OUT OF MY CONTROL: THE EFFECTS OF PERCEIVED GENETIC ETIOLOGY

by

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Abstract

Interpersonal judgements regarding other people's behaviours often involve understanding their underlying causes. People tend to ascribe such causes to some fundamental, vague essence that lies within the individual. The gene has gained prominence in recent decades as a specific instantiation of this essence, although laypeople's understanding of genetics is often inaccurate and overly simplistic. These inaccurate perceptions lead people to engage in genetic essentialism – the tendency to view genes and their associated attributes in overly deterministic and fatalistic terms. This dissertation discusses eight studies that: a) examine the consequences of genetic essentialism in various domains; b) expand on the existing theoretical framework of genetic essentialism by supplementing it with attribution theory; and c) attempt to find ways that can mitigate the impact of genetic essentialism.

Preface

This dissertation is my intellectual product, with guidance from Dr. Steven Heine. I assembled the theoretical framework discussed in this dissertation, designed all projects discussed here, and created all the research materials. I also performed all statistical analyses contained here.

The results from Studies 1 to 3 in Chapters 2 and 3 have been published at *Personality and Social Psychology Bulletin*. I wrote the first draft, and ran all of the analyses included in the manuscript. Dr. Heine and I edited subsequent versions of the manuscript in turn. The citation for the paper is:

> Cheung, B. Y., & Heine, S. J. (2015). The double-edged sword of genetic accounts of criminality: Causal attributions from genetic ascriptions affect legal decision-making. *Personality and Social Psychology Bulletin, 41*(12), 1723-1738. doi: 10.1177/0146167215610520

The results from Study 4 in Chapter 2 and 3 are published as Study 2 in a paper that was published in *Appetite*. All collaborators contributed our own studies to the publication, analyzed our own data, and independently wrote our own sections of the publication corresponding to our own studies. Dr. Heine provided suggestions to each project and helped with manuscript revisions. This paper can be found at:

> Dar-Nimrod, I., Cheung, B. Y., Ruby, M. B., & Heine, S. J. (2014). Can merely learning about obesity genes affect eating behavior? *Appetite, 81*, 269-276. doi: 10.1016/j.appet.2014.06.109

Parts of the theoretical discussions in Chapter 5 were adapted from a paper that was published in *Social and Personality Psychology Compass*. I wrote the first draft and did the

bulk of the literature review. My collaborators and I edited subsequent versions of the

manuscript. The citation for this paper is:

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All studies included in this dissertation were conducted under the approval of the

UBC Behavioural Research Ethics Board. Studies 1-3 and 6-8 were conducted under the

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Certificate Number H12-02470, titled 'Good genes study.' Study 5 was conducted under the

Certificate Number H15-00830, titled 'Learning styles.'

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This piece of writing is a summary of the most recent quarter of my life. This would not have been possible without the help of many people who deserve immense thanks.

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I am forever indebted to everyone who contributed to the completion of this dissertation, including Louise Jingco, Ilan Dar-Nimrod, Matthew B. Ruby, S. Michael Muthukrishna, Stephanie Salgado, M. Robert Muthukrishna, and the numerous people who are not explicitly named here.

Dedication

To my parents, Lawrence and Venny Cheung

To my partner, Louise Jingco

1.   **Chapter 1: Introduction**

> I think I was born with the drive for success because I have a certain gene. I'm a gene
>
> believer; hey, when you connect two race horses you…end up with a fast horse. I had
>
> a good gene pool [for success] so I was pretty much driven (Trump, 2010).

What inferences is Donald Trump making by saying that he has a certain gene, and that he comes from a good gene pool for success? More generally, this quote raises the question of how people interpret genetic explanations for various behaviours and characteristics. This question is especially pertinent given that different types of perceived causes elicit different types of responses. For instance, perceiving schizophrenia as being due to lacking self-control is associated with greater fear towards, and greater desire for social distance from, individuals with schizophrenia (Angermeyer & Matschinger, 2005). On the other hand, perceiving obesity as being due to personal responsibility versus external causes (e.g. the media, parenting) is associated with greater stigma against obesity, and greater perceived deservingness for someone with obesity (Ebneter, Latner, & O'Brien, 2011). These examples clearly illustrate the fact that different causal beliefs can have important implications for judgments in interpersonal contexts.

One important and common causal belief that one may hold is that a trait is genetic in etiology, much like Trump's ascription for his drive for success. Indeed, academics have noted that we are currently in the genomic age – when genetic technology is at the forefront of science, permeating through virtually all aspects of life (Andermann, Blancquaert, Beauchamp, & Déry, 2008; Leister, 2003; Smith, 2005). The availability of genetic information for public consumption has accelerated ever since the Human Genome Project

sequenced a human genome for the first time (Lander, et al., 2001), leading researchers to discover various genes that are associated with a plethora of traits and behaviours. Such discoveries include linking the 5-HTT gene with post-partum depression (Doornbos, et al., 2009), the DRD4 gene with novelty seeking (Ebstein, et al., 1996), and the MAOA gene with aggression (Caspi, et al., 2002; for a review, see Charney & English, 2012). As a result, references to genes have become ubiquitous in our culture, especially in the news media where it is replete with headlines such as "Gene that *makes* human brain unique identified by scientists" (Sample, 2015), "Calls for farmers to help find the genes that *control* their working dog's best traits" (Fitzgerald, 2015), and "Gene *switch* 'key to heart health'" (italics added; British Broadcasting Corporation, 2012). Such references are so pervasive that academics have called this phenomenon DNA-mania (Lippman, 1992; Noble, 2006; Nelkin & Lindee, 1995; but see Condit, 1999; Condit, Ofulue, & Sheedy, 1999; Hedgecoe, 1998). The primary problem with the commonplace proliferation of genetic causes in the media is that these accounts are frequently inaccurate. Indeed, researchers are often frustrated by media coverage of their research due to the oversimplification of their genetics findings (Dupont, 2009). This is particularly evident in a news article on obesity titled "Found – the obesity gene" (Utton, 2003). The simple and provocative title seemingly suggests that there exists one gene that is responsible for obesity. Furthermore, the author uses phrases such as "[t]reatments to 'switch off'…[the hormone associated with overeating,] GAD2…", as though it were a unitary entity that, if switched off, would no longer lead to obesity. This gives readers the impression that researchers have discovered the underlying mechanism behind obesity, even though the gene is actually responsible for a hormone that is associated with overeating, rather than obesity proper (Utton, 2003). A perusal of the paper on which

the article was based instead reveals a more tentative claim, proposing GAD2 as a potential candidate gene for studying obesity, stipulating that the gene manifests in overeating in certain circumstances (Boutin, et al., 2003). Similar references to other genes have also become popularized using overly simplistic labels such as "the warrior gene" (Hogenboom, 2014) and "the infidelity gene" (Carter, 2015), engendering the mistaken perception that these genes are the seats of control responsible for their respective traits.

Contrary to what the headlines and labels above imply, genes rarely control, make possible, or switch on traits. A full exegesis on how genotypes (i.e. one's genetic profile) lead to phenotypes (i.e. the physical manifestation associated with given genotypes) is beyond the scope of the present dissertation; but it is important to note that the relation between genotypes and phenotypes can be highly complex. While virtually all human behaviours and traits are based in genes to some degree (Turkheimer, 1998; 2000), only in rare instances does the possession of a single gene invariably lead to the associated outcome (e.g. autosomal dominant disorders like Huntington's disease or polydactyly). Rather, the vast majority of genetic effects are much more complicated. The expression of a gene may depend on its interaction with other genes, its interaction with the environment, or other forms of epigenetic processes (Jablonka & Lamb, 2006; Meaney, 2010). As many have argued, the overly simplistic genetic descriptions that are common in public discourse may lead people to hold equally simplistic assumptions about genetic effects (Evans & Burke, 2008; Murray, 1998; Nelkin & Lindee, 1995). All of these issues, together, comprise the fourth law of behavioural genetics, which argues that "any human trait has many genetic associations across the human genome; but any individual gene can only account for a very small proportion of variability in the expression of the trait" (Chabris, Lee, Cesarini,

Benjamin, & Laibson, 2015). This dilution of genetic effects greatly mitigates the importance that any individual gene possesses.

The purpose of this dissertation is to understand how people reason about genetic causes, examining the kinds of inferences that people make subsequent to encountering genetic explanations for various behaviours and traits. While academics such as Turkheimer and Chabris continue to discuss the banality of genetic associations with behavioural traits, cultural references to genes have become ubiquitous. Such references have reached beyond the media and into everyday parlance, even in reference to domains that are unrelated to genes, such as in popular books discussing a corporation's DNA (Morgan, 1986) and President Obama's lamentation that the legacy of slavery is part of the American DNA (Obama, 2015). The symbolic use of genes in this manner likely reflects, and co-opts, a broader cultural conceptualization of the gene. Thus, an important scientific endeavour is to examine how people conceptualize the gene, and the consequences that result from such conceptualizations. For instance, if Trump were to believe in the simplistic genetic messages to which one is generally exposed in the media, then one would expect Trump to assume that his particular gene *makes* him more driven, *controls* his motivation, and *switches on* his ambitions. In order to empirically and systematically examine whether or not people reason about genes in this way, this chapter will discuss: a) the concept of psychological essentialism, or people's general tendency to categorize objects in their world by appealing to the underlying entities or essences of objects; b) the construct of genetic essentialism, which uses genes as an instantiation of an underlying essence; and c) how the theory of attribution fits with genetic essentialism. This discussion will establish the structure within which the

package of studies in subsequent chapters of this dissertation will explore people's perceptions of genetic causes.

1.1. How Did We Get Here: Psychological Essentialism

There is a long tradition within philosophy of examining the topic of essentialism, which refers to whether or not objects have a true metaphysical nature, dating back to Platonic and Aristotelian philosophy (Wilson, Barker, & Brigandt, 2007). This metaphysical nature is represented by an object's essence – the deep and unobservable "stuff" that underlies the existence of the object, providing the object with identity, and is perceived as being enduring and immutable in nature (Mackie, 2006; Medin & Ortony, 1989). In other words, objects have identities and belong to certain categories, and membership in those categories is determined by some underlying essence. For instance, an animal is a tiger and it has a "tiger-essence" that allows it to be a tiger, whereas another animal is a falcon and it has a "falcon-essence" that allows it to be a falcon, and the two cannot be interchangeable by nature of having fundamentally different essences. Some debate exists over the validity of such a concept. There are proponents who argue that objects must have essences in order for people to be able to coherently communicate about different objects in our world (e.g. Lowe, 2008; also see Ellis, 2002). In other words, there is a "tiger-ness" to the big striped animal that roars and can climb trees, which allows me to talk about the animal as a tiger rather than a non-tiger. This "tiger-ness" may entail having been born via live birth, having sharp teeth, tree-climbing abilities, and orange fur with black stripes. On the other hand, opponents argue that essentialism is untenable as a construct because it ignores natural variation amongst, and historical changes experienced by, members of a category – especially with regard to variation that is fundamental to the existence of objects (Ereshefsky, 2010; Stone, 2004;

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Wilson et al., 2007). This is particularly applicable in the biological sciences. There can be no "tiger-ness" essence given that substantive variation between individual tigers. For example, while all tigers were born via live birth, not all tigers have orange fur (e.g. a tiger with albinism), and some tigers do not have stripes, even though such important identifying traits as fur pattern is necessary for the normal operation of biological processes such as sexual selection and natural selection. This inherent lack of uniformity, thus, precludes the applicability of essences to any biological kinds.[1] Despite the untenable nature of essences on a metaphysical level, people appear to naturally think about their world assuming that essences exist, as the following sections demonstrate.

