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ARTICLE

Exploring Relationships Among Belief in Genetic Determinism, Genetics Knowledge, and Social Factors

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Abstract Genetic determinism can be described as the attribution of the formation of traits to genes, where genes are ascribed more causal power than what scientific consensus suggests. Belief in genetic determinism is an educational problem because it contradicts scientific knowledge, and is a societal problem because it has the potential to foster intolerant attitudes such as racism and prejudice against sexual orientation. In this article, we begin by investigating the very nature of belief in genetic determinism. Then, we investigate whether knowledge of genetics and genomics is associated with beliefs in genetic determinism. Finally, we explore the extent to which social factors such as gender, education, and religiosity are associated with genetic determinism. Methodologically, we gathered and analyzed data on beliefs in genetic determinism, knowledge of genetics and genomics, and social variables using the "Public Understanding and Attitudes towards Genetics and Genomics" (PUGGS) instrument. Our analyses of PUGGS responses from a sample of Brazilian university freshmen undergraduates indicated that (1) belief in genetic determinism was best characterized as a construct built up by two dimensions or belief systems: beliefs concerning social traits and beliefs concerning biological traits; (2) levels of belief in genetic determination of social traits were low, which contradicts prior work; (3) associations between knowledge of genetics and genomics and levels of belief in genetic determinism were low; and (4) social factors such as age and religiosity had stronger associations with beliefs in genetic determinism than

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knowledge. Although our study design precludes causal inferences, our results raise questions about whether enhancing genetic literacy will decrease or prevent beliefs in genetic determinism.

1 Introduction

Genetic determinism is a concept with many different definitions. In this study, we build on the concept of genetic attribution (Tygart 2000) and define belief in genetic determinism as attributing to genes the formation of human traits at an individual level, perceiving them as having more causal power than what scientific consensus suggests. Simple understandings of genetics typically focus on a one-to-one relationship between genes, proteins, functions, and traits, as if particular traits or diseases were generally related to a single gene. The gene is seen as the active determinant of some kind of physical trait or behavior, to which it is given "power" or "agency" that supersedes a scientific explanation. This excessive belief in the attribution of trait formation to genes, when compared to a scientific viewpoint, has been identified as both an educational and a societal problem.

Beliefs in genetic determinism have been suggested to be a democratic problem because the power of genes could serve different social agendas (Geller et al. 2004; Shostak et al. 2009). Genetic explanations appear at first to provide rational and scientifically justifiable explanations, for instance, to social categories such as gender and race. However, this can be seen as an instance of the naturalistic fallacy, i.e., the claim that what is natural (in this case, genetically predetermined) is inherently good or right (Nelkin and Lindee 2004). Hence, biological explanations are thought to reinforce the sociological boundaries between groups, but this can be resolutely questioned.

From a scientific point of view, there has long been awareness that the description of the gene as an active agent determining phenotypic traits by itself is a "strawman model"—used in genetic studies as an instrumental model focusing on the genetic factor (nature). It ignored environmental factors (nurture), since environment was not the focus of genetic studies (Lawrence 1992). Already in the 1930s, Thomas Hunt Morgan stated the importance of the environment as an agent in the development of physical traits (Morgan 1934). Hence, it is reasonable to assume that knowledge about genetics and the influence of the environment on biological outcomes might reduce beliefs in genetic determinism. Moreover, in the last decades, the development of genomics and epigenetics has reinforced the notion of gene action as probabilistic and mutually interdependent with the environment through regulatory processes of gene activity, further contradicting the understanding of genes as sole active agents in the construction of phenotypes. Hence, one can conjecture that knowledge in modern genetics and genomics may counteract beliefs in the excessive attribution of trait formation to genes and, thus, beliefs in genetic determinism.

Genetic determinism has been found to be common in social discourse (Nelkin and Lindee 2004; Keller 2000) and the media (Condit et al. 1998, 2001). Moreover, in contrast to knowledge, social factors such as socioeconomic status and religiosity have also been suggested in the literature as potentially related to the formation of genetic deterministic beliefs (Nelkin and Lindee 2004; Parrott et al. 2004; Shostak et al. 2009).

In this article, we begin by investigating the nature of beliefs in genetic determinism. We intend to answer whether one can show that genetic determinism constitutes or is part of a coherent belief system. Then, we investigate the relationship between levels of belief in genetic

determinism and levels of genetics knowledge. The aim is to examine whether knowledge in genetics and genomics counteracts the development of beliefs in genetic determinism. Finally, we explore whether some social factors, such as gender, education, and religiosity, could be related to genetic determinism. To fulfill these three goals, we gathered and analyzed data by means of a newly developed questionnaire instrument, "Public Understanding and Attitudes towards Genetics and Genomics" (PUGGS) (Carver et al. 2017).

2 Background

2.1 Philosophical Ideas Related to Genetic Determinism

Determinism can be described as the philosophical idea that everything that happens, including human actions, is completely determined by previous events or entities. This leads to a belief that there is only one possible future and that the future then becomes predictable (Doyle 2011). Deterministic philosophical ideas had been developed in ancient Greece and were discussed in relation to human free will, which evidently is denied if determinism is accepted (Doyle 2011). The original discussion revolved mostly around whether there was a destiny or a fate that was inevitable to avoid, that is, around fatalism. This has ever since been one of the most disputed issues in philosophy.

Fatalistic thinking is found in a variety of religious worldviews, as we can see, for instance, in Christian and Hindu cultures (Young et al. 2011). Most cultural and religious traditions harbor some notion of superior powers that shape human fate, possibly because this offers a means to alleviate existential distress before the arbitrariness of human suffering (Geertz 1973) and motivates prosocial behavior (Johnson et al. 2003; Johnson and Krüger 2004).

After the Scientific Revolution in the seventeenth century and the Enlightenment, the findings of laws and regular patterns within nature induced a belief in determinism as stemming from nature itself rather than from fate or divine providence (Honderich 2005). Since that time, one of the major philosophical discussions concerns whether the determinants of "nature" or "nurture" should be considered as the most important for causing individual differences in physical and behavioral traits in organisms, generally speaking, and humans in particular.

The idea that humans acquire all or almost all their behavioral traits from "nurture" was termed "tabula rasa" ("blank slate") by the empirical philosopher John Locke in the sixteenth century. The blank slate view proposes that humans develop only from environmental influences. In opposition is the idea of "essentialism," which implies that the mind is born with certain ideas or knowledge. This philosophy goes back to Plato and assumes that these ideas are introduced by some divine being (Doyle 2011).

The "nature versus nurture" debate in its modern sense was coined by Francis Galton in the nineteenth century as a discussion about the influences of heredity and environment on social advancement (Galton 1874). The belief in the importance of nature for our characteristics and actions developed thereafter and has commonly been referred to as biological determinism (Allen 1984), which finds causes for our actions in our biological setup. Of course, most of our characteristics and behaviors can to some degree be explained by our biology (Resnik and Vorhaus 2006), and this is still investigated with scientific rigor, say, in heritability studies. However, the term "biological determinism" has been used within the literature to describe (and criticize) the excessive belief that human behavior is controlled by an individual's genes

(e.g., Lederman and Bartsch 2001) or some other biological cause, i.e., a misuse of biological explanations (Allen 1984).

Often biological determinism is ascribed a meaning that makes it synonymous with genetic determinism; however, there is no specific ontological referent which biological determinism refers to. The abstractness of the "essence" concept in biological determinism could be reduced and the idea made even more powerful by introducing an "essence placeholder" in order to link the essence to something material and concrete. Dar-Nimrod and Heine (2011) claimed that this is the role taken by the genes when they become the placeholder for essentialist ideas in biological determinism. By introducing the gene or DNA as the physical agent within biological determinism, they claim that genetic determinism arises. This way of understanding genetic determinism has gained general acceptance in public discourse, as can be seen in the definition provided by the *Oxford English Dictionary* (Genetic determinism 2016) in which genetic determinism is defined as the idea or belief of "the determination of a process or effect by genes; spec. the attribution of sole or excessive importance to genes in the determination of intelligence, behaviour, development, etc."

However, as pointed out by Turkheimer (2011), we cannot deny the importance of genetics in the genesis of human behavior. The problem then is to separate an "excessive" genetic deterministic explanation (i.e., an explanation of trait formation where genes are ascribed more causal power than what scientific consensus suggest) from a "sound genetic" explanation (i.e., an explanation where genes are ascribed the same level of attribution as suggested by scientific studies). In this study, we do this by operationalizing belief in genetic determinism in a similar way as suggested by Resnik and Vorhaus (2006), by classifying different traits on a continuum of probability that a genetic makeup leads to the development of the specific trait, from strong, via moderate to weak, and then comparing if the participants provide a similar attribution to the genes in question as has been documented in heritability studies. All the traits relate to humans and we included biological and behavioral or social traits along this continuum. The methodology is further elaborated in the methods section.

2.2 Genetic Determinism and Its Relationship to Genetics

How then do genes work? As in all sciences, different scientific theories and models have evolved over time (Gericke and Hagberg 2007; El-Hani 2007). Many of these scientific models use explanatory reduction that in various ways leads to the idea that the power of the genes is at the forefront, neglecting environmental interaction (Gericke et al. 2014). In this section, we outline a possible explanation for why higher knowledge levels in genetics and genomics might counteract genetic deterministic beliefs.

It is useful to consider, in this respect, Moss' (2001, 2003, 2008) proposal of a distinction between two ways of understanding genes that are often conflated. He called them "gene-P" and "gene-D." Gene-P amounts to the gene as a determinant of phenotypic differences. As Moss wrote, "when one speaks of a gene in the sense of Gene-P, one simply speaks *as if* it causes the phenotype… Gene-P is defined strictly on the basis of its instrumental utility in predicting a phenotypic outcome…" (Moss 2001, pp. 87–88). As an instrumental concept, gene-P is not accompanied by any hypothesis of correspondence to reality, and this is what makes it acceptable, as a simplified assumption of a preformationist determinism (as if the trait was already contained in the gene, albeit in potency). It provides a distal view of the gene, in which it is instrumentally inferred from the phenotype (hence the "P"). Gene-P is a useful view

about the gene, provided that it is properly understood as an instrumental concept. As Waters (1994, p. 172) wrote, "The basic dogma of classical genetics was that gene differences cause phenotypic differences. (…). What were studied were character differences, not characters, and what explained them were differences in genes, not the genes themselves". Accordingly, when someone refers to "genes for" traits, the correct interpretation is not in terms of some explanation that would relate single genes to phenotypes of an organism, but as an account of differences observed in phenotypes in a population that can be explained, to a certain (estimated) extent, based on genetic differences in that population (Waters 1994; Plaisance et al. 2012). That is, a gene is from this perspective a "difference maker" (Sterelny and Kitcher 1988), and a correlation between genetic and phenotypic differences is estimated in the form of a heritability measure.

