Studies suggest that students conflate genes with traits in a deterministic manner because biology education is ineffective at helping students understand that, while genes do encode proteins within cells, they do not directly encode behavioral or social-cognitive traits (Lewis & Kattmann, 2004). Therefore, genetics education may inadvertently contribute to belief in genetic essentialism through its inability to help students understand the molecular complexities and environmental contingencies of gene expression during development (Jamieson & Radick, 2013). On the flip side, gaining more knowledge of multifactorial genetics could reduce the risk of developing belief in genetic essentialism. Studies have found that possessing more genomics knowledge improves understanding of print and oral communications about genomic information (Lea et al., 2011). Since domain-specific prior knowledge allows one to construct more meaning from science texts (Ozuru et al., 2009; Pearson et al., 1979; Tarchi, 2010), students who gain more knowledge of multifactorial genetics could be more likely to develop an understanding of the scientific flaws of genetic essentialism.

10.3.2 The Teacher Beliefs Hypothesis

Depending on which country is sampled, Castéra and Clément (2014) estimated that 3–62% of biology teachers in European, South American, African, or Middle Eastern countries believe that "ethnic groups are genetically different and that is why some are superior to others" (e.g., 3% in France; 18% in Senegal; 34% in Poland; and 62% in Lebanon). In the US, studies estimate that 4% of pre-K–12 educators believe that racial inequalities are mainly due to a lower inborn potential to learn among African Americans (Quinn, 2017). Although we know of no studies showing the causal effect of teachers' beliefs on the development of genetic essentialism in students, it seems reasonable to predict that students who learn genetics with a teacher who believes in genetic essentialism are at greater risk of developing a stronger belief in genetic essentialism themselves.

One way this could occur is through the language used by teachers. Psychological essentialism is culturally transmitted through the use of generic noun phrases in our language (Rhodes et al., 2012). For instance, when people say things like "blacks get sickle cell", it implies that all "blacks" are uniform. When that statement is communicated along with the statement "whites get cystic fibrosis," it not only communicates the idea that all "whites" are the same, but it also communicates the idea that racial categories are discrete. In addition, studies have found that parents who endorse essentialism are more likely to use generics to describe the groups they essentialize. Parents' increased use of generics can create greater belief in essentialism in their children (Rhodes et al., 2012). Thus, it is possible that teachers could attenuate or amplify the development of genetic essentialism by using, or not using generic noun phrases when they teach genetics.

10.3.3 The Identity-Motivated Hypothesis

Social identity theory (Leyens et al., 2003; Tajfel & Turner, 2004) and identity protective cognition theory (Kahan et al., 2007) suggest that the impact of genetics education on genetic essentialism could vary according to the students' self-identified race.[2] This is because genetic ideas about race affirm the self-esteem of groups differently. African-American and Hispanic students are stereotyped as unintelligent relative to non-Hispanic white students or Asian-American students. Since people positively evaluate information when it contributes to their self-esteem (Tajfel & Turner, 2004), the impact of genetics education on belief in genetic essentialism could vary depending on the students' identities and whether the curriculum is providing students with information that refutes genetic essentialism or information that unintentionally augments it.

[2] This hypothesis can also apply to political identity (see Morin-Chassé et al., 2014).

10.3.4 The Conflict versus Contact Hypotheses

There are two different reasons why racial diversity of the science learning environment could moderate the impact of genetics education on belief in genetic essentialism. The first has to do with the social *contact* hypothesis (Allport, 1954; Pettigrew & Tropp, 2006). This hypothesis contends that contact with racial outgroup members during an interactive situation will reduce outgroup racial bias by helping people individuate others. The social *conflict* hypothesis (Putnam, 2007), on the other hand, contends that more racially diverse areas tend to have higher levels of outgroup distrust and lower levels of ingroup solidarity because of the anxiety associated with ambiguous social norms in ethnically diverse areas (Putnam, 2007). Since genetic essentialism reduces the ambiguity of social situations by facilitating quick stereotypical judgments (Keller, 2005; Yzerbyt et al., 2001), either of these mechanisms could moderate the relationship between genetics education and genetic essentialism. Interventions designed to reduce genetic essentialism might have little effect in a racially diverse school if intergroup contact has already caused students to disbelieve genetic essentialism. Alternatively, under a social conflict model, students in these schools might be resistant to changing their belief in genetic essentialism because of its social utility.

10.3.5 The Resource Competition Hypothesis

Tawa (2016) argued that belief in genetic essentialism of race should be less prevalent in racially diverse high socioeconomic status (SES) schools because students in those schools have less motivation to endorse it. Their lower motivation is due to a relatively greater supply of resources, which means less resource competition among students of different races. However, in racially diverse low SES schools, where resources are scarce, there could be more resource competition among students of different races. As people use essentialist ideas to justify actions that favor their racial ingroup (Morton et al., 2009), students in low SES schools could be more receptive to essentialist thinking if they perceive more resource competition. Thus, the impact of genetics education on genetic essentialism could be moderated by resource competition.

10.4 Implications for Teaching

Throughout history, genetic essentialism has been used to justify discrimination, violence, and genocide (Jackson & Depew, 2017). A genetics education that works to prevent the development of genetic essentialism is more humane than one that unintentionally perpetuates it. Mounting evidence now indicates that genetics education has the potential to perpetuate or prevent the development of genetic essentialism, but this potential could vary widely based on a variety of cognitive,

sociocultural, and demographic factors. If genetics education is one factor, among many, that is responsible for belief in genetic essentialism, then we think it should also be a venue for attempting to prevent its development. We call this kind of education a 'more humane genetics education'. We contend that there are two key ideas that are important to teach students if a genetics educator intends to implement a more humane genetics education. These are:

1. Genetic essentialism is a scientifically flawed world view that has been used to justify racism, violence, and genocide many times throughout history.
2. Genetic essentialism is scientifically flawed because:

    there is more genetic variation within human "races" than there is between them;
    most forms of human variation are best explained by multifactorial models of inheritance that include polygenic and social-environmental effects;
    the causes of variation in a trait within a group can differ from the causes of variation in a trait between two different groups;
    people inherit their genes with their social environments, so we need to be skeptical of anyone who claims that racial inequality is simply genetic;
    although racial beliefs and racial categories are culturally relative, racism is real and often justified through naïve and misinformed beliefs about genes and race.

Teaching students about these ideas is an extremely complex and challenging endeavor for any educator. These ideas are complex, and they may be threatening to a learner's identity or world view. However, there are several promising instructional approaches for helping learners change their beliefs when the learning context is emotionally fraught or cognitively complex. These include evidence-laden narratives (Darner, 2019), "the time for telling framework" (Schwartz & Bransford, 1998), and refutational texts (Lewandowsky et al., 2012). Donovan et al. (2019a) and Donovan (2015b) explain how these three instructional frameworks can be used to teach students these ideas. Donovan (2015a) makes a case for what knowledge teachers will need to possess to perform that kind of teaching. We suggest that anyone interested in pursuing a more humane genetics education read these papers before they begin the very important work of teaching genetics to reduce belief in genetic essentialism.

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