While the concept of a metaphysical essence has mostly fallen out of favour within modern philosophy, it has provided an interesting avenue of empirical research for social scientists. Specifically, the debate profiled above relates to whether or not philosophers think that objects contain essences, but it overlooks the fact that the general public readily perceives objects as containing underlying essences, with important consequences for doing so. In fact, researchers across different fields of study have noted a pervasive human tendency to engage in psychological essentialism (Bastian & Haslam, 2006; Gelman, 2004; Gil-White, 2001; Kim, 2013; Medin & Ortony, 1989). That is, people tend to perceive objects as possessing essences, and these essences determine how one categorizes such objects, which has important implications for how people interface with objects around them. Borrowing from the language of metaphysical essentialism, this means that people tend to

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[1] This discussion is only intended to provide the historical precedence for psychological essentialism, and to demonstrate that some debate exists within the literature as to the validity of metaphysical essentialism. As such, this discussion is brief and simplified. Readers who are interested in a more sophisticated overview of the debate involving philosophical essentialism should consult arguments by Bueno and Shalkowski (2015) and Della Rocca (1996).

mentally represent any given object in the world as belonging to certain categories, and membership in those categories is determined by the object's perceived essence.

How, then, do people conceptualize this deep, underlying essence? Medin and Ortony (1989) proposed that people mentally represent essences with what they termed "essence placeholders" – some nebulous container of what an essence for a category is. Gelman (2003) subsequently argued that representations of essences should be divided into two perspectives – specific essentialism, and placeholder essentialism. In accordance with Medin and Ortony, Gelman's placeholder essentialism suggests that there is some vague, ethereal "black box" that people believe to be the crux of category membership. Such a placeholder has been given different names in different fields depending on what may be the most culturally appropriate mental representation: some anthropologists have referred to it as *mana* (Fleising, 2001), while Neo-Confucian religious scholars have called it the *ki* (translated as 'energy'; Baker, 2008). Regardless of the term or the field, they all demonstrate that one way in which people can conceptualize essences is by invoking the existence of some nebulous placeholder.

The placeholder essentialism perspective is juxtaposed with specific essentialism, which holds that there is a concrete entity or unit of information that serves as the basis of category membership. This resembles the argument put forth by some philosophers that, in a metaphysical sense, an object's essence is its hidden structural composition (Kripke, 1980; Putnam, 1996). While such compositions may be hidden from apparent observation, careful scientific inquiry would ultimately uncover the true nature of these essences, which will be tangible in the future. In accordance with that line of thought, specific essences exist in some form in various fields, such as the atomic configurations that form the basis of chemistry

(Petrucci, Harwood, & Herring, 2002), and the brain for some in psychology (Gazzaniga, 2011; Haslam, 2011). Regardless of whether people engage in placeholder or specific essentialism, it is apparent that there are a variety of ways in which people engage in psychological essentialism.

1.1.1. Consequences of Psychological Essentialism

Much research has now demonstrated that psychological essentialism is associated with several kinds of cognitions. One such cognition is to perceive surface/observable characteristics as proxies for, or physical manifestations of, underlying identity-determining essences (Prentice & Miller, 2007). In other words, the essence allows us to infer a wide variety of attributions about category members in terms of expected behaviours and physical characteristics. If asked to think about "birds", that likely conjures up an image of a feathery winged bipedal animal in flight, presumably because these characteristics are manifestations of some "bird-essence". It is of note, however, that changes to surface characteristics do not accompany corresponding changes to the essences. According to psychological essentialist thinking, it matters not what is *externally* done to the bird; it is what is presumed to exist *internally* that categorizes different objects. For example, if a bird were to have contracted some terrible disease that caused its feathers to shed rendering its wings to be non-functional and confining it to a life of endlessly waddling up and down city streets, some "bird-essence' within the animal would still put it under the purview of ornithology. In other words, if it had not been for the disease, it is expected that the bird-essence would have led to the development of feathers. Indeed, inductions based on essences may be mistaken in certain circumstances as surface/observable characteristics are not necessarily perfect indicators of deep essences (Haslam et al. 2006; Medin & Ortony, 1989). One important observation here

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is that the layperson largely appears to be more lenient than philosophers in ascribing essences by perceiving more general, approximate essences. That is, birds have some bird-essence, regardless of the specific species, thus allowing for variability that does not invalidate category membership.

Another cognition that people engage in is the perception that different essences are highly distinct from each other, consequently leading people to perceive members of the same essentialized category are similar to each other, and fundamentally distinct from members of other categories (Dar-Nimrod & Heine, 2011). To illustrate, a bird-essence entails wings, flight, bipedalism, and egg-laying. In contrast, a fish-essence might entail fins, birthing by spawning, and the ability to breathe under water. As such, one would probably place a robin and a sparrow, both presumably containing the same bird-essence, in the same category. By the same token, one would probably place a pink salmon and a chinook salmon, both containing the same fish-essence, into the same category. However, it is unlikely for someone to categorize a robin and a pink salmon together, but rather see them as being fundamentally distinct from each other because they presumably possess different essences.

A third cognition is that, people perceive persistence in the essences that underlie categories, and membership in those categories, such that they are seen as being both involuntarily obtained and immutable (Haslam & Levy, 2006; Prentice & Miller, 2007). That is, a sparrow has a bird-essence simply by existing – it did not exercise volition in obtaining a bird-essence, and had no control over what essence it would possess. Moreover, this essence is seen as being unchanging, such that a sparrow would not be able to change its essence to obtain other essences. As a reflection of these perceptions of essences, membership within essentialized categories is similarly perceived as being both involuntary and immutable. In

other words, a sparrow had no choice in being a member of the bird family, with membership simply being the result of its existence. Furthermore, as essences are seen as immutable, there is also the expectation that a sparrow cannot somehow replace its bird-essence with a fish-essence.

One other cognition is that people tend to believe that an essentialized category is a natural kind (Prentice & Miller, 2007; for a review, see Haslam & Ernst, 2002). In other words, there is no arbitrariness in the grouping of objects into a category; rather, there is some objective natural property beyond human interpretation that dictates what objects belong to the same category. In a world with no humans, some natural objective property would still place sparrows with robins into one category, and pink salmon and chinook salmon into another category.

Essentialist thinking appears to be a universal psychological process, extending beyond Western countries (Kashima, et al., 1995; Peng & Knowles, 2003). It has, thus far, been documented around the world including amongst various adult Western populations (e.g. Haslam & Levy, 2006), patrilineal Mongolian nomads (Gil-White, 2002), and indigenous South African children (Giles, Legare, & Samson, 2008). For instance, Gil-White (2002) presented Torguud nomads in Mongolia with a scenario of a child with an ethnic Kazakh father but was raised by ethnic Mongol parents. The child learned the Mongol language and customs, never met Kazakhs, and thinks that his Mongol parents are his biological parents. The nomads were more likely to still perceive the child as a Kazakh rather than a Mongol, perceiving the child as having some deeper, explanatory properties that fundamentally identifies him as a Kazakh. Moreover, Giles et al. (2008) found that South African schoolchildren exhibited even stronger psychological essentialism than American

schoolchildren, perceiving people's traits as being more stable (i.e. immutable) and inborn (i.e. natural). These results, in conjunction with other evidence showing essentialist thinking amongst children as young as four years old (Gelman, 2004; Heyman & Gelman, 2000; for a review, see Hirschfeld, 1997), suggest that psychological essentialism appears to be a fundamental human psychological process that develops early in life.

In addition to psychological essentialism being a universal process, it also has important implications for people interacting with members of other essentialized categories, which may have both negative (e.g. greater stereotyping and prejudice) and positive (e.g. greater acceptance) consequences. For example, in the domain of race perception, European Americans who essentialize race more exhibit greater levels of prejudice against African Americans compared to those who essentialize race less (Jayaratne, et al., 2006). Furthermore, greater race essentialism is generally associated with a greater readiness to categorize people based on racial groups as opposed to categorizing people based on other themes (e.g. occupation; Chao, Hong, & Chiu, 2013). In terms of mental health, researchers have shown that greater essentialist thinking is associated with expressing more prejudice against, and less compassion towards, people with mental disorders (Howell, Weikum, & Dyck, 2011; Howell & Woolgar, 2013). On the other hand, other researchers have also demonstrated that essentialist thinking is associated with showing greater acceptance for someone with certain types of mental illnesses by assigning less blame to the patient (Deacon & Baird, 2009; Schomerus, Matschinger, & Angermeyer, 2014). Essentialist thinking can thus be shown to have important real-world implications.

1.2.   What Are We Doing: Genetic Essentialism – a Specific Essentialism

As discussed above, one way in which people conceptualize essences is to engage in specific essentialism, or to perceive that the essence that underlies a category can be found in the underlying composition of objects in a category (Gelman, 2003). Dar-Nimrod and Heine (2011) adopted this perspective by contending that one type of essence that gives rise to essentialized categories lies within a very tangible and deceptively simple entity – the gene.

The gene is an attractive specific essence because it is a simple entity whose meaning people can easily intuit regardless of how much genetic knowledge one has. Even Nobel laureate Walter Gilbert often gives lectures in which he produces a compact disc and tells audience members, "This is you," in reference to his vision that their personal genomes can one day be read from a disc, encompassing the totality of one's identity (Dreyfuss & Nelkin, 1992; Taussig, 2009). This simplistic appeal can be dangerous if people ascribe it undue power based on insufficient understanding of genetic concepts. Indeed, knowledge of genetics has been found to be deficient amongst the public (Lanie, et al., 2004) and medical professionals such as general practitioners and gynecologists (Baars, Henneman, & ten Kate, 2005), albeit to a lesser extent.

Numerous researchers (e.g. Dar-Nimrod & Heine, 2011; Nelkin & Lindee, 1995; Phelan, 2005) have suggested that this lack of understanding for genetic concepts lead people to engage in a certain suite of cognitive responses, resembling the general essentialist cognitions reviewed previously. Specifically, such cognitions entail that (a) people perceive surface characteristics as reflecting the workings of a single underlying gene; (b) objects in the same category share the same genes, thus are perceived as being similar, but distinct from objects in other categories; (c) genes, and thus membership in genetically essentialized

categories, are seen as uncontrollable and immutable; and (d) genetically essentialized categories are seen as ahistorical natural kinds. Dar-Nimrod and Heine (2011) proposed that people consider genes to be, in Gelman's (2004) terms, a specific essence, drawing upon observations of the similarities between how people view essentialized categories and people's lay perception of genes.

According to the genetic essentialism framework, learning about genetic behavioural explanations activates people's psychological essentialist ways of thought, leading them to engage in a set of predictable cognitions. Much like the psychological essentialist perception that surface characteristics reflect underlying essences, the first cognition associated with genetic essentialism is specific etiology, or the perception that genes are the fundamental causes of particular traits. A gene is, thus, seen as being solely responsible for the physical manifestation of an associated trait, minimizing the contribution of environmental or experiential factors. By extension, this means that lacking a certain gene will preclude one from developing the associated trait. In other words, people perceive a nearly perfect correlation between genes and their associated traits, which Conrad (1999) refers to as the "one gene one disease" perception. For example, a person will be expected to have dark skin if she has the relevant gene, but not if one is without the same gene. The reverse inference is also assumed, such that someone with dark skin is assumed to have relevant genes, while the absence of this trait is associated with the lack of the relevant genes. For Trump to suggest that he has a gene for success, he is arguing that his success is due purely to his genes, not to any of the environmental factors that allowed for him to succeed. As a corollary, if he did not have that gene, he would not be successful.

Corresponding to the perception of within-category similarity associated with psychological essentialism, another cognition resulting from exposure to genetic behavioural explanations is the perception of homogeneity and discreteness. That is, people perceive there to be rigid boundaries around a group based on a perceived shared genetic origin. As a consequence of such rigidity, people who have the same genetic foundation are seen as highly similar to each other, and fundamentally different from people without the shared gene. Thus, one is likelier to perceive people with the same skin colour as being highly similar due to the assumption that people who share the same trait must possess the same genetic marker. This perception then leads observers to view this group as being fundamentally different from a group in which members share a different skin colour, effectively granting different groups of people identities on the basis of some perceived genetic difference or similarity. Thus, Trump may identify himself as being someone who is fundamentally similar to other successful people, but fundamentally different from people who are not successful.