To see how genetic determinism enters the picture, we first need to consider gene-D. Gene-D "is defined by its molecular sequence. A Gene-D is a developmental resource (hence the 'D') which in itself is indeterminate with respect to phenotype" (Moss 2001, p. 88). It is related to a realist view of the gene, from the standpoint of DNA, thus entailing the necessity of taking into account its embedment into complex interaction networks in cell physiology and developmental pathways culminating in the phenotype. Gene-D is, therefore, a developmental resource in parity (Oyama 1985; Griffiths and Gray 1994; Griffiths and Knight 1998) with other developmental causes, such as epigenetic and environmental factors.

In the last three decades, findings in the science of genetics and related fields have reinforced the gene-D perspective. For example, genes are overlapping and can give rise to several different products (making the proteome qualitatively different from the genome), and there is no obvious relation between the amount of DNA in an organism and its morphological or behavioral complexity (for discussion, see, for example, El-Hani 2007; Falk 2014; Gericke and Hagberg 2007; Gericke and Smith 2014; Meyer et al. 2011; Portin 2009). Gene-D is not "controlling" or "purposively acting" in a specific direction but is a component among others within biochemical, physiological, developmental processes. It is as such that gene-D participates in explanations in genetics, molecular biology, physiology, developmental biology, etc. Given the recent findings that epigenetic mechanisms can lead to changes in gene expression patterns as a consequence of experience (see, e.g., Moore 2015), the very dichotomy between "nature" and "nurture" is called into question since the environmental factors and regulatory mechanisms are included in the explanation of gene-D. Therefore, it can be hypothesized that understanding of modern genetics and genomics as in a gene-D view could counteract excessive belief in the attribution of trait formation to genes and therefore genetic determinism.

Genetic deterministic views do not follow simply from gene-P, as we pointed out above. What might be problematic is when lay people, typically for lack of sufficient scientific understanding, do not recognize the instrumentalist nature of gene-P and interpret the instrumental gene as a realist concept, conflating gene-P and gene-D. If this happens, the power of the instrumentalist gene at a phenotypic level (genes-for-traits) is paired with the realist gene (DNA). In that way, a powerful nonscientific genetic deterministic explanatory model is created that links the "essence placeholder," the DNA, with powers of determining phenotypic characters and behaviors, and the stage is set for genetic deterministic beliefs. In previous research, it has been shown that this conflation is common in textbook discourse worldwide (Aivelo and Uitto 2015; Gericke and Hagberg 2010a, b; Gericke et al. 2014; Santos et al. 2012) and that high school biology students lack the scientific understanding to discern between different models in genetics (Gericke et al. 2013; Gericke and Wahlberg 2013). Also, teachers conflate them in their talk while teaching (Thörne et al. 2013). Based on these

findings and previous arguments, we can make the assumption that more advanced understanding in genetics could counteract genetic deterministic beliefs.

As outlined by Lewontin (2011), the relationship between genotype and phenotype can be described by four basic models that have been, and still are, used in genetics: one-to-one, one-to-many, many-to-one, and many-to-many (see Fig. 1). The first goes back to the unit factor theory at the beginning of the twentieth century, i.e., one gene gives rise to one trait (Mayr 1982). The second model describes one gene affecting many traits (pleiotropy), while the third model accounts for many genes affecting one trait (polygeny). It is undoubtedly correct that every part of the genome is connected causally with the phenome (a set of phenotypes) by at least some molecular mechanistic pathways, but there is variation in this relation, which can make all of these four models valid at least for some cases. But generally for most eukaryotic organisms, model 4 (many-to-many) is the most acceptable description for most cases of the relationship between phenotype and genotype (Lewontin 2011). And often, the many-to-many model is insufficient, since genes and environment are usually both involved in the development of phenotypes, as captured by the norm-of-reaction concept (see, e.g., Falk 2001).

In the last decades, research in genetics, genomics, and related fields have advanced so deeply and fast that our understanding of genes and genomes and how they relate to development, phenotypic traits, cell physiology, has radically changed (Keller 2000, 2005a). There seems to be a general shift within the scientific community from a more deterministic to a more probabilistic understanding of the relationship between genes and traits (see Fig. 1). Those advances have made it clear that gene action and function should be conceived as embedded into multiple hierarchical levels, in which complex networks of interactions between components are the rule (Ideker et al. 2001). Consequently, the probabilistic understanding of the structure, dynamics, and functions of genes demands that they are located in complex informational networks and pathways.

Deterministic understandings of genetics typically focus on one-to-one causal relationships between genes, proteins, functions, and traits, as if particular traits or diseases were generally related to a single gene (Lewontin 2011). Hence, beliefs in genetic determinism can be related to the use or "misuse" of simplified explanatory models of genetics. If a one-to-one model is used where it is more appropriate to use a many-to-many model, we can conjecture that this simplified way of understanding genetics would be correlated with elevated levels of genetic deterministic belief, i.e.,

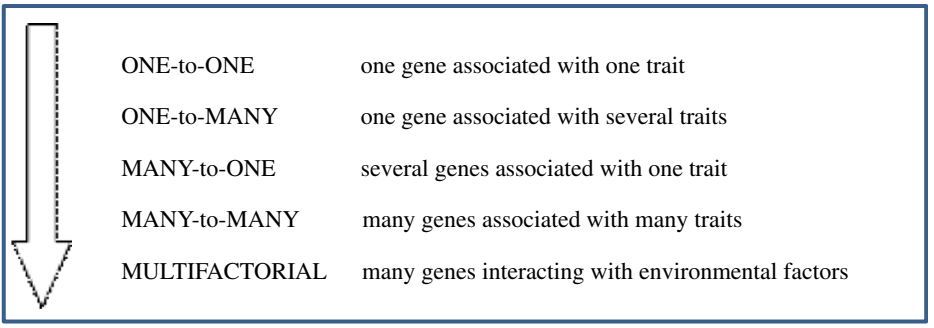

Fig. 1 From deterministic to probabilistic understanding about genotype-phenotype relations, from Carver et al. (2017)

belief in which greater attribution is given to the genes compared to the environment, even when this is not supported by our current knowledge in genetics. In line with this argument, it could be expected that improved knowledge of contemporary multifactorial genetics and genomics could suppress or at least moderate genetic deterministic beliefs.

Genetic science now tells us that, despite the usefulness of the deterministic gene as an instrumental concept in some explanatory tasks, in realistic terms, it is not possible for any trait—even "single-gene disorders"—to be determined by genes only, due to the influence of epigenetic and environmental factors (Sarkar 1998). There can be many genes associated with one trait, or many different traits associated with the same genes, which in turn are affected by a myriad of environmental factors. As shown in Fig. 1, as we move from a "one-to-one" (deterministic) to a "multifactorial" (probabilistic) model of the relationship between genes and traits, environmental and epigenetic factors are seen as playing an increasingly more important role in the development of traits and diseases. In the probabilistic model, genes are embedded in the context of an internal and external environment, with due attention to the fact that many genetic and epigenetic factors interact with one another. Also, genetic and environmental factors often interact nonadditively, so that genes show different expressivity and penetrance depending on the influence of environmental factors (Moore 2013; Sarkar 1998, 2011).

2.3 Social Explanations for Genetic Determinism

Besides genetics knowledge, or more precisely lack of understanding of contemporary genetics knowledge, beliefs in genetic determinism have also been suggested to be embedded within social discourses that influence the perceptions of people (Lewontin 1993; Keller 2000), or as ways of making meaning of the social world in psychological theories (e.g., Haslam et al. 2000, 2002; Keller 2005b). For example, Nelkin and Lindee (2004) have argued that genetic determinism or "genetic essentialism," as they name it, is not simply a result of misunderstanding or simplification of science but could be anchored in deep beliefs about social phenomena. Psychological research has recognized that people's minds generally tend to essentialize the particular entities they encounter. The belief in a causal relationship between essence and expected characteristics together with the stability of essence is the defining elements of psychological essentialism.

In sociopsychological research, the studies of psychological essentialism have evolved as a field of research that explores essential beliefs in which biological essentialism is one of several investigated constructs. Psychological essentialism is always related to social categorizing, i.e., essentialist lay theories are used for social categorization, and often investigated in relation to prejudice and stereotyping regarding social groups such as race, gender, and sexual orientation (e.g., Haslam et al. 2002, 2006). Psychological essentialism describes how people tend to reason and categorize the members of groups, say, of certain races or genders, and essentialist reasoning has been demonstrated in a wide range of cultures (Norenzayan and Heine 2005).

According to Yzerbyt et al. (1997), essentialist categorization is based on the following features: (1) specific ontological status, i.e., all members are seen as having an essential feature in common. (2) Category membership is seen as immutable. (3) Essentialist categories allow inferences about the members of the category. (4) The features of the category members are explained through the lens of a unifying theme. (5) The categorization is exclusive and a

member of one category can seldom be seen as a member of another. The underlying idea is that formation of beliefs can be understood as the result of social cognition (Jost et al. 2003).

Three different motives have been identified for social cognition: the first is the desire to reach a conclusion, i.e., that one's position of privilege will be preserved (ideological motive); second that the self is worthy and valuable (existential motives); and third a desire to arrive at an understanding independently of content (epistemic motive) (Jost et al. 2003). Haslam et al. (2004) found that essentialist beliefs consist of two dimensions that are both social and biological: natural kinds (that social categories are natural) and entitativity (the similarity and common fate of a group). Keller (2005b) explored the biological component of psychological essentialism, which he denotes as genetic determinism, and found that belief in genetic determinism, as a lay theory, is correlated with negative racial stereotyping, prejudice and sexism. Keller further concluded that he found support that the biological component of psychological essentialism is related to the two basic mechanisms of social cognition discussed above (ideological and existential motives), but also called for the need of further studies investigating different forms of biological determinism (Keller 2005b).

Recently, Andreychik and Gill (2014) developed this conceptual structure further by suggesting that the biological component is only part of the natural kinds category, which they considered to consist of two dimensions: biosomatic essentialism and biobehavioral essentialism. Biosomatic essentialism is related by the authors to physical traits and biobehavioral essentialism to behavioral traits. In their study, it was found that biobehavioral essentialism—but not biosomatic essentialism—contributes to prejudice and negative attitudes toward other social groups (Andreychik and Gill 2014).

Dar-Nimrod and Heine (2011) suggested that essentialist thinking could be reinforced by a superficial understanding of genetics, in which genes take the role of concrete placeholders for essentialist ideas in genetic determinism, i.e., the gene or DNA becomes a material unit of nature to which biological essentialist ideas can be referred to. The gene or DNA can then acquire the properties of biological essentialism. If this occurs, it might have profound importance to how people respond and perceive genetic information about issues such as race, ethnicity, gender, and other social aspects. These authors also suggest that people in general tend to use what they call "strong genetic explanations" instead of "weak genetic explanations" for most human phenomena in which nature and nurture interact (Dar-Nimrod and Heine 2011), i.e., explanations including deterministic causal relations and not probabilistic ones, which would be more scientifically correct.