A third cognition is that the genetic process that links genes with outcomes are seen as immutable and determined, which is akin to the psychological essentialist perception that essences and their outcomes are seen as involuntary and immutable. According to genetic essentialist thinking, leaving the gene to its supposed natural developmental trajectory will invariably lead to the development of the associated trait. Additionally, this process is also seen as being non-malleable, such that no external force, including personal control, can alter the expression of a trait given the presence of the associated gene. This leads to a fatalistic perception of the genotype-phenotype relationship. For example, genetic essentialist thinking assumes that if someone is born with genes that code for dark skin, then little can be done to

change the underlying genetic mechanism. This means that the individual is powerless to change their skin colour, such that she is bound to be dark-skinned. This sentiment of determinism is most poignantly captured by a controversial set of statements that Dr. James Watson, famous for his role in discovering the helical structure of DNA, made regarding racial differences in intelligence. Specifically, he was "inherently gloomy about the prospect of Africa" given that we cannot "anticipate the intellectual capacities of peoples geographically separated in their evolution should prove to have evolved identically. Our wanting to reserve equal powers of reason as some universal heritage of humanity will not be enough to make it so" (Milmo, 2007). Watson perceives intelligence to be a function of the evolutionary history of the world's populations, which is necessarily encoded within one's genes. While expecting different populations to have had different evolutionary pressures is not essentialist, his expression of skepticism communicates a fatalistic perspective that intelligence is determined by one's genes in such a way that makes racial differences in this domain immutable. Following this line of thought, Trump may feel like he can rest assured that he can put in minimal effort and still attain success.

The last cognition proposed by Dar-Nimrod and Heine is akin to the psychological essentialist perception of natural kinds, which is the tendency to moralize natural characteristics and traits. That is, people engage in the naturalistic fallacy by perceiving a genetically caused outcome to be a natural outcome, which is more morally acceptable than an unnatural outcome (Ismail, Martens, Landau, Greenberg, & Weise, 2012). This is particularly the case in situations where a trait is perceived to be potentially affected by volition. Because volition is seen as a key component of moral responsibility (Easter, 2012; Shultz, Wright, & Schleifer, 1986), the potential for genes to negate volition absolves the

moral responsibility of individuals, thus making behaviours appear more normal and acceptable. Along these lines, people generally view others as "readouts of their genes" (Nelkin & Lindee, 1995, p. 6), or compare a genome to "the Bible, the Holy Grail, and the Book of Man…suggesting [the genome to be] a powerful guide to moral order" (Nelkin & Lindee, 1995, p. 8). In other words, people are perceived to be merely passive physical vessels through which genomes are expressed, harkening back to Walter Gilbert's perception that a CD containing one's genetic code is enough to describe the entirety of a person. As such, their behaviours and traits are being expressed as nature or god intended, precluding any involvement of volition and free will from the individual. Thus, Trump may assume that it only makes sense that he is successful, and is simply fulfilling his moral destiny of being successful. This negation of personal control may have important implications in a variety of different domains. For example, there exists a preponderance of free will within the criminal justice system such that crimes are perceived as being commissions as a result of one's volition (Coffey, 1993). Therefore, when considering criminal behaviour, perceiving an underlying genetic cause should lead to more positive (or less negative) moral ascriptions of perpetrators, resulting in more lenient perceptions of criminal guilt.

1.2.1.   Consequences of Genetic Essentialism

There is evidence that the way in which people think about a trait is affected by whether or not the trait is believed to have genetic associations, particularly given that people reason fatalistically about genes (Gould & Heine, 2012). One important domain in which this plays a role is with regard to perceptions of mental health. Many academics have argued that medical professionals should focus on the genetic etiology of mental illnesses when communicating with the public because this should diminish perceived personal control in

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developing the illness, thereby decreasing stigma against people who have mental illnesses (e.g. Austin & Honer, 2004; Hill & Sahhar, 2006). This would be reflective of the genetic essentialist perception that there is an immutable and determined quality to depression if it has a genetic origin, leading people to perceive this as simply a natural development amongst affected individuals. While there is some evidence for this for certain aspects of specific mental health conditions (e.g. sympathy for someone with depression; Schomerus et al., 2014), the general thrust of the literature suggests the contrary. In fact, a large number of studies have demonstrated that such messages actually exacerbate stigma. For instance, thinking that depression is genetically caused leads people to perceive the patient's family members as being at a greater risk of developing the same illness (Phelan, 2005; Phelan, Cruz-Rojas, & Reiff, 2002). Similarly, thinking about schizophrenia as a genetic mental illness leads people to stigmatize family members of patients (Bennett, Thirlaway, & Murray, 2008), desire greater social distance from patients (Rüsch, Todd, Bodenhausen, & Corrigan, 2010), and express more fear of them due to increased perception of danger posed by someone with schizophrenia (Schnittker, 2008). These fearful responses are also reflective of genetic essentialist cognitions, primarily with regard to perceiving people with mental illnesses as being fundamentally different from people who presumably do not have mental illnesses. By instantiating mental illnesses into the form of genes, people group families together even more strongly by regarding them to be highly similar to each other, thus warranting associative stigma to spread outwards from the person with mental illness. Furthermore, as perceived differentness and dangerousness of people with schizophrenia are associated with greater fear responses (Levey & Howells, 1995), it follows that attributing schizophrenia to genetic causes would similarly engender feelings of fear.

Another way in which genetic essentialism has been shown to lead to important

consequences is with the criminal justice system (CJS)[2]. As previously discussed, one of the

most important and relevant concepts in the CJS is free will in relation to determining

criminal guilt and punishment. This concept is codified in the CJS by requiring that the

prosecution prove that the defendant had *mens rea* during the commission of the crime (i.e.

the malicious intention to act in contravention to the law; Aharoni, Funk, Sinnott-Armstrong,

& Gazzaniga, 2008). As an extension of this requirement, defendants are allowed to argue

that they lacked *mens rea* by showing that they acted not out of free will but by some

uncontrollable compulsion. Indeed, several court cases around the world grabbed media

attention because the defendants were either given a shorter prison sentence than the original

punishment (Forzano, et al., 2010; Owens, 2011), or convicted of a lesser charge on account

of genetic evidence with one juror saying that "…[a] diagnosis is a diagnosis, it's there. A

bad gene is a bad gene" (Hagerty, 2010). Given that genetic evidence has become

increasingly utilized in trials since the 1990s (Denno, 2006), these examples are particularly

pertinent to the longstanding debate about the applicability of genetic evidence in the

courtroom (Berryessa & Cho, 2013; Hoffmann & Rothenberg, 2007). Thus, the outcomes of

the court cases above highlight how genetic evidence affects the CJS, particularly in

naturalistic settings.

Beyond anecdotal evidence, some researchers have systematically examined how

genetic evidence affects decisions within a CJS context. For instance, biological explanations

for crimes lead to weaker ascriptions of willpower to a criminal compared to experiential

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[2] The discussion in this section also applies to most industrialized Western nations, particularly those that follow English common law. For simplicity's sake, this dissertation will focus on the intersecting parts of the Canadian and American criminal justice systems as they are the most relevant for the samples in the subsequent experimental chapters.

explanations for crimes (Monterosso, Royzman, & Schwartz, 2005). A similar, but more ecologically valid, study found that judges who were provided with expert testimony on the genetic cause of a defendant's behaviour perceived less aggression from the defendant, and subsequently gave a shorter prison sentence (Aspinwall, Brown, & Tabery, 2012). Moreover, men who were provided with a genetic explanation for sexual violence were more lenient on a rapist compared to men who were given a sociocultural explanation for sexual violence by prescribing a lower prison sentence for the perpetrator (Dar-Nimrod, Cheung, Ruby, & Heine, 2014; but see Appelbaum & Scurich, 2013; Raad & Appelbaum, 2015). Taken together, these studies suggest that the use of genetic evidence in the CJS would lead people to engage in genetic essentialist thoughts, thereby taking moral responsibility away from perpetrators, ultimately leading to the prescription of more lenient punishments. There exists a contradiction within this rationale, however, because ascribing less willpower and control to one's criminal behaviour should also lead to greater expected recidivism. If part of criminal punishment is to protect the public from the perpetrator (Wilson & Herrnstein, 1985), then that function should be especially pertinent when a perpetrator is expected to reoffend. It should follow, then, that genetic evidence should have led to stricter punishments. This contradiction has been the subject of much legal debate (see Beecher-Monas & Garcia-Rill, 2006; Tehrani & Mednick, 2000), with little empirical evidence that can address this issue. I will discuss how this unresolved contradiction plays out amongst laypeople in subsequent experimental chapters.

1.3. What Else Is Missing: Theory of Attribution

The current genetic essentialism framework has contributed greatly to our understanding of how information about genetic etiological accounts can have important

consequences in various domains; however, it does not encompass the complete suite of biased cognitions. As the aforementioned cognitions would suggest, exposure to genetic information affects how someone perceives the resultant trait or behaviour; but it may also be the case that such information capitalizes on how people generally think about genes on a more fundamental level. Perhaps genetic essentialism is not just about how people understand the consequences of genes, but also about how people understand the nature of genes as a causal agent. In other words, the current framework of genetic essentialism can be more comprehensive by incorporating the causal attributions that people may make about genetic causes.

Drawing causal attributions reflects people's motivation to interpret phenomena around them, including events and other people's behaviours. This motivation hinges on one important principle – the idea that humans seek to predict and control their environment, which requires people to ascribe unchanging underlying properties to the causes of events and behaviours that otherwise seem unstable and uncontrollable (Heider, 1958). Without making such attributions, the environment around us becomes seemingly unmanageable and uninterpretable.

Attribution theories are a class of theories that researchers have proposed to explain how people draw explanatory inferences for various phenomena in their environment, including correspondent inference theory (Jones & Davis, 1965) and the covariation model (Kelley, 1967). The theory that has received the most attention within this class is Weiner's theory of attribution. Based on the motivation and attributional work by Thorndike (e.g. 1911), Rotter (e.g. 1966), and Heider (1958), Weiner and colleagues (Anderson, Krull, & Weiner, 1996; Weiner, et al., 1971) formulated an intended grand and unifying theory about

self-attributions within an achievement context. According to this theory, people's causal

attributions for their successes and failures hinge on several dimensions: causal locus; causal

stability; causal control; and causal specificity (Abramson, Seligman, & Teasdale, 1978;

Weiner, 2012). The causal locus dimension describes whether the perceived cause of an

action is internal or external to the actor. In other words, a cause can be seen as either

originating from within the actor versus coming from a factor outside of the actor. For

example, self-attributions for academic successes and failures can be seen as resulting from

internal causes such as one's ability or effort, or from external causes such as luck or test

difficulty.

Causal stability refers to whether the cause of a particular behaviour is expected to be

present and impact the individual over time. A stable cause is one which is expected to

endure, such as lacking the ability to perform well on an exam (assuming that one perceives

ability as a static trait that does not change over time). Conversely, an unstable cause is one

which is expected to fluctuate over time, such as loud noises hindering satisfactory

performance on an exam. Furthermore, according to Weiner, the causal stability dimension

determines whether people's expectations and feelings about a particular behaviour will shift;

that is, if a cause is stable, then expectations about the associated behaviour is not expected to

change, whereas an unstable cause should be associated with changing expectations. For

example, if the cause of failure is seen as being due to a stable cause, then the expectation of

failure will persist for future tasks of a similar nature. In contrast, if the cause of success is

seen as being due to an unstable cause, then one would not have the same expectations of

success in the future.

A third dimension is causal control, which refers to the degree to which the cause of some behaviour is perceived to be subject to one's volitional control. To the extent that a cause is seen as being controllable, the actor feels that there is potential for effecting change in the cause. If a cause is seen to be uncontrollable, the actor feels that little can be done to change the cause. For example, a controllable cause may be represented by a lack of effort leading to poor performance on a task, whereas an uncontrollable cause may be evidenced by bad luck.

Other researchers have proposed an additional fourth dimension, a global-specific dimension (henceforth referred to as causal specificity), which relates to the extent to which a cause will affect outcomes in various domains (Abramson et al., 1978). A global cause is expected to affect outcomes across domains, whereas a specific cause is only expected to affect outcomes in one particular domain. For example, a lack of ability in chemistry can be considered a specific cause, thereby affecting performance only on a chemistry exam. On the other hand, a global cause may be a lack of intelligence, which would likely negatively affect one's performance on exams across multiple subjects. Although this dimension was neither posited, nor examined in detail, by Weiner, he concurs that there are likely other dimensions that contribute to people's attribution-making process, including the global-specific dimension (Weiner, 2012).

While it may be tempting to draw relations between the different causal dimensions, such as assuming causal locus to be related to causal control (i.e. thinking that an internal cause is also uncontrollable), Weiner's theory treats them all as being independent from each other (Weiner, 2012). Furthermore, even though Weiner's theory was originally formulated to explain self-perceptions within the context of academic performance, it has since been

applied to interpersonal perceptions in a variety of scenarios including understanding corporate and organizational behaviour such as leadership and group dynamics (Chang & Chen, 2012; Martinko, Harvey, & Dasborough, 2011), differentiating between various forms of psychopathology (Weiss, Süsser, & Catron, 1998), homosexuality and bisexuality (Haider-Markel & Joslyn, 2008; Lewis, 2009), and obesity (Crandall & Moriarty, 1995; Sikorski, Luppa, Brähler, König, & Riedel-Heller, 2012). This theory has, thus, been demonstrated to be effective at explaining a variety of phenomena.

1.3.1.   Causal Attributions for Genetic Causes

The prospects of incorporating Weiner's attribution theory into a genetic essentialism framework were already apparent to Weiner early on, albeit unintentionally, when he posited that "…a genetically determined aptitude will not be perceived as controllable by a failing math pupil" (Weiner, 1985, p. 551). Furthermore, he specifically referenced genetics as an intuitively uncontrollable cause, arguing that such an attribution should lead to reduced perception of responsibility and greater sympathy for certain populations (without specifying which populations; Weiner, 2012). Through these statements, he expressed the recognition that the presence or absence of a gene also signalled the presence or absence of one's ability to control the associated behavioural outcome. A perusal of Dar-Nimrod and Heine's (2011) genetic essentialism framework reveals a few relevant connections with Weiner's attribution theory. First, the cognition of specific etiology, or the perception that behaviours are caused only by genes rather than the environment, should be associated with an internal causal attribution given that genes inherently reside within a person. Thus, people who encounter genetic etiological explanations should engage in internal causal attributions.

Another connection involves the cognition of immutability and determinism, or the perception that the process through which latent genes manifest in surface traits cannot be changed. If someone engages in this cognition, then possessing a gene for depression means that there is little that one can do to change the developmental trajectory of depressive symptoms. Furthermore, such an effect should continue throughout her lifetime if the effect is deterministic. As such, this cognition should be related to uncontrollable and stable causal attributions.

Some work has used attribution theory and genetic essentialism in the same study; however, a close examination of the state of research in this regard poses two outstanding issues. One issue is that researchers tend to pit the two frameworks against each other to determine whether one is more predictive of important outcomes than the other. In other words, researchers try to conclude whether one is more useful than the other, rather than whether the two are complementary. For instance, Phelan (2005) concluded that the genetic essentialism perspective is more predictive than attribution theory because genetic etiological ascriptions for mental illnesses were associated with expected consequences of genetic essentialism (e.g. perceived persistence and seriousness), but not with expected consequences of attribution theory (e.g. perceived blame). On the other hand, Boysen and Gabreski (2012) found that both frameworks make complementary predictions, such that genetic etiological ascriptions of mental illnesses were associated with less blame. This, in turn, was correlated with perceived immutability. These two outcome variables were seen as reflecting consequences of attribution theory and genetic essentialism, respectively. Little attention is paid, however, to the possibility that genetic essentialist biases may actually include engaging in a certain suite of causal attributions, engendering a more integrative approach.

The lack of attention to an integrative approach may be related to a second issue, which is the fact that researchers tend to examine downstream consequences of causal attributions (e.g. pity, sympathy) while ignoring the more basic cognitions (i.e. the actual causal attributions). As an example of this downstream focus, patients with bulimia nervosa felt that communicating a genetic etiology for eating disorders to others would exacerbate stigma by making it seem like a permanent part of them, highlighting the association between genetic causes and stable causal attributions, although the researcher never asked for the actual causal dimensions (Easter, 2012). Furthermore, a qualitative study found that some patients felt that genetic explanations would lead to the perception that it is an inherent part of them. That leads others to label them as "anorexics" or "bulimics", and assume that a specific cure exists to treat that specific condition. As such, they theoretically evince internal and specific causal attributions, despite the lack of direct measures of causal dimensions. Similarly, ascribing lung cancer to genetic causes leads people to perceive less volitional control in developing lung cancer, subsequently showing more pity for someone with that condition (Harmann, Howell, & McDonald, 2013). This highlights the relation between genetic ascriptions and uncontrollable causal attributions. Such clear links, however, are rarely so clearly made in quantitative studies. As a result, linking outcomes to particular causal attributions is still conjecture without measuring participants' actual causal attributions. As such, to properly examine the applicability of attribution theory to studying how people perceive genetic ascriptions, it may be more appropriate to study how people's actual causal attributions change based on perceived genetic causal explanations.

1.4.   Where Do We Go From Here: Overview of Experimental Chapters

To recapitulate, there is a universal human tendency to perceive people as possessing unchanging, ahistorical essences that grant them an identity, separating them from others who have different essences (e.g. Gil-White, 2001). Different cultures have different instantiations of this essence, with genes emerging as a popular one, partly owing to the immense media attention on behavioural genetics research that often oversimplifies genetic effects (Nelkin & Lindee, 1995). This leads people to engage in a particular set of cognitions, a phenomenon known as genetic essentialism (Dar-Nimrod & Heine, 2011). Researchers have noted some interesting implications of genetic essentialism, including for mental health stigma (e.g. Phelan, 2005) and legal decision-making (e.g. Dar-Nimrod et al., 2014). The genetic essentialism framework has immense potential for examining how people's understanding of behavioural genetics has implications for a variety of other domains.

One additional theoretical approach that can benefit the genetic essentialism framework is attribution theory (Weiner, et al., 1971). Integrating the two frameworks would suggest that people's genetic essentialist cognitions may also include people's causal attributions about genes. Based on extant research, one should expect genes to be seen as being causally internal, stable, uncontrollable, and specific (e.g. Harmann et al., 2013), although much of this research has been based on proxies of causal attributions rather than the attributions proper.

The following experimental chapters seek to investigate several questions in relation to genetic essentialism. Specifically, Chapter 2 examines the effect of genetic essentialism on a variety of domains including expanding on previous research regarding legal decision-making; obesity-related cognitions; and beliefs about educational efficacy. Chapter 3

examines how genetic etiological ascriptions affect various causal attributions. While it is important to examine how genetic essentialism affects cognitions in various domains, it is also important to study how to mitigate genetic essentialism given that these cognitions are based on inaccurate perceptions of genetic effects. As such, Chapter 4 describes attempts at mitigating genetic essentialist cognitions, using legal decision-making as a case study.

2.   Chapter 2: Examining the Effects of Genetic Essentialism

In order to fully grasp the genetic essentialism framework, one must understand the consequences that genetic essentialist cognitions have, instantiated within specific important domains. The previous chapter profiled some instances in which genetic essentialist cognitions have played a role, such as decreasing and increasing different aspects of mental health stigma, and leading to more lenient punishments within the criminal justice system. The present chapter examines the impact of genetic essentialism within a greater breadth of domains, and further expands on existing work linking genetic essentialism to legal decision-making. This was accomplished using five studies examining three domains: Studies 1-3 involve legal decision-making, Study 4 involves obesity, and Study 5 involves pedagogy.

2.1.   Studies 1-3: A Born Criminal – Genetic Essentialism and Criminality

Previous research examining how genetic essentialism affects legal decision-making has generally focused on two primary variables – perceived conscious control, and sentencing (Appelbaum & Scurich, 2013; Dar-Nimrod et al., 2011; Monterosso et al., 2005). These are important variables given their close relations with *mens rea*, such that less conscious control should lead to less perceived *mens rea*, thereby resulting in shorter prison sentences; however, the debate about the applicability of genetic evidence in the courtroom also involves additional aspects of the legal system as well, particularly the perception of criminal defenses. Given that genetic accounts of criminal behaviour should affect *mens rea*, then they should also impact the perceived applicability of various criminal defenses that either mitigate or fully negate one's *mens rea*. Indeed, in addition to outcomes such as verdicts and sentencing, scholars have pondered about whether or not genetic behavioural

explanations *should* affect the perceived applicability of defenses (Alper, 1998); however, little empirical work has examined whether or not they *actually* have this effect.

Another aspect of legal decision-making that has been largely absent in the literature is directly examining cognitive mechanisms behind such decisions. That is, a key step in understanding how genetic explanations affect outcome variables is to determine what social cognitive factors underlie one's decisions and behaviours. This point is important particularly because the literature on mental health stigma suggests that essentialist cognitions can have both ameliorative and deleterious effects based on simultaneously opposing beliefs. As previously discussed, people generally attribute less conscious control and personal responsibility to someone with clinical depression that is due to genes (Schomerus et al., 2014), which should alleviate blame and stigma (Corrigan, et al., 2000). On the other hand, people also expect a genetically-caused depression to be more persistent and stable, thereby exacerbating stigma (Phelan, 2005). This highlights the fact that genetic essentialism involves perceiving genetic effects as immutable and deterministic, which often elicits more positive and sympathetic perceptions of a target; but it also leads one to perceive the target as being more different, and the associated trait as being more stable, which often leads to more negative perceptions. This double-edged perception of genetic effects is a consistent theme that is evident in other domains as well, including legal decision-making. For instance, people ascribe less conscious control to a perpetrator with a genetic predisposition for criminal behaviour, thereby leading to a shorter prison sentence (Dar-Nimrod, Heine, Cheung, & Schaller, 2011); however, qualitative evidence suggests that a genetic predisposition also appears to be linked to greater perceived recidivism, although the researchers did not directly test for its impact on sentencing (Aspinwall et al., 2012). The latter point is reflective of the

field in general, because researchers have proposed various mediators that may impact

sentencing (e.g. Saks, Schweitzer, Aharoni, & Kiehl, 2014), although few have directly

tested them (e.g. Dar-Nimrod et al, 2011). Such mechanisms can be highly informative for

understanding how people come to make important legal decisions, which may generalize to

behaviours in other domains.

Study 1 investigated the impact of genetic behavioural explanations, relative to other

explanations, on various parts of the criminal justice system by examining variables rarely

considered in past research on genetic essentialist biases in legal decision-making, such as

preferred verdicts and perception of criminal defenses. Furthermore, mediation analyses

sought to uncover social cognitive mechanisms underlying sentencing decisions. Study 2 was

a direct replication of Study 1 using different samples, with an additional component on

causal attributions that will be discussed in Chapter 3.

Study 3 sought to understand how people think a candidate gene for a behaviour

works to affect one's behaviour. In other words, do people think that these genes work by

increasing one's impulse, or by decreasing one's impulse control? Such a distinction follows

from the conception of a dual process model (Kahneman, 2011), which argues that human

psychology consists of two systems: System 1 is associated with automatic processes and

unconscious reasoning, reflecting one's impulse; and System 2 is associated with more

controlled processes and conscious reasoning, reflecting one's impulse control. This may

have important implications because if the problem lies in having a greater impulse, then

there may be a possibility that the individual can be rehabilitated by providing support to

improve impulse control. If the problem lies in having poorer impulse control, however, then

rehabilitation may not be as successful. Thus, determining whether people assume that

genetic effects impact automatic versus controlled processes may allow for a better understanding of people's punitive motivations.