However, in all the above reported studies and almost all studies on psychological essentialism, biological essentialism or genetic determinism is studied at a group level looking for ideological or existential motives. Though Suhay and Jayaratne (2012) did a comparative study in which genetic differences at individual and group level were compared, they found that genetic differences were used to explain group differences regarding race, class, and sexual orientation differently between ideological groups, but these differences were not observed at the individual level within groups. However, as shown by Morin-Chassé (2014), people's beliefs in genetic determination of behavioral traits at the individual level can be reinforced by the media.

In this study, we are investigating genetic determinism from an individual perspective. The reason is that this study focuses on an educational perspective and our main interest concerns epistemic motives, not ideological and existential ones. The underlying premise is that knowledge impacts beliefs in genetic determinism. In school science, the effects of genes on different traits are also mostly taught at the individual level, seldom at the group level. Hence,

the possible effect of knowledge on the epistemic beliefs relating to genetic determinism should be visible at the individual level. However, despite the findings of Suhay and Jayaratne (2012), there might of course be overlaps and possible effects at the group level. To be able to find such overlaps, we also decided to investigate the effects of social groups on genetic deterministic beliefs, focusing on age, gender, education, religiosity, and previous experiences with genetics. These social categories have been in focus for many studies of genetic essentialism (e.g., Jayaratne et al. 2006).

Education and personal experience of genetics are ways of gaining genetic knowledge and are therefore of particular interest in our study as possible factors for counteracting genetic deterministic beliefs. Religiosity is of interest in this study since effects from this factor have been shown to influence peoples' beliefs regarding individual traits. Parrott et al. (2004) found that some people believe God plays an important role in how genes are expressed and impact health. This is consistent, as we argued previously, with fatalistic thinking in a number of religions (Young et al. 2011). We conjecture, therefore, that people who consider themselves religious may be more inclined to believe that genes are "fixed," and that their genetic makeup is their destiny, thereby holding more deterministic views about genes. Gender has also been shown to be correlated to sexism and genetic determinism (Keller 2005b), and men could therefore be hypothesized to show a stronger tendency toward genetic determinism. As a consequence of these suggestions, it is also of interest to investigate the relationship between these social factors and belief in genetic determinism.

Moreover, in line with the findings of Andreychik and Gill (2014), we have also included biological as well as behavioral traits in the study, ranging from totally genetically regulated traits, such as blood group, to almost totally environmentally determined ones, for instance, interest in fashion. Tygart (2000) suggested that genetic attribution, the way in which people perceive the influence of genetics on individual characteristics, depends on the types of traits, and therefore, it is important to investigate a large diversity of traits. Likewise, Morin-Chassé (2014) found that people convey perceptions of genetic attribution from one behavioral trait to another, but not to biological traits, indicating the existence of subdimensions of genetic determination. Similarly, Condit et al. (2009) found in an interview study that laypeople have incorporated two sets of public discourses—one that describes genetic causation and another that describes behavioral causation. By including a great diversity of traits in the current study, we are able to investigate if genetic determinism at an individual level is a coherent belief system, or if it might be composed of different subdimensions as indicated at group level by Andreychik and Gill (2014).

3 Aim and Research Questions

Belief in genetic determinism has been identified as problematic for society because it has the potential to foster intolerant attitudes (such as racism and homophobia; Dambrun et al. 2009; Shostak et al. 2009). Consequently, efforts to teach the multifactorial model of genetics could be considered worthwhile if it could be established that increased knowledge of modern genetics and genomics is associated with low levels of belief in genetic determinism (Gericke et al. 2014; Smith and Gericke 2015). However, little is known about the putative relationships between beliefs in genetic determinism and genetics and genomics knowledge. In

this study, we intend to address this gap in the literature by addressing the following research questions:

1. Do beliefs in genetic determinism form a unitary construct?
2. Do significant relationships exist between levels of genetics and genomics knowledge and levels of belief in genetic determinism?
3. To what extent are factors such as age, gender, education, religiosity, and experience with genetics from everyday life associated with genetic deterministic beliefs?

4 Methods

We employed a quantitative survey research design to explore putative relationships among beliefs in genetic determinism, knowledge of genetics and genomics, and social factors.

4.1 Instrument

The two core concepts we measure and compare in this study are "beliefs" and "knowledge." Here we define "belief" as the state of mind in which a person thinks something to be the case, with or without there being convincing reasons that something is the case with factual certainty (Wyer and Albarracín 2005). This is in line with Pajares' (1992, p. 316) definition of belief as "individual's judgment of the truth." As argued by Bandura (1997), beliefs more than truth guide our decisions, actions, and reactions. Hence, by investigating beliefs in genetic determinism, we can learn more about how people think and act in relation to biological essentialism. In this study, we investigate beliefs about human characteristics and to what degree characteristics are attributed to genetic determinants.

As concluded by Dretske (1990, p. 183), "it takes something more to *know* because knowledge requires, besides mere belief, some reliable coordination of internal belief with external reality." Here is an important connection between beliefs and knowledge that we explore in this study. Knowledge in the revised Bloom taxonomy of educational objectives is defined as: "the knowledge that shares a consensus of acceptance within the discipline" (Airasian 2001, p. 13). Likewise, Dretske (2000, p. 81) concludes that knowledge can be seen as "the result of an assessment and evaluation procedure in which conclusions are reached from the premises in conformity with rules that are…rationally justifiable." Moreover, Airasian concludes that it is scholars (or experts) who have spent their lives studying and working in a field that determine the substance of a given subject matter, though this subject matter constantly changes over time (Airasian 2001, p. 13). In this study, we adopt this definition of knowledge and investigate the extent to which a person can answer correctly in line with the scholarly (or expert) knowledge on the subject matter.

The PUGGS instrument (Carver et al. 2017) was designed to measure the constructs we seek to investigate. The PUGGS was developed as part of the Public Understanding and Attitudes towards Genetics and Genomics Study (PUGGS) and includes five different sections measuring various constructs: (1) *social background information* (e.g., age, gender, education, religiosity, and personal experiences with genetics), (2) *belief in genetic determinism*, (3) *knowledge of the complexity of gene-environment interactions*, (4) *knowledge of modern genetics and genomics*, and (5) *attitudes toward applications of modern genetics and genomics* relating to gene therapy, genetic testing, prenatal genetic testing, personalized medicine, and

pharmacogenomics. The instrument, along with descriptions of the development and validation procedures, coding schemes, and data, is available in Carver et al. (2017). In this study, we are utilizing data from PUGGS sections 1, 2, 3, and 4. We elaborate on the tasks and constructs from these sections below.

4.1.1 Social Background Factors

Social factors have long been considered to be important in the development of beliefs in genetic determinism (e.g., Nelkin and Lindee 2004; Keller 2000). Nevertheless, local ethical guidelines made it more difficult to gather information on our participants' political beliefs, religious affiliations, and socioeconomic statuses. Such variables have been suggested as being related to belief in genetic determinism (e.g., Geller et al. 2004, Nelkin and Lindee 2004; Shostak et al. 2009). We could include questions about participants' age, gender, field of study at university, personal experience with genetics, and religiosity, as specified in the first section of the PUGGS questionnaire.

We included age in this study because it is possible that older students gained more knowledge about genetics, either through education, life experience, or exposure to the media, which in turn might affect their beliefs. We included gender because previous studies have suggested that males and females may use genetic explanations to classify themselves differently (Shostak et al. 2009). We asked students to specify their field of study in case those studying science and technology-related fields would have more interest in genetics, which in turn might affect their knowledge and beliefs. We included a question about participants' personal experiences with genetics (e.g., personal or family history of diseases or genetic testing) because such experiences have been shown to be important to genetic belief formation (Senior et al. 1999). For example, if a student has a close relative with a rare genetic disease, this might lead him or her to have a more deterministic belief in genetics. In contrast, students or family members who have tested positive for certain genetic markers, but have not developed the particular disease or trait, might think less deterministically about genetics. Finally, we included an item about participants' religiosity, as other empirical studies have suggested this is an important factor related to fatalism and genetic deterministic beliefs (i.e., Castéra and Clément 2014; Parrott et al. 2004).

4.1.2 Belief in Genetic Determinism

In this study, we define belief in genetic determinism as the attribution of human trait formation to genes (all or a certain subset thereof), where genes are ascribed more causal power than scientific consensus suggests. We use section 2 in the PUGGS instrument to measure magnitudes of belief in genetic determinism. Specifically, the PUGGS includes a task in which participants are prompted to indicate the relative importance of genes or environments in the determination of 15 different traits on a five-point, Likert-type scale (1: only environmental; 2: mainly environmental; 3: both, to the same extent; 4: mainly genetic; 5: only genetic; see Table 1). We refer to this PUGGS task as the "table of traits" or "T T". In the PUGGS, this task was designed to include traits mostly associated with genetic causes and traits mostly associated with environmental causes. The approach to operationalizing genetic determinism builds on previous literature. For example, Turkheimer (1998) referred to "strong genetic explanations" for monogenic traits that involve a small number of genes, and contrasted such cases with "weak genetic explanations," in which conditions are known to

Table 1 Table of traits. In section 2, the participants were asked to fill in the table below after reading the following question: "People vary in traits (physical features, behaviours, diseases and disorders), such as those shown in the table below. Genetic differences and environmental differences may contribute to this variation. Environmental differences can for example be differences in culture, upbringing, lifestyle, eating habits, or exposure to pollution. In the table below please indicate to what extent you think genetic and environmental differences contribute to these traits"

Code	For each trait mark with an "X" in ONLY ONE of the columns from 1 to 5.	Only environmental differences contribute to the trait	Mainly environmental differences contribute to the trait	Both genetic and environmental differences contribute to the same extent to the trait	Mainly genetic differences contribute to the trait	Only genetic differences contribute to the trait
		1	2	3	4	5
TT1	Height					
TT2	Bipolar disorder					
TT3	Diabetes (type 2)					
TT4	Color blindness					
TT5	Schizophrenia					
TT6	Alcoholism					
TT7	Breast cancer					
TT8	Interest in fashion					
TT9	Addiction to gambling					
TT10	Political beliefs					
TT11	Intelligence in adults					
TT12	Severe depression					
TT13	Attention deficit hyperactivity disorder (ADHD)					
TT14	Asthma					
TT15	Violent behavior					
TT16	Religious beliefs					
TT17	Blood group (ABO)					

have many genetic as well as environmental causes. Many of the ways in which genes relate to human traits are best characterized in terms of weak genetic explanations (Turkheimer 1998). Resnik and Vorhaus (2006) developed Turkheimer's ideas a bit further by distinguishing between three forms of "genetic determinism," which they relate to the probability of developing a trait when a specific gene variant is present:[1]

- Strong genetic determinism: the gene in question almost always leads to the development of a specific trait.
- Moderate genetic determinism: the gene leads to the development of a trait in more than 50% of the cases.
- Weak genetic determinism: the gene sometimes leads to the development of a trait, though in less than 50% of the cases.

[1] We would rather talk about different forms of attribution of traits to genes, since we endorse a probabilistic rather than a determinist perspective on the genes-phenotype relation.