Because the three studies shared the two primary conditions in common (discussed below), the three datasets were combined into an aggregate dataset. In the interest of brevity, this dissertation will discuss the results of the aggregate dataset; but results for individual studies can also be seen in Appendices D and E for Studies 1 and 2, respectively. Results for Study 3 are reported in full. Most of the dependent variables were the same across all three studies, with some exceptions that will be noted. Moreover, this aggregate dataset provided an appropriate sample size to conduct a path analysis (Preacher & Coffman, 2006), directly examining the impact of the potential simultaneous double-edged nature of genetic attributions on one's punitive tendencies.

In sum, Studies 1 to 3 examined how genetic explanations might impact legal decisions, and what people think is the underlying mechanism behind how genes affect criminal behaviour.

2.1.1. Methods

2.1.1.1. Participants

Study 1 involved 132 participants (40% males; $M_{age} = 35.48$, $SD = 12.71$) from Amazon's Mechanical Turk (MTurk). The sample consisted of 76% Euro-Americans, 9% Asian Americans, 11% Others, and 4% unspecified. Furthermore, because political orientation is associated with legal decision-making (Appelbaum, Scurich, & Raad, 2015; Cochran, Boots, & Chamlin, 2006), participants also indicated this using a 5-point scale (1 =

Very liberal, 5 = Very conservative; $M = 2.59$, $SD = 0.97$). Participants received \$0.25 for participating in the study.

Study 2 involved 165 undergraduate students from the University of British Columbia (23% males; $M_{age} = 21.48$, $SD = 4.89$). The sample consisted of 38% Euro-Canadians, 33% East Asians, and 28% Others. Participants' political orientation was assessed using the same measure as in Study 1 ($M = 2.47$, $SD = 0.98$). Participants received one course credit for their participation.

Study 3 involved 303 participants from MTurk, with an effective sample size of 298 participants (42% males; $M_{age} = 34.62$, $SD = 11.26$) who passed our comprehension check questions by correctly responding to two out of three questions about the vignette that they had read. These questions were multiple-choice questions asking participants the following questions: "At what location did the incidence in question take place?", "What weapon did the defendant allegedly use to attack the victim?", and "According to the case file, what have researchers attributed the defendant's actions to?" The sample consisted of 68% Euro-Americans, 7% African Americans, 3% Asian Americans, and 22% Others. Participants' political orientation was assessed using the same measure as before ($M = 2.63$; $SD = 1.05$).

2.1.1.2. Materials

2.1.1.2.1. Experimental Manipulation

Participants first encountered the experimental manipulation. In Studies 1 and 2, the goal was to determine how a genetic behavioural explanation leads people to think differently. As such, there must be another type of explanation to serve as a basis of comparison. The counterpoint that was chosen was an environmental behavioural

explanation, reflecting more nurture-based explanations of criminal behaviour involving one's rearing environment (Delgado, 1985). To that end, the participants were randomly assigned to read one of three vignettes about a college student, Patrick, who fatally stabbed another person after an altercation. The three vignettes differed only in terms of the explanation given for his behavior: the Genetic vignette described Patrick's genetic predisposition for responding to provocations with violence ($n_1 = 47$, $n_2 = 57$, $n_3 = 76$); the Environmental vignette described Patrick's rearing environment as predisposing him to violence ($n_1 = 41$, $n_2 = 54$, $n_3 = 75$); and the Control vignette offered no explanation ($n_1 = 44$, $n_2 = 54$; see Appendix A). The Control condition should allow for the manifestation of the participants' default assumptions. Importantly, the magnitude of the purported effect of each explanation was identical; both causes were described as leading to a fourfold increase in the likelihood of Patrick responding to provocations with violence.

Study 3 focused on the genetic and environmental explanations of criminal behaviour, particularly whether people think genetic causes enhance one's criminal impulses, or they hinder one's ability to inhibit their criminal impulses. As such, Study 3 used the same Genetic and Environmental scenarios as in Studies 1 and 2, with the addition of two other scenarios, thereby creating four conditions. Both new conditions can be seen in Appendix B. The Gene-Impulse condition was the first newly-added condition, in which participants learned that Patrick's gene had created a stronger impulse to act violently compared to people without the gene ($n = 72$). The Gene-Control scenario was the second newly-added condition, in which participants learned that Patrick's gene had decreased his ability to control his violent impulses relative to people without the gene ($n = 76$).

2.1.1.2.2. Defense Claims

Participants then rated the applicability of three defense claims (Insanity; Diminished Capacity; Intoxication) for Patrick on a 7-point scale (0 = Not at all applicable; 6 = Perfectly applicable). Each defense claim was accompanied by corresponding definitions based on legal texts (see Appendix C; American Law Institute, 1962; Brody, Acker, & Logan, 2001; Padfield, 2008). Insanity and Diminished Capacity were included as they constitute a full and partial defense, respectively (Baum, 2013). That is, Insanity is a defense that fully negates one's *mens rea*, while Diminished Capacity only mitigates it. This allowed for the examination of people's perception of the applicability of different degrees of defense claims. Intoxication was included to ensure that participants would not simply deem any defense claim to be more or less applicable, given that intoxication was unrelated to information from the vignette. The Insanity and Diminished Capacity defenses should be deemed more applicable in the Genetic condition than the other conditions, but not for the Intoxication defense.

2.1.1.2.3. Verdicts

Participants made the same judgement regarding four verdicts (First degree murder; Second degree murder; Manslaughter; Not guilty) as they did with the defense claims discussed above, using the same 7-point scale. Each verdict was presented with an accompanying definition based on those that appear in the United States Code (see Appendix C; 18 U.S.C. § 1111-1112). These measures were not included in Study 3 due to non-significant results in Studies 1 and 2.

2.1.1.2.4. Sentencing

Participants read that Patrick had been convicted of manslaughter. In Studies 1 and 2, participants were to assign an appropriate prison sentence using an 11-point scale with 5-year increments (1 = 5 years, 11 = >50 years). Due to concerns about lack of sensitivity of this methodology, and to bring it more in line with other work, this measure was changed into an open-ended question in Study 3. Based on previous studies (e.g. Aspinwall et al., 2012; Dar-Nimrod et al., 2011), I expected that participants in the Genetic condition would assign a shorter prison sentence compared to participants in the other conditions.

2.1.1.2.5. Perceptions of the Perpetrator

Finally, participants used a 7-point scale to rate the degree to which Patrick: (a) had conscious control over his actions (0 = Not at all, 6 = Complete control); (b) intended to kill the victim (0 = Not at all, 6 = Full intention); (c) had knowledge that his actions would lead to the victim's death (0 = Not at all, 6 = Full knowledge); and (d) would reoffend if he were released back into the public (0 = Not at all likely, 6 = Completely likely)[3]. Participants also indicated how criminally responsible they felt that Patrick was on an 11-point scale (0 = Not at all criminally responsible, 10 = Completely criminally responsible), similar to questions used previously (Higgins, Heath, & Grannemann, 2007). Past research (e.g. Aspinwall et al., 2012) suggests that participants should perceive less criminal responsibility, less conscious control, less intention to kill, and greater recidivism in the Genetic condition compared to the other conditions. In contrast, the perpetrator's knowledge that his actions would kill the victim was not expected to differ across conditions as the kind of explanation should be

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[3] A mistake in copying the recidivism question from Study 1 to Study 2 was discovered after data collection had ended. As a result, this question could not be used for analysis. This question was corrected and reused in Study 3.

irrelevant to knowledge. Moreover, based on previous findings, particularly regarding the perceived double-edged nature of biological explanations of human behavior (e.g. Aspinwall et al., 2012; Dar-Nimrod et al., 2011), I also predicted that perceptions of conscious control and recidivism on the part of the perpetrator should mediate differences between groups in terms of prescribed prison sentences – in opposite directions.

2.1.2.   Results

Because Studies 1 to 3 intended to address two questions, the results will be discussed in two parts. First, analyses of the aggregate dataset are presented to address the impact of genetic explanations relative to environmental explanations of behaviour. As previously mentioned, in the interest of brevity, data for these two conditions across all three studies were combined into an aggregate study. Because the comparison between genetic and environmental explanations is the most informative in terms of allowing us to understand the impact of genetic explanations, only analyses involving these two conditions will be discussed in depth. Full analyses involving Studies 1 and 2 can be found in Appendices D and E. Second, analyses from Study 3 are discussed separately in full to address how people think genes influence one's behaviour.

All analyses involved multiple regressions. The conditions were dummy coded such that the Genetic group was the reference group (i.e. the Genetic group was coded as 0) against which the other groups were compared. Table 1 shows a summary of all effect sizes from the aggregate dataset. The primary predictor for the aggregate dataset was the Genetic-Environment contrast, while the primary predictors for Study 3 involved the contrasts between each of the four groups. All studies had participant age, sex (0 = male, 1 = female), and political orientation as covariates. All means reported here are adjusted means. Also, all

continuous variables (criterion variables and covariates) were standardized to generate standardized regression coefficients that can be interpreted on the same scale as Cohen's d's. All confidence intervals and mediation coefficients were determined through resampling based on 50,000 resamples (e.g. Kelley, 2005). The confidence intervals were resampled using the percentile bootstrap (see Biesanz, Falk, & Savalei, 2010). Except for places in which the significance is marginal, p-values are not reported.

2.1.2.1.   Aggregate Dataset[4]

2.1.2.1.1. Defense Claims

A graph depicting the perceived applicability of all defense claims from the aggregate dataset can be found in Figure 1. If genetic explanations do lead to reduced perceptions of *mens rea* on the part of a criminal, then defense claims that rely on arguing that the defendant had a general lack of *mens rea* should be preferred in the Genetic condition. Indeed, there was a main effect of Explanation on the insanity defense, being significantly higher in the Genetic condition ($M = 2.47$, $SE = 0.20$) than in the Environmental condition ($M = 1.26$, $SE = 0.22$), $\beta = -0.57$, $CI_{.95} = [-0.77, -0.36]$. This pattern also emerged with the diminished capacity defense, with this defense being endorsed more by participants in the Genetic condition ($M = 3.28$, $SE = 0.20$) than those in the Environmental condition ($M = 1.93$, $SE = 0.23$), $\beta = -0.62$, $CI_{.95} = [-0.78, -0.37]$. On the other hand, no such difference was found for the intoxication defense, $\beta = 0.04$, $CI_{.95} = [-0.14, 0.31]$. This latter finding is notable because it suggests that participants were not exhibiting a general tendency to be more lenient

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[4] Separate analyses for the aggregate dataset controlled for main effects of different studies and interactions between different studies and the condition contrasts. The results do not generally reach conventional levels of significance, and the findings do not change with the extra predictors (i.e. the main effects and interactions involving different studies) included. As a result, the analyses reported here exclude the extra predictors.

towards someone with a genetic predisposition for criminal activity. Given that the scenarios presented to the participants did not mention the use of any substances, there would have been little reason to deem this defense to be applicable. Participants were selective in their leniency, opting for defense claims that impinged on one's general ability to wilfully behave in contravention to the law.
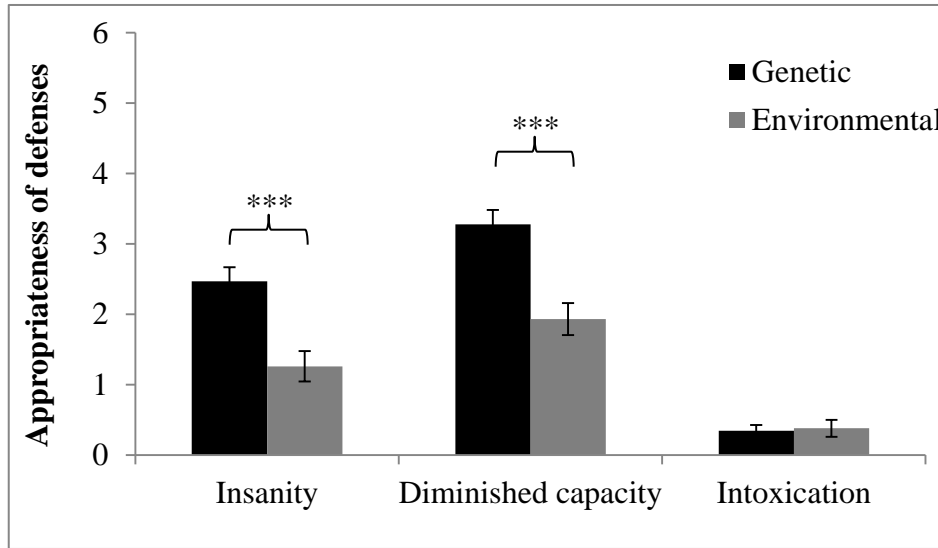
Figure 1. Adjusted means for perceived applicability for each defense claim, separated by Condition. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.1.2.1.2. Verdicts

By perceiving the defendant as having had less *mens rea*, and finding defense claims that mitigate *mens rea* to be more applicable, participants should also perceive the defendant as being less guilty if his behaviour was supposedly genetically caused. As such, participants in the Genetic condition should have preferred less severe verdicts more than those in the Environmental condition. Contrary to expectations, there were no main effects of Explanation on any of the verdicts, $|\beta|$'s < 0.25 (see Figure 2).

2.1.2.1.3. Sentencing

By perceiving less *mens rea* on the part of the defendant, the participants should also have felt that he was less deserving of punishment. Indeed, there was a marginal effect of Explanation on sentencing, such that participants in the Environmental condition prescribed marginally longer prison sentences ($M = 23.34$, $SE = 1.61$) than those in the Genetic condition ($M = 20.68$, $SE = 1.36$), $p = .097$, $\beta = 0.17$, $CI_{.95} = [-0.04, 0.38]$. Of note, though, is the fact that this was significant only in Study 3, but not in Studies 1 and 2. As discussed later, this may reflect the fact that the format of the question was different in Study 3 compared to Studies 1 and 2.
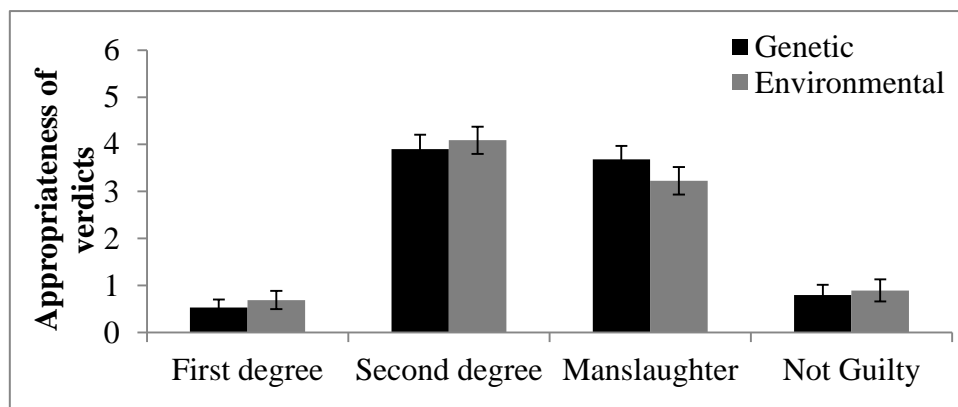
Figure 2. Adjusted means for perceived applicability for each verdict, separated by Condition. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$† $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.1.2.1.4. Perceptions of the Perpetrator

Participants' lowered perception of *mens rea* for the defendant should also be borne out in their explicit perceptions of conscious control over his behaviour and his intention to kill. On the other hand, different behavioural explanations should not impact the defendant's perceived knowledge that his actions would have killed the person as the explanations did not impinge upon his intelligence. In accordance with these expectations, Explanation affected perceived conscious control, being significantly higher in the Environmental condition ($M = $

5.00, $SE = 0.15$) than in the Genetic condition ($M = 4.10$, $SE = 0.12$), $\beta = 0.63$, $CI_{.95} = [0.40,$ 0.79]. The results were similar regarding intention to kill, with participants in the Environmental condition perceiving more intention ($M = 3.77$, $SE = 0.18$) than those in the Genetic condition ($M = 3.24$, $SE = 0.16$), $\beta = 0.30$, $CI_{.95} = [0.09, 0.51]$. No such differences were found when predicting how much Patrick knew that his actions would have killed the victim, $\beta = 0.15$, $CI_{.95} = [-0.13, 0.44]$. These results can be seen in Figure 3.

As a result of perceiving less conscious control and intention into Patrick's behaviour if he had a genetic cause, he should also be seen as being less criminally responsible for his crime. Furthermore, as the judges in Aspinwall et al.'s study argued (2012), having a genetic explanation should also lead to the expectation that the behaviour would persist. The first expectation was not borne out. Despite Patrick being seen as having had both less control over his criminal behaviour and less intention to engage in that behaviour on account of his genetic cause, he was not perceived to be any less criminally responsible than if he had an environmental cause. Specifically, perceived criminal responsibility did not differ between conditions, $\beta = 0.09$, $CI_{.95} = [-0.07, 0.34]$. On the other hand, in accordance with expectations, participants in the Genetic condition expected greater recidivism ($M = 4.57$, $SE = 0.13$) compared to those in the Environmental condition ($M = 4.15$, $SE = 0.16$), $d = -0.31$, $CI_{.95} = [-0.53, -0.02]$ (see Figure 3).
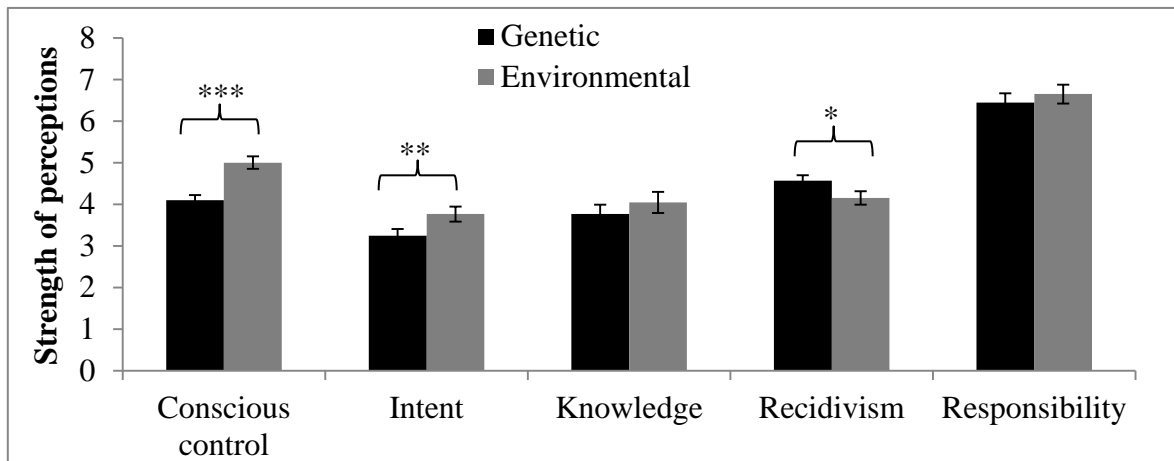
Figure 3. Adjusted means for various perceptions of the perpetrator, separated by Condition. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

Dependent variables	Overall β	95% C.I.
Insanity defense	-0.57***	[-0.77, -0.36]
Diminished capacity defense	-0.62***	[-0.78, -0.37]
Intoxication defense	0.04	[-0.14, 0.31]
First degree murder	0.12	[-0.15, 0.41]
Second degree murder	0.09	[-0.18, 0.36]
Manslaughter	-0.22	[-0.50, 0.06]
Not guilty	0.06	[-0.22, 0.34]
Sentencing	0.17†	[-0.04, 0.38]
Criminal responsibility	0.09	[-0.07, 0.34]
Conscious control	0.63***	[0.40, 0.79]
Intent to kill	0.30**	[0.09, 0.51]
Knowledge of consequences	0.15	[-0.13, 0.44]
Recidivism	-0.31*	[-0.53, -0.02]

Table 1. Summary of all effect sizes and 95% confidence intervals from the aggregate dataset comparing the Genetic and Environmental conditions. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.1.2.1.5. Mediation Analyses

One important purpose behind Studies 1 to 3 was to uncover social cognitive mechanisms that mediate how exposure to genetic behavioural explanations affect people's sentencing tendencies. In particular, previous work has suggested that it is important to consider the potentially double-edged nature of genetic explanations of criminal behaviour in impacting punitive motivations. That is, genetic explanations should affect punitive motivations in opposite ways by leading to both aggravating and mitigating perceptions of the criminal. On the one hand, genetic explanations should lead to less severe punishments due to lower perceptions of conscious behavioural control. On the other hand, such explanations should lead to more severe punishments due to increased concerns about recidivism. To that end, two mediation analyses sought to examine the underlying role of conscious control and expected recidivism on punitive tendencies. Despite the main effect of Explanation on sentencing being only marginally significant, the lack of significance may mask informative mechanisms for theory building and confirmation (see Rucker, Preacher, Tormala, & Petty, 2011; Zhao, Lynch, & Chen, 2010). This is especially pertinent when there are *a priori* expectations of underlying mechanisms, and those mechanisms are expected to be in opposite directions, which is the case based on previous research (e.g. Aspinwall et al., 2012; Dar-Nimrod et al., 2011). Furthermore, the traditional Baron and Kenny (1986) requirement for a significant direct effect to test for subsequent mediations leads to an underpowered approach for testing mediation (Edwards & Lambert, 2007; MacKinnon, Fairchild, & Fritz, 2007); therefore, the indirect effects served as the focus for determining mediation.

Across all three studies, the Environmental condition was associated with higher ascriptions of conscious control compared to the Genetic condition, $\beta = 0.63$, $CI_{.95} = [0.40, 0.79]$, which predicted lengthier sentences, $\beta = 0.29$, $CI_{.95} = [0.17, 0.41]$. The indirect effect was significant, $\beta = 0.18$, $CI_{.95} = [0.09, 0.29]$. Compared to the marginal non-mediated direct effect, $\beta = 0.17$, $CI_{.95} = [-0.04, 0.38]$, the mediated direct effect decreased significantly, $\beta = -0.02$, $CI_{.95} = [-0.24, 0.18]$ (see Figure 4a).