In line with this framework, the PUGGS tasks prompt participants to identify levels of genetic influence (strong, moderate, and weak). The tasks include both physical traits (e.g., color blindness, breast cancer, and height) and behavioral traits (e.g., alcoholism, violent behavior, severe depression). We use heritability studies as a benchmark for classifying the traits along this continuum. We recognize the limitations of heritability scores (e.g., Lynch and Bourrat 2017; Stickel et al. 2017; van der Sluis et al. 2010). Specifically, we are aware that heritability measures are context-dependent and ascribed to the population level. Nevertheless, heritability can be defined as "the ratio of genetically caused variation to total variation (environmental and genetic)" (Block 1995, p. 103), and because we posed our TT tasks in alignment with this framework (as a ratio between genetic and environmental attribution), we consider heritability estimates to be rough but acceptable approximations for ascribing traits to being strongly, moderately, or weakly influenced by genetics (see Table 2).

The results from the TT items are compared with heritability estimates by first transforming the mean values into an index from 0 to 1. This is calculated according to the equation: $n = (x - 1)/4$, where x is the mean score from Table 7 and n is an index of the weight given to the genetic factor. The values in Table 7 are based on a scale from 1 to 5 (1 = only environmental differences, and 5 = only genetic differences) used in the PUGGS questionnaire, and thus by subtracting 1 the scale is reversed to a scale from 0 to 4, and by dividing by 4 an index from 0 to 1 is created where 1 stands for totally genetically weighted.

Table 2 differentiates task traits in terms of current scientific evidence (i.e., whether they can be conceived of as mostly genetically influenced, environmentally influenced, or a mix of the two). Moreover, controversial traits (e.g., violent behavior) are also included in this task. In this way, the task makes it possible to examine how different traits evoke beliefs in genetic determinism among participants. Among the controversial traits, three traits (i.e., interest in fashion, political beliefs, and religious beliefs) are included despite the fact that no heritability

Table 2 Categorization of traits according to hereditability studies, considering whether the traits are predominantly environmentally influenced (with heritability indexes mostly reported below 0.4), genetically influenced (with heritability indexes mostly reported above 0.6), or approximately equally by both (with heritability indexes mostly reported between 0.4 and 0.6)

Predominantly environmentally influenced	Mix (approximately equally influenced by both)	Predominantly genetically influenced
TT3 Diabetes (type 2)	TT6 Alcoholism	TT1 Height
TT7 Breast cancer	TT9 Addiction to gambling	TT2 Bipolar disorder
TT8 Interest in fashion[a]	TT11 Intelligence in adults	TT4 Color blindness
TT10 Political beliefs[a]	TT12 Severe depression	TT5 Schizophrenia
TT16 Religious beliefs[a]	TT15 Violent behavior	TT13 ADHD
		TT14 Asthma
		TT17 Blood group

Sources for the heritability estimates (when available): *height*: Jelenkovic et al. (2016), Silventoinen et al. (2000), Visscher et al. (2008); *bipolar disorder*: McGuffin and Sargeant (1991), McGuffin et al. (2003); *diabetes* (type 2): Almgren et al. (2011); *color blindness*: Osborne et al. (1968); *schizophrenia*: Cardno et al. (1999), Visscher et al. (2008); *alcoholism/alcohol dependence*: McGue (1999), Prescott and Kendler (1999); *breast cancer*: Hemminki et al. (2004), Locatelli et al. (2004); *addiction to gambling behavior*: Eisen et al. (1998), Lobo and Kennedy (2009); *intelligence*: Devlin et al. (1997), Plomin et al. (2016); *severe/major depression*: Kendler et al. 1992, 2001), Kendler and Prescott (1999); *ADHD*: Chang et al. (2013), Kan et al. (2013), Pingault et al. (2015), Rutter et al. (1999); *asthma*: Fagnani et al. (2008), Thomsen et al. (2010); *violent behavior*: Ferguson (2010), Frisell et al. (2012); *blood group*: Griffiths et al. (2015), Meneely et al. (2017)

[a] For these traits, no heritability studies were found

estimates could be found. These traits are classified as predominantly environmentally/socially influenced, which was validated in prior work by an expert panel (see Carver et al. 2017).

4.1.3 Knowledge of Genetics and Genomics

The PUGGS instrument contains two sections designed to measure genetics and genomics knowledge: *knowledge of the complexity of gene-environment interactions* (section 3) and *Knowledge of modern genetics and genomics* (section 4). PUGGS section 3 consists of nine items that measure understanding of the degrees of complexity of gene-environment interactions. These items correspond to five core ideas that address different models of the genotype-phenotype relationship, ranging from the one-to-one model of genetic effect on phenotypes to the multifactorial model (see Fig. 1). This framework includes concepts such as polygenic traits, pleiotropy, gene-environment interaction, and multifactorial processes at different organizational levels, as well as the one-to-one "genes only" model.[2] Only one of the items (item 8) tests knowledge of the degree to which both genes and environmental factors have an influence on traits. The other items test the effect of the environment only (item 6), or the various effects of genes on traits (items 1–5, 7, and 9; for example, that many different genes can influence the same trait).[2] Higher scores on these sections suggest a more advanced understanding of the genotype-phenotype relationship (e.g., being aware of certain levels of complexity), whereas lower scores suggest a simpler understanding of genetics (e.g., a causal one-to one model). Items in PUGGS section 3 are designed to measure participants' knowledge of scientific principles rather than detailed knowledge. The items are designed to avoid using specific examples of traits and diseases (except for height, which all students are expected to be familiar with). The items refer to human traits and diseases, which could be either physical or behavioral.

PUGGS section 4 includes items designed to measure participants' knowledge of contemporary scientific ideas relating to genomics, gene expression/regulation, and epigenetics.[3] PUGGS sections 3 and 4 were designed to measure different aspects of genetics knowledge. First, the items in these sections separate knowledge of genetics that is already taught in schools (section 3) and knowledge relating to newer fields like genomics and epigenetics (section 4). This differentiation allows us to determine how different types of knowledge might be associated with different magnitudes of belief in genetic determinism. Second, section 3 was specifically designed to reflect the knowledge dimension behind genetic determinism, whereas section 4 was not.

The items in PUGGS sections 3 and 4 measure the overall magnitudes of participants' subject matter knowledge in genetics and genomics. Therefore, three options were included for each statement in the questionnaire: "true," "false," or "do not know" (Carver et al. 2017).

4.2 Sampling and Data Collection

In order to investigate putative relationships among the magnitudes of knowledge of genetics and genomics and beliefs in genetic determinism, we sought a participant population that would have a basic level of genetic knowledge (i.e., at least a high school education) and varying exposure to more advanced topics in genomics. First-year university students with

[2] See Appendix (Table 13) for the items and the core ideas.
[3] See Appendix (Table 14) for the items and the core ideas.

varying career trajectories would meet these criteria. Consequently, we chose a targeted sampling strategy to identify a large sample of first-year university students available for participation in the study. In February and March 2015, we applied the questionnaire to several classes of first-year Brazilian undergraduates enrolled in an Interdisciplinary Bachelor Program at a Brazilian Federal (public) university in northeast Brazil. Data were also collected "on-site" at two registration days. In total, 446 students participated. Participants were asked to fill in the questionnaire while two of the authors of this article were present. The questionnaire took 20–25 min to complete. Of the participants, 51% were females and 49% were male. Most (27%) of the students were attending humanities as their main field of study, followed by science and technology (26%), arts (23%), health sciences (23%), and others (1%).

Our sample is likely to be representative of an "educated youth" in Brazil, but it is by no means representative of all Brazilians or university undergraduates in general. Freshmen students constitute a large proportion of young adults. In Brazil, 35.9% of the population at 25 years of age or more have completed high school education, according to census data from 2010 (IBGE, (BIoGaS) 2010). The sample is representative of both high- and low-income families because it is a federal university, where at least 50% of the students come from public (nonprivate) schools. Our aim is not to make generalizations about a population at large, since our questionnaire is probably less applicable to groups that have not completed high school.

The study follows the Brazilian guidelines of ethical conduct in research involving humans and is approved by the Committee for Ethics in Research from the Nursing School of the University at which the study was conducted (No.: 1.023.782). All participants gave written informed consent before answering the questionnaire, and after completion, the data were deidentified.

4.3 Statistical Analyses

Questionnaires with more than eight missing answers (10% of the total) were eliminated from the sample. In total, we found 19 missing answers, comprising 4% of the sample.

We used Cronbach's alpha to measure internal consistency reliability using the statistical packages SPSS© (version 22) and R (with the Psych package) (all calculations were run in both, to ensure accuracy). The calculation of the Cronbach's alpha does not support missing data, and so we used modal value imputation to replace the missing values (Watanabe and Yamaguchi 2003).

In order to explore whether belief in genetic determinism formed a unitary dimension, we performed an exploratory principal component analysis (PCA). Specifically, the PCA was used to identify the main dimensions (Lebart et al. 1995) characterizing the items in PUGGS sections 2, 3, and 4. We performed the PCA by using a polychoric correlation matrix in order to take into account the dichotomous nature of our data. We did not perform factor rotation. After the PCA, we performed a confirmatory factor analysis (CFA) to investigate the model structure emerging from the PCA. Thus, the most representative items from each axis were summed to generate new composite variables as follows: social traits (TT6, TT8, TT9, TT10, TT11, TT12, TT15, TT16); biological traits (TT2, TT3, TT4, TT5, TT7, TT13, TT14, TT17); and knowledge (Q1, Q2, Q3, Q4, Q5, Q7, Q9, Q13, Q15, Q16, Q17, Q18, Q19, Q20, Q21, Q23, Q24). Using these methods, we outlined the descriptive data for three sections of the PUGGS instrument: (2) *belief in genetic determinism*, (3) *knowledge of the complexity of gene-environment interaction*, and (4) *knowledge of modern genetics and genomics*.

After using the PCA and CFA to establish and confirm the three new composite variables, we conducted Kendall's correlations among participant scores for these three instrument sections in order to test for the strength of the relationships among these composite variables and thereby address the second research question. For Kendall's correlation, one-tailed p values were calculated in order to test the association between genetics and genomics knowledge and belief in genetic determinism.

The third research question explored the possible associations between social variables and beliefs in genetic determinism and was answered using multiple one-way ANOVAs (each social variable was tested using an ANOVA). The ANOVAs were used to test whether the different social groups differed in terms of their levels of belief in genetic determinism. In cases of significant differences, we performed post hoc tests (Tukey's HSD) to identify which means significantly differed from one another. We performed power analyses on the social background variables (age, gender, education, religiosity, and previous experience with genetics) by using the "pwr" package in R software. The calculations of the sample sizes were performed by using classical parameter values for this type of survey (power = 0.8; significance level = 0.05). We used Cohen's d (Cohen 1988) to characterize effect sizes (for ANOVAs: small: $f = 0.1$, medium: $f = 0.25$, large: $f = 0.4$; for correlations: small: $r = 0.1$, medium: $r = 0.3$, large: $r = 0.5$).

5 Results

5.1 Reliability

We quantified internal consistency reliability using Cronbach's alpha. A satisfactory coefficient is typically > 0.7, although coefficients of 0.6 or greater are acceptable for newly created scales (Nunnally 1978). As can be seen in Table 3, the Cronbach's alpha values for the three sections of the PUGGS questionnaire used in this study did not reach the 0.7 level but were above the 0.6 level. Considering that the PUGGS questionnaire is a newly developed scale and this is the first major study in which it has been used, we regard these coefficients to be acceptable. The low alpha values for PUGGS section 2 suggest that the items could constitute separate subdimensions. This issue is discussed in greater detail below (see the PCA and CFA analyses). If PUGGS sections 3 and 4 are combined, the alpha value increases (alpha = 0.74) suggesting that these two sections might constitute a single dimension encompassing "global knowledge in genetics." In sum, the internal reliability findings suggest that the PUGGS instrument sections have acceptable but low internal reliability and that alternative item groupings may be needed.

Table 3 Cronbach's alpha coefficient for sections 2, 3, and 4 of the PUGGS questionnaire

Section 2 *Belief in genetic determinism*	Section 3 *Knowledge about the complexity of gene-environment interaction*	Section 4 *Knowledge about modern genetics and genomics*
0.67	0.63	0.69

5.2 PCA Analysis

A scree plot (Cattell 1966) indicated that three principal components capture the structure of PUGGS scores for the belief in genetic determinism and knowledge items (Table 4, Fig. 2). The first three components explain 14.3, 9.2, and 7.2% of the overall variance, respectively. As seen in Table 4, component 1 has the strongest loadings for the knowledge items in PUGGS sections 3 and 4 (i.e., *knowledge of the complexity of gene-environment interactions* and *knowledge of modern genetics and genomics*). These results suggest that component 1 is related to general knowledge in genetics. Because the items in sections 3 and 4 were located on

Table 4 Loadings of 42 items on the three first three principal components. Loadings above 0.5 are marked in bold

Items	Component 1 (C1)	Component 2 (C2)	Component 3 (C3)
TT1	−0.21	−0.16	0.31
TT2	0.03	0.21	**0.54**
TT3	−0.11	0.02	0.37
TT4	0.27	−0.35	**0.51**
TT5	−0.04	0.07	**0.62**
TT6	−0.09	**0.58**	0.24
TT7	−0.11	−0.01	0.44
TT8	0.04	**0.78**	−0.03
TT9	0.08	**0.78**	0.19
TT10	0.02	**0.81**	−0.08
TT11	−0.02	0.34	0.32
TT12	−0.03	0.37	**0.52**
TT13	−0.06	0.04	**0.62**
TT14	−0.06	0.02	0.37
TT15	−0.08	0.46	0.26
TT16	0.01	**0.81**	−0.07
TT17	0.29	−0.35	0.39
Q1	**0.57**	0.00	−0.11
Q2	**0.63**	0.19	−0.13
Q3	0.49	0.07	−0.11
Q4	**0.56**	−0.06	−0.06
Q5	0.34	0.13	−0.01
Q6	0.26	−0.17	0.29
Q7	**0.54**	0.20	−0.19
Q8	0.26	0.13	0.04
Q9	**0.56**	−0.03	−0.08
Q10	0.46	0.07	−0.16
Q11	0.34	0.02	0.23
Q12	0.21	0.10	0.18
Q13	**0.55**	−0.16	0.13
Q14	0.26	−0.20	−0.01
Q15	0.45	0.00	0.14
Q16	**0.52**	0.08	−0.19
Q17	**0.56**	0.04	0.11
Q18	**0.66**	−0.04	0.08
Q19	**0.53**	−0.05	0.16
Q20	0.47	0.06	−0.19
Q21	0.37	−0.13	0.20
Q22	0.32	−0.06	0.19
Q23	**0.52**	−0.05	0.17
Q24	**0.54**	−0.05	0.09
Q25	0.24	−0.10	0.24

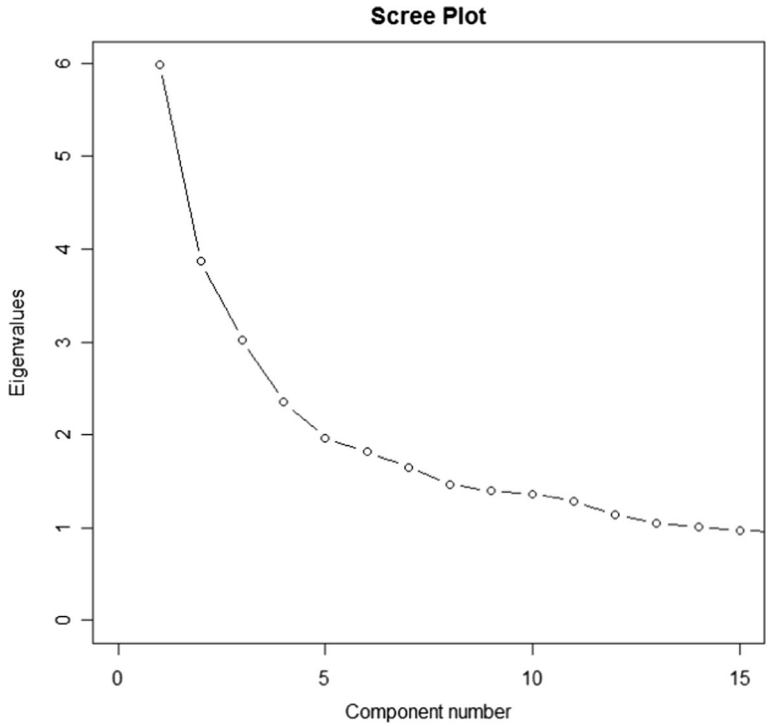

Fig. 2 Scree plot of the eigenvalues of the PCA

the same component, it appears that knowledge of the complexity of gene-environment interaction (e.g., the multifactorial model) is related to knowledge of modern genetics and genomics (e.g., epigenetics and gene activity regulation). The PUGGS knowledge items (Q items) differ in their degree of relationship to component 1. Items Q1, Q2, Q4, Q7, Q9, Q13, Q16, Q17, Q18 Q19, Q23, and Q24 are more strongly correlated to component 1 (coordinates over 0.5) compared to items Q6, Q8, Q12, Q14, and Q25 (below 0.3).

Components 2 and 3 appear to capture items related to beliefs in genetic determinism (Table 4, Fig. 2). However, some differences between components 2 and 3 are also apparent. Component 2 correlates to the PUGGS TT items that to a large degree are environmentally influenced: *interest in fashion*, *political beliefs*, *religious beliefs*, and *addiction to gambling*. In addition, two traits determined by both genes and environment also loaded on this component: *alcoholism* and *violent behavior* (close to 0.46). What these traits have in common is not so much on how they are determined, but more on how they are expressed. All of these traits are to a large extent expressed as social behaviors and not as biological traits per se. For simplicity, from here on, we will refer to component 2 as *social traits*.

Fewer PUGGS items had strong loadings on component 3. The following traits had loadings of 0.5 or higher (see Table 4): *color blindness*, *schizophrenia*, *ADHD*, *severe depression*, and *bipolar disorder*. When including loadings above 0.3, the following traits also related to component 3: *breast cancer*, *height*, *diabetes*, *intelligence in adults*, *asthma*, and *blood groups*. Component 3 relates to traits with strong genetic influence but also includes a few traits with strong environmental influence. However, the traits with moderate to strong loadings on component 3 are biologically or physiologically expressed. For simplicity, from

here on, we will refer to component 3 as *biological traits* even though some degree of overlap with social traits is apparent.

5.3 CFA Analysis

We performed a CFA based on the results of the PCA analysis. The CFA tested the relationships among the three dimensions (components) suggested by the PCA: *knowledge*, *social traits*, and *biological traits*.

Goodness of fit indices for the CFA are shown in Table 5. The chi-square value is significant, but this index is not very reliable because of the large sample size (> 200) and nonnormal data distribution (Schermelleh-Engel et al. 2003). However, the relative chi-square value ($\chi^2/df = 2.5$) is acceptable (less than 5). The RMSEA, AGFI, and SRMR indices indicate good fit. The CFI is an index that compares the CFA model to a null model (a model assuming that all variables are uncorrelated). If the correlations between variables are low, then the difference with the null model will be small. Kenny (2017) suggests that a reasonable rule of thumb is to examine the RMSEA for the null model and make sure that it is no smaller than 0.158. If the RMSEA for the null model is less than 0.158, then an incremental measure of fit may not be informative. Although we found the CFI index to be low, the RMSEA for the null model is 0.094; therefore, the CFI may not be very informative.

Standard estimates of good item saturation on CFA factors include values > 0.4 (see Table 6). By removing some items (e.g., items not explaining the factors enough, which include TT11, TT12, TT15, TT3, TT4, TT7, TT14, TT17, Q1, Q2, Q3, Q5, Q7, Q15, Q16, Q20, Q21, Q23, Q24), the CFI index fits better with the model (0.889). Consequently, future work should examine the centrality of these items for the proposed dimensions in other participant samples.

5.4 Descriptive Data

5.4.1 Belief in Genetic Determinism

The mean values for items related to belief in genetic determinism are shown in Table 7. As is apparent, traits with the highest values (> 4) include *height* (TT1), *color blindness* (TT4), and *blood group* (TT17). At the opposite end of the scale, traits with the lowest values (< 2) include *interest in fashion* (TT8), *addiction to gambling* (TT9), *political beliefs* (TT10), and *religious beliefs* (TT16).