Another mediation analysis revealed that perceived recidivism is also important in explaining how the Genetic condition may affect prison sentences differently than the Environmental condition. Specifically, the Genetic condition elicited greater perceived recidivism than the Environmental condition, $\beta = -0.31$, $CI_{.95} = [-0.53, -0.02]$. This, in turn, predicted lengthier sentences overall, $\beta = 0.38$, $CI_{.95} = [0.26, 0.50]$. The indirect effect of recidivism was significant, $\beta = -0.12$, $CI_{.95} = [-0.23, -0.02]$. This analysis revealed a reliable suppression effect, with the mediated direct effect, $\beta = 0.32$, $CI_{.95} = [0.05, 0.54]$, being stronger than the non-mediated direct effect, $\beta = 0.17$, as reported above (see Figure 4b)[5].
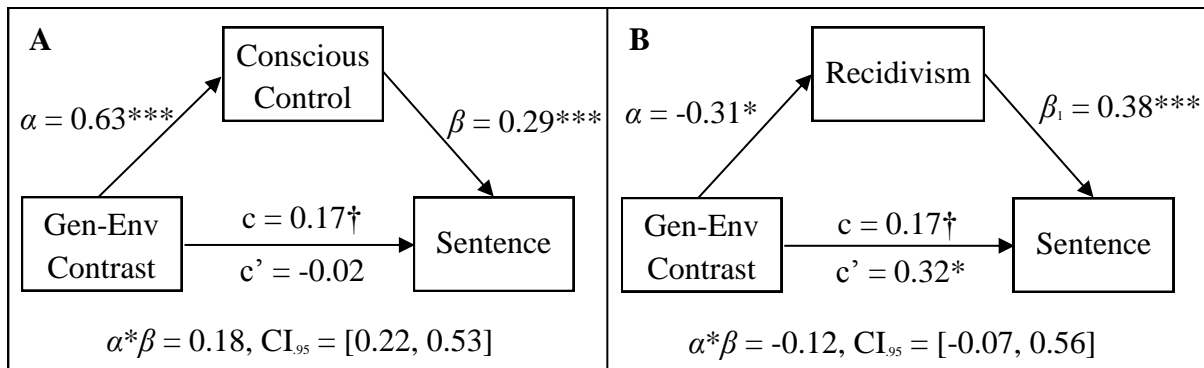
Figure 4. Mediation diagrams predicting differences in prison sentence between Genetic and Environmental conditions from the aggregate study. Gen-Env denotes the contrast between Genetic and Environmental conditions. All effect sizes are standardized. Panel A refers to mediation through conscious control, and Panel B refers to mediation through expected recidivism. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

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[5] The same mediation analyses were also performed on the verdicts; but since no significant results emerged for any of the studies regarding the verdict variables in this dissertation, these results are not discussed further.

To better model how genetic versus environmental explanations affect different levels of punishment, a path analysis examined the simultaneous contributions of both the mitigating and aggravating effects of genetic perceptions using the "lavaan" package in R (Rosseel, 2012). The path analysis model predicted prison sentence length using the explanations that participants read (0 = Genetic, 1 = Environmental), mediated through both perceived conscious control and expected recidivism. Covariates included participant age, sex (0 = male, 1 = female), and political orientation, as with all previous regression models. This analysis yielded a model with good fit, CFI = 1.00, RMSEA = 0.03, SRMR = 0.01 (see Figure 5). The correlation matrix for all predictors (including covariates) can be found in Table 2. In particular, the environmental explanation led to greater ascriptions of conscious control compared to the genetic explanation, $\beta = 0.63$, $p < .001$, which also predicted lengthier prison sentences, $\beta = 0.31$, $p < .001$. At the same time, the environmental condition led to weaker expectations of recidivism, $\beta = -0.27$, $p = .003$, while greater expectations of recidivism predicted longer prison sentences, $\beta = 0.44$, $p < .001$. After accounting for all of these covariates and mediators, the different explanations no longer accounted for differences in prison sentence length, $\beta = 0.10$, $p = .358$.

	1	2	3	4	5	6
1. Condition contrast	1.00					
2. Internal attributions	-0.34***	1.00				
3. Conscious control	0.37***	-0.00	1.00			
4. Recidivism	-0.13*	0.16**	0.03	1.00		
5. Sentence	0.17**	0.20***	0.33***	0.39***	1.00	
8. Political orientation	0.08	0.10 ·	0.19***	0.14**	0.27***	1.00

Table 2. Correlation matrix for all variables entered into the path analysis in the aggregate study. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$
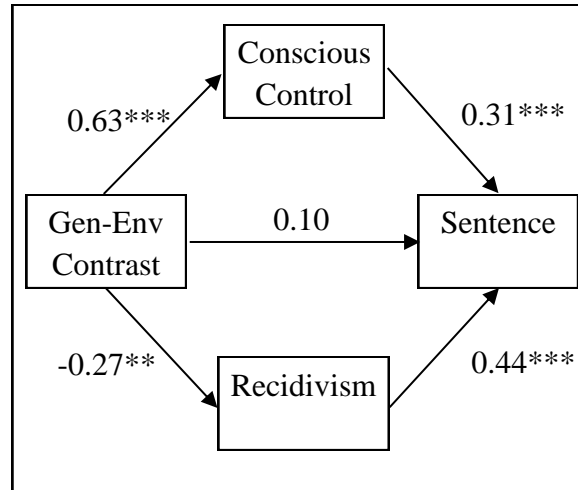
Figure 5. Path diagram examining control and recidivism as simultaneously countervailing pathways in predicting sentencing. All effect sizes are standardized.
† *p* < .10 * *p* < .05, ** *p* < .01, * *p* < .001**

2.1.2.1.6. Discussion of Aggregate Dataset

Across Studies 1 to 3 there was consistent evidence that exposure to genetic explanations of criminal behaviour impacted various aspects of legal decision-making – such explanations led to higher perceived applicability of both the insanity and diminished capacity defenses, both of which either mitigate or fully negate *mens rea*. This effect is not generalizable to a general tendency to be amenable towards all defense claims given that the intoxication defense was not seen as more applicable as a result of genetic explanations. This is the first demonstration of a genetic ascription's effect on people's endorsement of various mitigating accounts of a crime. This suggests that people are more willing to view genes as being responsible for one's actions by liberating them from their own responsibility.

Consistent with previous research, genetic explanations affect people's perception of another's mental state, leading them to ascribe less behavioural control and intention to do harm on the part of the perpetrator than environmental explanations (Dar-Nimrod et al., 2011). Furthermore, people also expected that the perpetrator would be more likely to

reoffend in the future if there was a genetic explanation for his crime compared to an environmental explanation. In contrast, as predicted, perceptions of the perpetrator's knowledge of the consequences of his actions did not differ, as the genetic explanation affected neither the perpetrator's intelligence nor knowledge.

Counter to expectations, though, explanations of criminal behavior did not affect either perceived criminal responsibility or perceived appropriateness of various verdicts, despite affecting perceived control and intention. While lower ascriptions in these domains should have led to more lenient sentences and preferences for less serious verdicts, these expectations were not supported by the data. Most notably, there was only a marginally significant effect of behavioural explanations on the length of prescribed prison sentences. Moreover, the marginal effect was primarily driven by the significant effect of Study 3, while Studies 1 and 2 did not yield significant differences. One main difference between these studies that may explain this discrepancy is that a change in methodology regarding how participants recommended a prison sentence yielded a significant difference between the Genetic and Environmental conditions in prescribed prison sentence length, more in line with past research (e.g. Aspinwall et al., 2012; Dar-Nimrod et al., 2011). In Studies 1 and 2, the recommended prison sentence was solicited using a closed-ended question that restricted responses to 5-year intervals. In contrast, Study 3 provided participants with more freedom to respond by using an open-ended question to solicit recommended prison sentences. This suggests two important implications. First, given the small real difference in prison sentences across conditions in Aspinwall et al. (2012) and the 5-year increment used in the previous designs of the current study, the raw effect of genetic explanations may only lead to a reduction in prison sentence of fewer than five years when compared to environmental

explanations. Second, as a consequence of the first point, it stresses the importance of methodological concerns when examining sentencing practises – this variable may need high sensitivity in order for a reliable difference to be detected. Alternatively, it may be that the countervailing forces demonstrated in the mediation analyses do not consistently yield main effects despite the presence of real underlying effects

To uncover potential mechanisms that underlie some of our findings, specifically regarding sentencing, subsequent mediation and path analyses revealed that there were indirect effects of behavioral explanations on sentencing through perceived conscious control and perceived recidivism, but in opposite directions. Specifically, genetic ascriptions led to lower perceptions of perceived conscious control of one's behavior, predicting shorter prison sentences. On the other hand, genetic ascriptions led to significantly higher perceived recidivism, leading to lengthier prison sentences. These two mediators worked in opposition to each other, providing preliminary empirical evidence for the double-edged perceptions given by judges in Aspinwall et al.'s study (2012).

2.1.2.2.   Study 3

In order to better understand the impact of genetic behavioural explanations, it is also important to determine the mechanism by which people think genetics affect behaviour. That is, do people think of genes as affecting behaviour by increasing one's impulse to act, or by impinging on one's ability to inhibit a normal impulse to act? Study 3 addresses this question by including two scenarios that were described earlier: one that describes Patrick's gene as amplifying his impulse (Gene-Impulse condition), and one that describes his gene as inhibiting his impulse control (Gene-Control condition). The following results show how the

various genetic conditions differ from each other, and how the Environmental condition differs from each of the genetic conditions.[6]

2.1.2.2.1. Defense Claims

Graphs for the defense claims can be seen in Figure 6. Overall, the various genetic explanations did not differ from each other in terms of the acceptance of the insanity and diminished capacity defenses, but they were collectively different from the environmental explanation. Specifically, there was a main effect of Explanation on the applicability of the insanity defense, such that it was endorsed less in the Environmental condition ($M = 0.83$, $SE = 0.31$) than in the Genetic condition ($M = 1.76$, $SE = 0.24$), $\beta = -0.47$, $CI_{.95} = [-0.78, -0.17]$, the Gene-Impulse condition ($M = 1.70$, $SE = 0.32$), $\beta = 0.44$, $CI_{.95} = [0.15, 0.74]$, and the Gene-Control condition ($M = 1.90$, $SE = 0.33$), $\beta = 0.54$, $CI_{.95} = [0.23, 0.85]$. The latter three conditions did not differ significantly from each other, $|\beta|$'s < 0.10.

The same pattern emerged with the perceived applicability of the diminished capacity defense, wherein participants preferred it less in the Environmental condition ($M = 2.01$, $SE = 0.33$) compared to those in the Genetic condition ($M = 3.59$, $SE = 0.24$), $\beta = -0.77$, $CI_{.95} = [-1.08, -0.46]$, those in the Gene-Impulse condition ($M = 3.73$, $SE = 0.31$), $\beta = 0.84$, $CI_{.95} = [0.51, 1.16]$, and those in the Gene-Control condition ($M = 3.56$, $SE = 0.30$), $\beta = 0.75$, $CI_{.95} = [0.44, 1.06]$. The latter three conditions did not differ significantly from each other, $|\beta|$'s < 0.10.

None of the conditions differed significantly from each other in terms of the perceived applicability of the intoxication defense, $|\beta|$'s < 0.10.