5.4.2 Knowledge of Genetics

PUGGS section 3 measured knowledge of the complexity of gene-environment interactions. The majority of the participants had high scores for this section (see Table 8 and Fig. 3). For items Q2, Q3, Q6, and Q8, the frequencies of correct answers were > 73%. For Q4 and Q7, the

Table 5 Goodness of fit indices

χ^2	*df*	*p* value	RMSEA	AGFI	SRMR	CFI
1249	492	0	0.060	0.986	0.067	0.622

Table 6 Standardized estimates

Left hand side	Right hand side	Standardized estimates
Social	TT6	0.4971
Social	TT8	0.6015
Social	TT9	0.7203
Social	TT10	0.5957
Social	TT11	0.3013
Social	TT12	0.3090
Social	TT15	0.3515
Social	TT16	0.5718
Biological	TT2	0.5392
Biological	TT3	0.2496
Biological	TT4	0.2563
Biological	TT5	0.5744
Biological	TT7	0.3880
Biological	TT13	0.5800
Biological	TT14	0.3120
Biological	TT17	0.1432
Knowledge	Q1	0.3638
Knowledge	Q2	0.3894
Knowledge	Q3	0.3035
Knowledge	Q4	0.4523
Knowledge	Q5	0.2296
Knowledge	Q7	0.3442
Knowledge	Q9	0.4372
Knowledge	Q13	0.4208
Knowledge	Q15	0.3437
Knowledge	Q16	0.3101
Knowledge	Q17	0.4891
Knowledge	Q18	0.5436
Knowledge	Q19	0.3931
Knowledge	Q20	0.2710
Knowledge	Q21	0.2890
Knowledge	Q23	0.3585
Knowledge	Q24	0.3654

Table 7 Mean values and standard deviations of the items measuring belief in genetic determinism (table of traits, $N = 427$)

Item	Mean	SD
TT1	4.15	0.79
TT2	3.03	0.89
TT3	3.34	0.75
TT4	4.72	0.64
TT5	3.51	1.05
TT6	2.11	0.93
TT7	3.62	0.92
TT8	1.36	0.65
TT9	1.52	0.74
TT10	1.30	0.62
TT11	2.61	0.99
TT12	2.66	0.91
TT13	3.49	1.01
TT14	3.40	1.03
TT15	2.21	0.84
TT16	1.24	0.55
TT17	4.91	0.40

1: Only environmental influences; 2: mainly environmental influences; 3: equal genetic and environmental influences; 4: mainly genetic influences; 5: only genetic influences

Table 8 Mean values and standard deviations of the items measuring "knowledge about the complexity of gene-environment interaction" in a scale from 0 to 1, where 1 corresponds to all respondents scoring correct answer ($N = 427$)

All "Do not know" answers are here coded as wrong answers. 1 = True; 0 = False; 0 = Do not know

Item	Mean	SD
Q1	0.26	0.44
Q2	0.79	0.41
Q3	0.73	0.44
Q4	0.56	0.50
Q5	0.58	0.49
Q6	0.88	0.32
Q7	0.62	0.49
Q8	0.85	0.35
Q9	0.41	0.49

correct answers were well over 50%. However, for items Q1 (A gene codes directly for a trait or disease) and Q9 (a person's height is influenced by many different genes), the percentage of correct answers only reached 26% and 41%, respectively.

Results from PUGGS section 4 (knowledge of modern genetics and genomics) indicate much more diverse, but mainly lower, knowledge levels compared to the results from PUGGS section 3 (see Table 9 and Fig. 4). In section 4, only four items displayed high frequencies of correct responses (> 66%): Q11, Q12, Q23, and Q24. Other than Q13 and Q25, all other items had low frequencies of correct answers, and for Q19 (epigenetic changes are caused by mutations), the results were as low as 16% correct. The large differences in correct answers relate to the fact that many of the participants answered "do not know" in genomics and epigenetic items, which was also the case in previous research using the PUGGS (Carver et al. 2017).

5.5 Correlations and ANOVA

5.5.1 The Relationship Between Beliefs in Genetic Determinism and Knowledge of Genetics

We conducted Kendall's correlation analyses to test for significant associations among the three PUGGS sections (*belief in genetic determinism*, *knowledge of the complexity of gene-environment interactions*, and *knowledge about modern genetics and genomics*). We did not find any significant correlations between beliefs in genetic determinism to knowledge about the complexity of gene-environment interaction or to knowledge about modern genetics and genomics. The hypothesis, underlying the second research question, that greater knowledge of multifactorial genetics and/or modern genomics would be associated with significantly lower beliefs in genetic determinism was not supported.

However, we did find a significant correlation between the two knowledge scales: *knowledge of the complexity of gene-environment interactions* and *knowledge about modern genetics and genomics*. This finding aligns with the PCA results (see Table 10).

The PCA analysis also indicated that beliefs in genetic determinism could be divided into two components, *social traits*[4] and *biological traits.*[5] Consequently, we categorized the items into these two groups and examined their correlations with scores on PUGGS sections 3

[4] Alcoholism [TT6], interest in fashion [TT8], addiction to gambling [TT9), political beliefs [TT10], intelligence in adults[TT11], severe depression [TT12], violent behavior [TT15], and religious beliefs [TT16]

[5] Bipolar disorder [TT2],, diabetes [TT3], color blindness[TT4], schizophrenia [TT5], breast cancer [TT7], ADHD [TT13], asthma [TT14], and blood group [TT17]

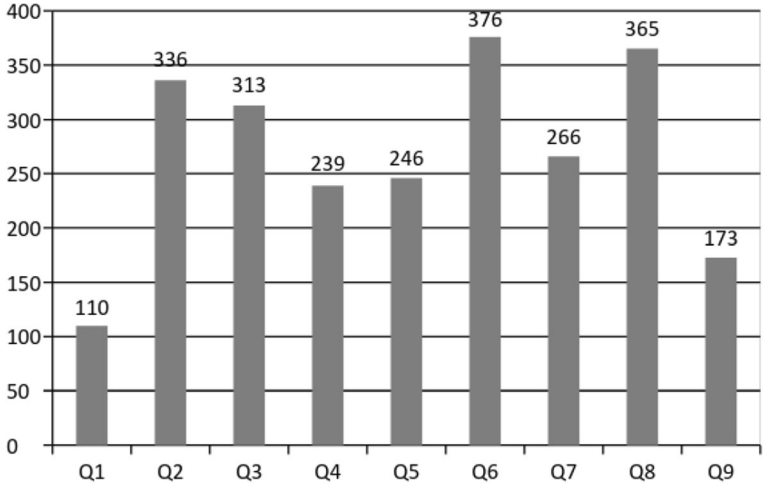

Fig. 3 Frequency of correct answers to the knowledge items of PUGGS section 3 regarding the complexity of gene-environment interactions

(*knowledge about the complexity of gene-environment interaction*) and 4 (*knowledge about modern genetics and genomics*). We used a one-tailed Kendall's correlation test to determine if higher scores for *knowledge in genetics/genomics* were associated with lower scores for *genetic determinism*, but no significant correlations between these scores were found. Only a moderate association between the two knowledge subscales could be established (tau = 0.251, p value < 0.001, see Table 10). The power analysis conducted showed that for a small effect size (0.3 at the upper limit) the minimum sample size would be $N = 84$, which is much lower than the sample tested ($N = 427$).

5.5.2 Social Factors and Beliefs in Genetic Determinism

In order to determine if *age*, *gender*, *education*, *religiosity*, and *previous experience with genetics* had significant relationships with belief in genetic determinism, we conducted

Table 9 Mean values and standard deviations of the items measuring "knowledge about modern genetics and genomics" in a scale from 0 to 1, where 1 corresponds to all participants scoring correct answer ($N = 427$)

Item	Mean	SD
Q10	0.24	0.43
Q11	0.66	0.47
Q12	0.73	0.44
Q13	0.51	0.50
Q14	0.38	0.49
Q15	0.29	0.45
Q16	0.33	0.47
Q17	0.37	0.48
Q18	0.35	0.48
Q19	0.16	0.37
Q20	0.26	0.44
Q21	0.21	0.41
Q22	0.34	0.47
Q23	0.69	0.46
Q24	0.70	0.46
Q25	0.47	0.50

All "Do not know" answers are here coded as wrong answers. 1 = True; 0 = False; 0 = Do not know

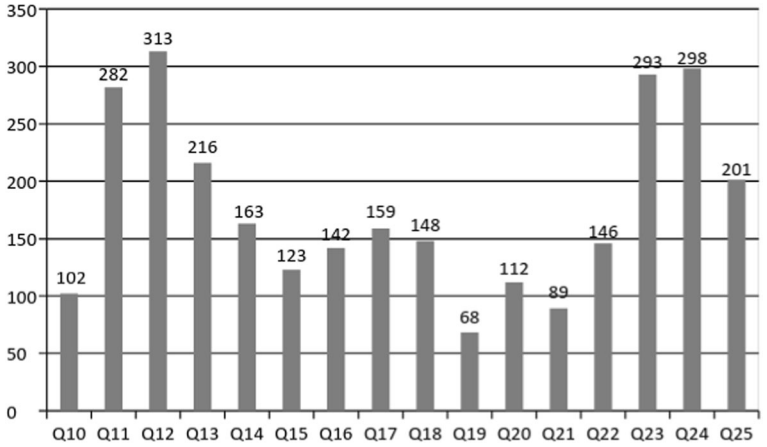

Fig. 4 Frequency of correct answers to the knowledge items of section 4 regarding gene-environment interaction

multiple one-way ANOVAs for all of the social variables against total TT ("table of traits") scores. We also tested for significant relationships for *social trait* scores and *biological trait* scores.

Total TT scores did not produce any significant relationships with age, gender, education, religiosity, or previous experience with genetics. However, participants who indicated that they were greatly influenced by religion had stronger and more positive associations with total TT scores (i.e., more religious students had stronger beliefs in genetic determinism). These results were marginally significant ($p = 0.066$) and suggest that religiosity might be a factor influencing genetic deterministic beliefs. The data set lacked large numbers of highly religious students ($n = 37$) which limits our ability to detect differences in this regard. Further analyses of this group of participants indicated that they had significantly lower knowledge scores for PUGGS section 3 (*knowledge about the complexity of gene-environment interaction*) compared to the group as a whole ($p = 0.047$).

Based on the power analyses conducted on the five groups of social factors, we can conclude that all of the subgroups were large enough to detect a small effect size (see Tables 11 and 12, the sample size is always below $f = 0.25$), except for the group "greatly influenced" by religion, which is composed of 37 individuals. For this group, the effect size that could be detected was medium ($0.25 < f < 0.4$). In other words, these results show that there is a limited risk of concluding that there is no effect when there in fact is one.

Separate analyses of the *social traits* and *biological traits* scores against social factors indicated an effect of age on belief in the genetic determinism of social traits [$F(2, 424) = 8.32$,

Table 10 Kendall's tau scores (between knowledge scores in modern genetics and genomics, and belief in genetic determinism score)

	Knowledge about the complexity of gene-environment interaction	Knowledge about modern genetics and genomics
Belief in genetic determinism	− 0.027	0.022
Knowledge about gene-environment interaction	1.000	0.251***

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$ (one-tailed test)

Table 11 Power analysis conducted on five social factors (religion, age, gender, study, previous experience with genetic issues): expected size of subgroups according to Cohen's size effect benchmark (power = 0.8; significant level = 0.05)

Social factors	Number of groups for each social factor (k)	Minimum size of the groups (N) for a small size effect ($f = 0.1$)	Minimum size of the groups (N) for a medium size effect ($f = 0.25$)	Minimum size of the groups (N) for a large size effect ($f = 0.4$)
Religion	3	322	52	21
Gender	2*	393	64	26
Study	4*	274	45	18
Previous experience with genetic issues	2	393	64	26
Age	3	322	52	21

*The tests (ANOVA and power analyses) have been performed excluding very small subgroups: 2 individuals who answered *others* for the category of *gender* and 4 individuals who answered *others* for *types of study*

$p = 0.0002$]. Tukey's HSD post hoc comparisons indicated that the mean scores for the youngest participants (16–18 years old; $M = 14.17$, SD = 3.39) were significantly lower ($p < 0.001$) than those of the oldest students (22 years or older; $M = 15.87$, SD = 3.63) and significantly lower ($p < 0.05$) than those of the middle age group (19–21 years old; $M = 15.25$, SD = 3.69). Overall, for social traits, older participants showed significantly greater beliefs in genetic determinism compared to the younger participants.

6 Discussion

6.1 Is Genetic Determinism a Coherent Belief System?

Our first research question explored whether beliefs in genetic determinism form a unitary construct. The principal component analysis indicated that PUGGS component 2 primarily relates to traits associated with social behavior and PUGGS component 3 relates to biological

Table 12 Number of individuals in each social factor category

Social factors	Modalities of social factors	Number of individuals
Religion	Greatly influenced	37
	Somewhat influenced	157
	Not influenced	233
Gender	Male	209
	Female	216
Study	Science and technology	112
	Humanities	115
	Health	98
	Arts	98
Previous experience with genetic issues	Yes	124
	No	303
Age	16–18 years old	176
	19–21 years old	124
	22 or older	127

or physiological traits. The CFA also suggested that these dimensions are distinct. The conclusion we draw from these results is that belief in genetic determinism consists of two dimensions. Specifically, participants appear to conceptualize social behaviors as something different and separate from biological traits. This dichotomization of traits into a biological and a social component appears to be related to how the traits are expressed rather than to how they are determined (i.e., the attribution given to genes). These results support the findings of Andreychik and Gill (2014) who identified two dimensions of biological essentialism at the group level related to somatic and behavioral essentialism. In that study, biobehavioral essentialism contributed to prejudice whereas biosomatic essentialism did not. Although our study did not collect the same measures, we found that participants showed lower levels of belief in genetic determinism than expected for the social dimension, while the opposite tendency was shown for the biological component (see discussion below). Hence, our results do not provide support for the existence for genetic deterministic beliefs for behavioral traits at the individual level. Importantly, the study of Andreychik and Gill (2014) was conducted at the group level. Perhaps belief formation differs between these two levels. Morin-Chassé (2014) found that it is possible to influence people's attribution of social behavior to genes at the individual level, inclining them toward genetic determinism, by exposing them to media implying genetic determinism for other social traits in the news articles. Hence, more research is needed to investigate possible differences between beliefs in genetic determinism at the individual and group levels.

The finding of differences in participants' thinking about social and biological traits aligns with the results from an interview study by Condit et al. (2009). They found that laypeople incorporate two sets of public discourse—one that attributes genetic causation to human diseases (e.g., heart disease, lung cancer, and diabetes) and another that attributes behavioral causation to human diseases. The results we found align with these two discourses. Future studies should further investigate these two dimensions: belief in genetic determinism of social behavior and belief in genetic determinism of biological traits.

Although different principal components were associated with social and biological traits, some items loaded on both and could be envisioned as bridges across these two dimensions. Traits linked to the brain and mind (such as intelligence and severe depression) related to both components, but to a higher degree to the biological component (C3). These results need to be explored in further research, but they suggest that when there is a psychological condition that is diagnosable (e.g., ADHD) participants link it to the biological component, whereas characteristics such as intelligence are viewed as overlapping social and biological components. In the study by Condit et al. (2009), no significant relationships were identified between the genetic and behavioral discourses. Our findings concerning the participants' dual perception of traits linked to the mind reveal a possible entry point for exploring such relationships.

The absence of PUGGS scores from other samples precludes comparative claims about the levels of belief in genetic determinism in our participant sample. However, we can compare participant findings to those of scientific heritability studies, as discussed in Section 4.1.2. When comparing the mean scores of the TT items (after transforming them into an index showing the estimated attribution of the genetic factor between 0 and 1, see Section 4.1.2.) with those of published heritability coefficients, we can see that the following traits had lower scores for belief in genetic determinism compared to heritability coefficient studies: *bipolar disorder* scored 3.03, equivalent to 0.51 attribution to the genetic factor in our study, compared to a heritability estimate of 0.85–0.89 in a study by McGuffin et al. (2003) or 0.8 by McGuffin and Sargeant (1991); *schizophrenia* scored 3.51, equivalent to 0.63 attribution to the genetic

factor in our study, compared to a heritability estimate of 0.82–0.85 in the study by Cardno et al. (1999); *alcoholism* scored 2.11, equivalent to 0.28 attribution to the genetic factor in our study, compared to heritability estimates of 0.50–0.60 reported by McGue (1999), and 0.48–0.58, by Prescott and Kendler (1999). For these traits (*bipolar disorder*, *schizophrenia*, and *alcoholism*), participants believed in weak genetic influence, whereas the heritability studies suggested moderate genetic influence and moderate genetic influence, whereas the heritability studies implied strong genetic influence. Also for the traits *intelligence*, *severe depression*, *ADHD*, and *violent behavior*, participants attributed less importance to genetic factors, weighting the genetic factor 0.1–0.2 lower compared to the heritability coefficients (see Table 2).

From these comparisons, we can see that bipolar disorder, schizophrenia, alcoholism and, to a lesser degree, intelligence, severe depression, ADHD, and violent behavior scored lower for genetic deterministic beliefs among the participants when compared to the results from scientific heritability studies. Thus, for the traits related to the human mind, which bridges the gap between social and biological traits, the participants attribute less influence to the genetic dimension compared to heritability studies. However, it is important to acknowledge that the approximations made using heritability coefficients are not equivalent to genetic determination.

For the other social traits (i.e., *interest in fashion*, *political beliefs*, and *religious beliefs*), there are no heritability studies available to ground our comparisons. However, if we extrapolate the results from other items related to social components, we would expect that the participants would respond with low levels of genetic determinism for these traits. To conclude, our results indicate that elevated levels of belief in genetic determinism for social behaviors appear to be lacking for our sample of Brazilian students. Much of the literature expresses the potential problem of widespread beliefs in genetic determinism and a belief in the power of genes to determine social traits (i.e., Dar-Nimrod and Heine 2011; Dambrun et al. 2009; Keller 2000; Nelkin and Lindee 2004). However, our results do not support these suggestions for the sample investigated; on the contrary, the participants attributed less influence to genes in the formation of several traits related to the social component. Once again, we need to emphasize that our study was conducted at the individual level, and almost all psychological studies focus at the group level. This difference could explain the discrepancy between our findings and those from prior studies.

For two traits related to the biological component (i.e., *diabetes* and *breast cancer*), the participants gave larger emphasis to genetic factors compared to results from heritability studies (i.e., they showed genetic deterministic beliefs in the attribution of these traits to genes). *Diabetes* scored 3.34, equivalent to 0.59 attribution to the genetic factor in our study, compared to a heritability estimate of 0.31[6] in a study by Almgren et al. (2011). *Breast cancer* scored 3.62, equivalent to 0.66 attribution to the genetic factor in our study, compared to a heritability estimate of 0.27 reported by Hemminki et al. (2004) and 0.30 in a study by Locatelli et al. (2004). Considering that the PCA suggested that belief in genetic determinism encompasses two dimensions (i.e., social traits and biological traits), this finding suggests that participants upgrade their attribution of biological traits to genes in the same way as they downgrade them when it comes to social traits. However, the fact that the participants did not

[6] This heritability score is representative for type 2 diabetes for the population as a whole. If the population was divided into subgroups, it was found that the heritability coefficient was higher for younger patients (35–60 years) and lower for older patients (60–75 years).

express any elevated levels of genetic attribution regarding, for example, the traits *height*, *color blindness*, and *blood group* shows that this hypothesis requires further exploration. One explanation for why students did not express elevated levels of genetic deterministic beliefs for these traits might be due to ceiling effects[7] of the instrument. Therefore, one suggestion to improve the instrument is to incorporate more items corresponding to biological traits that to a large degree are determined by the environment, as in moderate and weak genetic determinism. This modification would make it possible for participants to provide answers corresponding to greater genetic determinism than expected, thereby limiting measurement ceiling effects. Similarly, it would be of interest to include more social traits that to a large degree are genetically determined. In this way, it would be possible to more carefully test the hypothesis generated in this study, i.e., that genetic factors are given greater importance in biological traits and lesser importance in social traits when compared to heritability studies.

Some of the traits in this study can be compared with the study of Morin-Chassé (2014) who also looked into genetic attribution of traits, though not in relation to environmental attribution. When comparing the findings, it is possible to see that the traits *height* and *breast cancer* are given almost identical genetic attribution in the two studies, while *alcoholism*, *addiction to gambling*, and *intelligence* all differ 15–17% in relation to the participants in Morin-Chassé's study, who gave greater attribution to genes. For the trait *violent behavior*, the difference was 5% in the same direction. Hence, we can see that the Brazilian participants in our study seem to exhibit less genetic deterministic beliefs for social traits in comparison to the US participants in Morin-Chassé's study. Perhaps there are cultural differences explaining belief formation in the case of genetic deterministic views of social traits, as discussed by Castéra and Clément (2014). However, the participants in the study of Morin-Chassé were self-selected, and this could also explain the differences.

6.2 The Relationship Between Knowledge and Belief in Genetic Determinism

One of the main goals of this study was to explore putative relationships between knowledge of genetics and belief in genetic determinism. We expected that higher levels of multifactorial genetics knowledge (cf. Lewontin 2011) would be significantly associated with lower levels of belief in genetic determinism, and that higher levels of understanding of modern genomics and genetics (outlining how genes interact with the environment through the regulation of gene expression and epigenetic mechanisms) would be associated with low levels of belief in genetic determinism. These expectations were not supported by the data we gathered (see Table 10).

These results are in line with some previous work. In a sample of 8285 teachers, Castéra and Clément (2014) showed that measures of belief in genetic determinism were not significantly different between biology teachers and nonbiology teachers. These findings suggest that more knowledge of biology and genetics is not associated with different beliefs or opinions about genetic determinism. In response to these findings, Castéra et al. (2013) suggested that approaching discussions of genetic determinism through philosophy (vs. content knowledge alone) might be a more effective approach for altering students' thinking about genetic determinism. Similarly, Jamieson and Radick (2017) suggested approaching genetics education from a novel perspective. They found that by comparing two groups of undergraduate students, one taking a classical genetics course based on Mendelian genetics

[7] The ceiling effect occurs when the variance of a variable (in this case belief in genetic determinism) is no longer measured or estimated since the mean score is too close to one end of the scale.

and the other taking a course based on developmental processes and their role in bringing about phenotypic variation, the students in the latter group were less deterministic about genes (Jamieson and Radick 2017). Clearly, more work is needed in order to better understand the relationship between forms of genetics education and beliefs in genetic determinism.

Our results suggest that more sophisticated understandings of genetics do not significantly impact beliefs in genetic determinism. By scoring high on PUGGS section 3, the respondents are showing that they understand genes and their function in a manner that is closer to the multifactorial model (Lewontin 2011) and in line with the gene-D concept (Moss 2001, 2003). By scoring low, the respondents are understanding genes and their function as in the gene-P concept (Moss 2001, 2003). Our results indicate that it is possible to understand genes as active determining components and talk about "genes for" but still not develop excessive beliefs in the attribution of the development of traits to genes. These findings are in line with the suggestions by Condit et al. (2009) that different discourse tracks can be used by laypeople as a way to shift reasoning between biological and behavioral reasoning.

PUGGS section 4 (*knowledge about modern genetics and genomics*) was developed to mirror the evolving fields of modern genetics and genomics. Recent work indicates that the entire genome participates in cellular processes; that cell functions involve complex networks of interactions between metabolic components and are thus irreducible to gene products or coding per se; that gene expression is regulated through many processes, including epigenetic ones; and that phenotypes are developed through interactions among genetic, epigenetic, and environmental factors (Carey 2012; Noble 2013; Shapiro 2009). PUGGS section 4 also included some basic genomic knowledge, including ideas that have evolved as a consequence of recent scientific advances. The underlying goal was to investigate whether more up-to-date genomic literacy would be associated with reduced belief in genetic determinism. We did not detect any significant association between elevated knowledge levels of modern genetics and genomics and reduced levels of beliefs in genetic determinism. These are very interesting results, but we need to interpret them cautiously because of the high frequencies of "incorrect" and "do not know" answers in this section of the instrument (section 4). Additional studies with participant samples varying in genetics knowledge are clearly in order.

6.3 The Effect of Social Background Factors on Belief in Genetic Determinism

When investigating the social background factors, we once again found mostly negative results. Hence, the social factors considered in our study were not associated with belief in genetic determinism in the studied sample at the individual level. These results are not entirely unexpected because we did not have the possibility to include some of the more relevant social indicators associated with genetic deterministic beliefs according to the literature (e.g., political beliefs, cultural differences, socioeconomic conditions, etc.; Castéra and Clément 2014; Haslam et al. 2006; Shostak et al. 2009). Note that the items about genetic determinism relating to political beliefs (TT10) and religious beliefs (TT16) showed very strong correlations to component 2 in the PCA analysis (see Table 4). Based on this finding, it could be hypothesized that measuring different groups based on those specific social factors (political and religious beliefs) might reveal interesting results for belief formation related to genetic determinism. Our results provide some support for this hypothesis.

We found that the students who acknowledged that they were greatly influenced by religion tended to show more genetic deterministic beliefs, at the upper limit of statistical significance. Moreover, this group had lower levels (at the lower limit of statistical significance) of knowledge

as probed in section 3 (*knowledge about the complexity of gene-environment interaction*). Hence, here we could establish a possible link between religious beliefs, genetic deterministic beliefs, and knowledge in genetics. These results support the importance of religiosity for the formation of genetic deterministic beliefs, as suggested by Parrott et al. (2004). The sample of this group was very small, including only 37 participants, making it more difficult to find statistically significant effects. Therefore, more studies investigating this issue are called for.

Participant age revealed significant results. The older students showed a higher commitment to genetic deterministic beliefs related to social traits when compared to younger students. This effect was not related to their knowledge level but could possibly be explained by the formation of other ideas or personal experiences. Prior work has suggested that people seek out and change their ideologies based on their own political outlooks (Bell and Kandler 2015). Perhaps the participants form more conservative political ideas as they get older, or reach higher socioeconomic status. These are factors that have been suggested to induce genetic deterministic thinking as a way to preserve and justify advantages for the social group one belongs to (Keller 2005b; Shostak et al. 2009; Suhay and Jayaratne 2012). Alternatively, these results could be explained by older participants having different personal experiences when starting families and thereby evolving more traditional lifestyles, which in turn might influence the way they perceive and explain social traits. These are also very interesting results worthy of further investigation.

7 Implications and Further Research

Our study provides new insights into student thinking about genetic determinism and its relationship to genetics knowledge and social factors. Belief in genetic determinism is a complex construct that seems to have at least two dimensions: a social component and a biological component. We also found that belief in genetic determinism is not necessarily a widespread phenomenon, at least in our sample of Brazilian undergraduates. This is an unexpected result contradicting much of what has been stated in the literature (e.g., Dambrun et al. 2009; Keller 2000; Nelkin and Lindee 2004). However, further work is clearly needed, as our study was conducted at the individual level, whereas much prior work has studied thinking at the group level.

We did find that social factors (i.e., age and religiosity) have importance for the formation of genetic deterministic beliefs. Moreover, since no significant correlations were found with knowledge, our study does not provide empirical support to the argument that to enhance genetic literacy is a way to limit beliefs in genetic determinism. However, there are of course many other incentives for improving genetics education, including a better understanding of gene-environment interactions or current genomic methods and discoveries.

It is important to acknowledge that the results of this study were obtained from a specific participant sample that is embedded in a unique sociocultural context. The participants were freshman university students in Brazil and the findings cannot be generalized beyond this sample. A previous study involving participants from 20 countries revealed that conceptions related to essentialism and genetic determinism can vary dramatically depending on cultural contexts, political stances, and religious beliefs (Castéra and Clément 2014; Castéra et al. 2008). Also, systematic studies in which different social factors are investigated are needed, as argued in the literature (Keller 2005b; Suhay and Jayaratne 2012). It would also be interesting to investigate in what ways beliefs in genetic determinism relate to other values and belief systems. One interesting possibility to explore is Schwartz's (2012) theory of basic human values. It would be of interest to investigate whether beliefs in genetic determinism relate to the second dimension of Schwartz's

basic values, which considers "self-enhancement" and "self-transcendence" values. This dimension captures the conflict between values that emphasize concern for the welfare and interests of others (universalism, benevolence) and values that emphasize pursuit of one's own interests and relative success and dominance over others (power achievement). Some of the literature suggests that genetic determinism can be used as an explanatory model justifying and maintaining unequal social benefits (Shostak et al. 2009; Suhay and Jayaratne 2012), which is very similar to the self-transcendence dimension measured in Schwartz's scale of basic values (2012).

The PUGGS instrument probes beliefs in genetic determinism relating to human traits (not plants, nonhuman animals, or other life forms) (see Table 1). It is possible that beliefs in genetic determinism in humans may not generalize to other living systems. Indeed, our results revealed conceptual divergence between participants' thinking about social and biological traits. A large body of work in evolution and genetics education has explored the roles that taxa and traits play in student reasoning (e.g., Kargbo et al. 1980; Nehm and Ha 2011; Ha and Nehm 2014; Schmiemann et al. 2017). An interesting expansion of our work would be to test different versions of the PUGGS questionnaire framing the items in different taxonomic contexts. This could help to establish whether beliefs in genetic determinism comprise a broad reasoning framework or a more fragmented approach to biological thinking.

Compliance with ethical standards

Conflict of interest None.

Appendix

Table 13 The core ideas of section 3 (knowledge about the complexity of gene-environment interaction) with explanations and corresponding items to the core ideas

Core idea	Description	Corresponding items (item no.)
A. Most traits and diseases are polygenic (caused by many different genes); far fewer traits and diseases are monogenic (caused by changes in a single gene).	Whether participants are aware hat *most* traits are polygenic—determined by many genes.	(Q2) Most human traits and diseases are caused by a single gene. (False) (Q5) Most traits and diseases are influenced by many different genes. (True)
B. One gene can influence several different traits and diseases (pleiotropy).	Awareness that the causal relationship between genes and traits is more complex than a one-to-one relationship.	(Q3) A single gene can influence several different traits or diseases. (True) (Q7) A gene can only influence a single trait or disease. (False)
C. One trait or disease can be influenced by several genes.	Awareness that a trait may be polygenic—determined by many genes.	(Q4) A person's height is influenced by one gene only. (False) (Q9) A person's height is influenced by many different genes. (True)

Table 13 (continued)

Core idea	Description	Corresponding items (item no.)
D. Most traits and diseases are caused by the interaction between many genes and environmental factors.	Awareness of the multifactorial process; that environmental factors also play a role in the gene-trait relationship.	(Q6) Most traits and diseases are caused by environmental factors only (such as diet and lifestyle). (False) (Q8) Most traits and diseases are caused by both genes and environmental factors. (True)
E. A gene's influence on a trait starts with proteins, but the resulting trait can be described on different levels of biological organization; the trait is the outcome of a multifactorial developmental process.	Similar to core idea D, but adds the dimension that there are several "steps" between genes and traits, reflected in a developmental process.	(Q1) A gene codes directly for a trait or disease. (False)

Table 14 The core ideas of section 4 (knowledge about modern genetics and genomics) with explanations and corresponding items to the core ideas

Core idea	Description	Corresponding items (item no.)
F. A genome is an organism's complete set of DNA. It includes both the genes and the noncoding sequences of the DNA; only a small proportion of the human genome consists of protein-coding genes.	This is a definition of "a genome." It tests whether participants are aware that the genome has a lot of noncoding DNA sequences.	(Q10) The genome consists only of the genes in an organism that code for the production of proteins. (False) (Q16) Only a small proportion of the human genome consists of genes that code for proteins. (True) (Q20) Most of the human genome consists of genes that code for proteins. (False)
G. There is no correlation between the number of genes and the complexity of an organism, i.e., humans do not necessarily have more genes than other animals, such as plants, insects, or birds.	This tests whether participants are aware that the complexity of an organism is not just a result of the number of genes, but that there are other mechanisms involved.	(Q13) The human genome contains more genes than the genome of any other living being. (False) (Q21) The human genome has fewer genes than some less complex organisms such as tomato plants and rice. (True)
H. Every cell of the body contains the same genome; what makes cells different is that different genes are expressed.	The main idea here is that all the body's cells contain all our genes, but different genes are active in different cells at any given time.	(Q11) Cells, tissues and organs differ because they have different sets of genes that are activated ("turned on") and deactivated ("turned off"). (True) (Q14) Every cell of the body contains the whole genome. (True) (Q24) Only eye cells have genetic information for eye color. (False)
I. Genes can be turned on and off by the influence of other genes, by substances present in the cell (such as signaling and	This idea is about what makes genes active or inactive.	(Q12) Environmental factors, such as cigarette smoke, can affect gene activity. (True)

Table 14 (continued)

Core idea	Description	Corresponding items (item no.)
transcription factors), or by environmental factors.		(Q23) Genes can be activated or deactivated by other genes. (True) **(Q25)** If a cell lacks a certain substance, such as a vitamin, a gene can be deactivated. (True)
J. The term "epigenetics" encompasses mechanisms for heritable changes (in genetic activity and its subsequent effects) that do not involve alterations of the coding sequence of DNA.	This is a definition of the term "epigenetics." It tests whether participants understand the basic principle of epigenetics; that epigenetic changes do NOT involve changes in the DNA sequence.	(Q15) When someone says something is "epigenetic," it means that you can inherit changes in gene activity without inheriting changes in the genes. (True) (Q18) When someone talks of an epigenetic change, he or she is referring to a large change in the DNA sequence. (False) (Q22) When someone says something is "epigenetic," it means that environmental factors can change part of the DNA sequence. (False)
K. Epigenetic changes can be triggered by environmental influences.	This idea is about what causes epigenetic effects.	(Q17) Epigenetic changes are influenced by environmental factors. (True) (Q19) Epigenetic changes are caused by mutations. (False)

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