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[6] The Genetic-Environment contrasts in this section were part of the aggregate study results presented above. They were presented here for the sake of completeness.
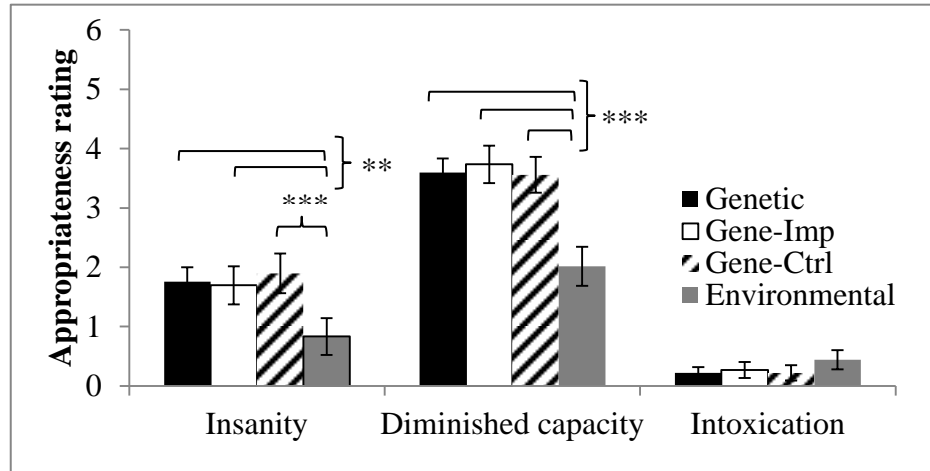
Figure 6. Perceived appropriateness of defense claims by condition. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.1.2.2.2. Sentencing

Reflecting the greater acceptance of insanity and diminished capacity defenses as a result of genetic explanations relative to environmental explanations of criminal behaviour, there was a main effect of Explanation on sentencing. Specifically, the prescribed prison sentence length was significantly longer in the Environmental condition ($M = 26.34$, $SE = 2.43$) compared to the Genetic condition ($M = 21.58$, $SE = 1.81$), $\beta = 0.30$, $CI_{.95} = [0.00, 0.62]$, the Gene-Impulse condition ($M = 15.58$, $SE = 2.45$), $\beta = -0.69$, $CI_{.95} = [-1.00, -0.38]$, and the Gene-Control condition ($M = 17.56$, $SE = 2.44$), $\beta = -0.56$, $CI_{.95} = [-0.87, -0.25]$. The only other significantly contrast was between the Genetic and Gene-Impulse conditions, with the sentence length being longer in the Genetic condition than in the Gene-Impulse condition, $\beta = -0.38$, $CI_{.95} = [-0.68, -0.07]$. The remaining contrasts were not significant, $|\beta|$'s < 0.30. These results can be seen in Figure 7.
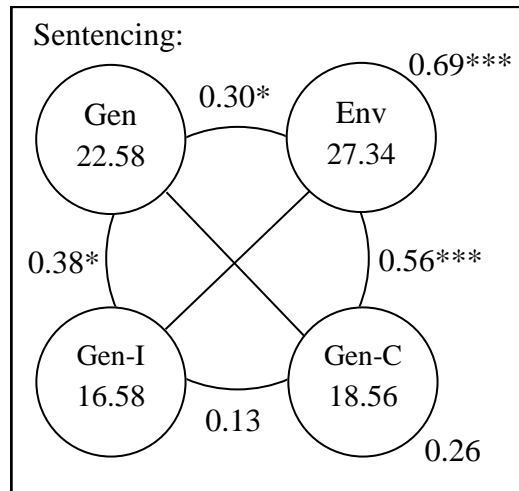
Figure 7. Adjusted means and absolute standardized effect sizes comparing sentencing between conditions. Effect sizes in the corners relate to diagonal contrasts involving the nearest and farthest conditions. Gen = Genetic, Env = Environmental, Gen-I = Gene-Impulse, Gen-C = Gene-Control. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.1.2.2.3. Perceptions of the Perpetrator

Perceptions of the perpetrator resembled results from previous studies, such that the environmental explanation led to less perceived conscious control compared to the three genetic conditions, and these three conditions were not significantly different from each other. In particular, participants in the Environmental condition ($M = 5.13$, $SE = 0.22$) perceived Patrick as having more conscious control than those in the Genetic condition ($M = 4.08$, $SE = 0.18$), $\beta = 0.74$, $CI_{.95} = [0.44, 1.04]$, those in the Gene-Impulse condition ($M = 4.13$, $SE = 0.23$), $\beta = -0.70$, $CI_{.95} = [-0.99, -0.40]$, and those in the Gene-Control condition ($M = 4.10$, $SE = 0.24$), $\beta = -0.72$, $CI_{.95} = [-1.02, -0.43]$. The latter three conditions did not differ significantly from each other, $|\beta|$'s < 0.05 (see Figure 8).
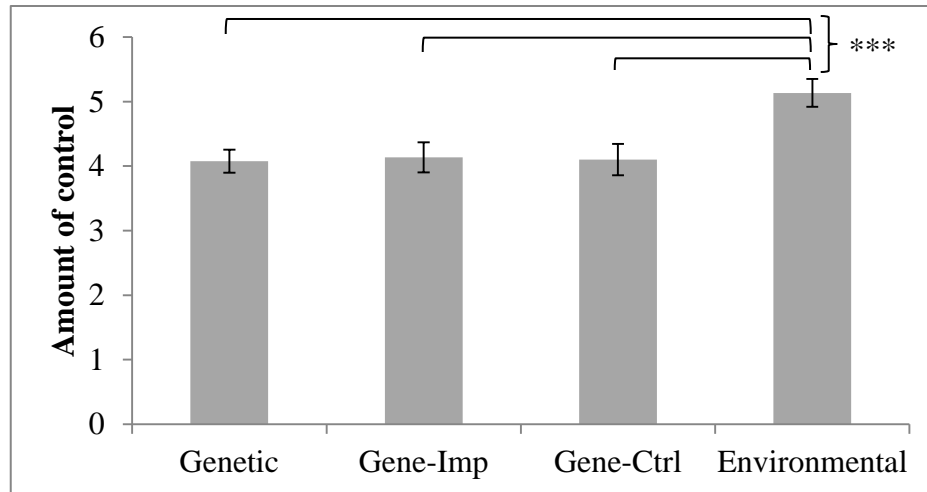
Figure 8. Perceived of behavioural control, by condition. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

Despite the main effect of Explanation on perceived conscious control, this was not the case with intention to kill, $|\beta|$'s < 0.05; however, there was an effect of Explanation on perceived recidivism. In particular, participants in the Genetic condition saw Patrick as being marginally more likely to reoffend ($M = 4.81$, $SE = 0.13$) than participants in the Environmental condition ($M = 4.47$, $SE = 0.19$), $p = .095$, $\beta = -0.26$, $CI_{.95} = [-0.56, 0.01]$, and participants in the Gene-Impulse condition ($M = 4.47$, $SE = 0.20$), $p = 098$, $\beta = -0.26$, $CI_{.95} = [-0.60, 0.05]$. The other contrasts were not significant, $|\beta|$'s < 0.05. This suggests that participants may be more likely to assume that genes impact one's ability to inhibit one's behavioural control rather than assuming that genes impact one's impulse.

Reflecting participants' greater acceptance of insanity and diminished capacity defense claims, and their lower perceptions of conscious control due to the genetic behavioural explanation for Patrick, it also led to lower perceptions of criminal responsibility. In particular, participants in the Environmental condition ($M = 5.30$, $SE = 0.18$) saw Patrick as being more criminally responsible than those in the Genetic condition ($M = 4.80$, $SE = 0.15$), $\beta = 0.40$, $CI_{.95} = [0.11, 0.68]$, those in the Gene-Impulse condition ($M = 4.70$, $SE =$

51

0.20), $\beta = -0.49$, CI$_{.95}$ = [-0.81, -0.18], and those in the Gene-Control conditions ($M = 4.72$,

$SE = 0.20$), $\beta = -0.47$, CI$_{.95}$ = [-0.78, -0.18]. The latter three conditions did not differ

significantly from each other, $|\beta|$'s < 0.10 (see Figure 9).
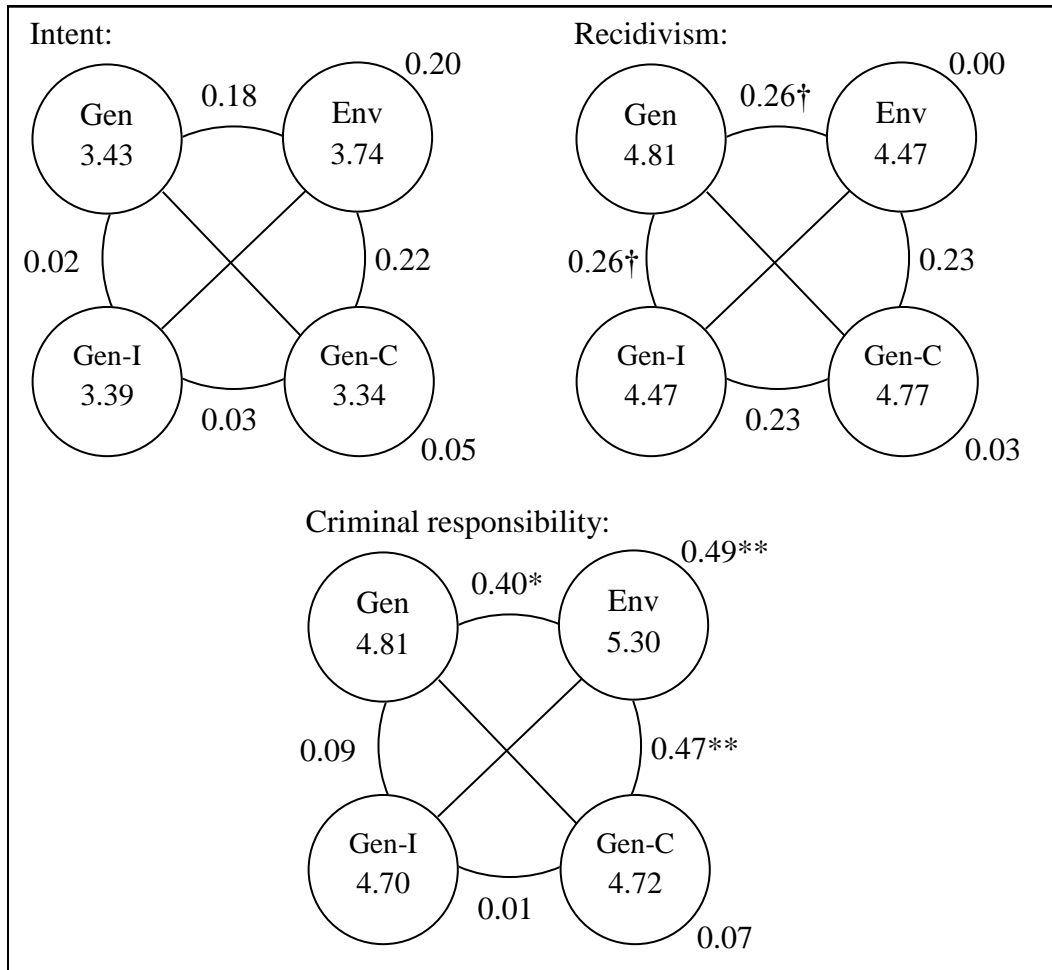
Figure 9. Adjusted means and absolute standardized effect sizes for comparing various perceptions of the perpetrator between conditions. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.1.2.2.4. Discussion of Study 3

It is notable that the Gene-Impulse and Gene-Control conditions do not generally

yield differences from the Genetic condition. Across most of the dependent variables

examined in Study 3, these three gene-based conditions did not differ significantly from each

other. It is even more curious that participants in all three gene-based conditions perceived

Patrick as having the same level of decreased conscious control, even though one of the conditions is about a stronger impulse. The one exception to this was when the original genetic explanation led to greater expected recidivism compared to an impulse-based genetic explanation. This may suggest that people consider genetic explanations to generally be similar to an impulse control-based genetic explanation, and different from an impulse-based explanation. In other words, people may assume that genes act by inhibiting one's impulse control rather than increasing the strength of one's impulse. The robustness of this finding has yet to be determined given that it only emerged in one dependent variable. Aside from this exception, the three gene-based explanations were all similar to each other. This similarity suggests several possibilities. One possibility is that when people encounter genetic behavioural explanations, they may not consider what mechanisms link genotypes to phenotypes, instead defaulting to a simplistic "cause-and-effect" schematic of genetic effects on behaviour. Another possibility is that people consider both of these mechanisms as possibilities by default, rendering the three genetic conditions mostly equivalent. The current study was not able to resolve this conundrum. Future research is needed that can discriminate between these two possibilities.

2.1.2.3. Discussion

Studies 1 to 3 all examined whether genetic explanations of criminal behaviour affect legal decision-making differently than environmental explanations of criminal behaviour, resulting in findings that carry important implications.

One particularly important implication of these studies pertains to *mens rea*, a legal concept relating to one's malicious intent and volition to commit a crime, and is necessary for a criminal conviction (American Law Institute, 1962). Perceiving someone's actions as

being beyond their control likely leads to the perception that the perpetrator lacked *mens rea* during the commission of the crime. Indeed, many defense claims, some of which we adopted for our study, are meant to either mitigate or negate *mens rea* by arguing for lack of intention and/or behavioural control. In line with expectations laid out by the genetic essentialism framework (Dar-Nimrod & Heine, 2011), these studies support the idea that genetic explanations more robustly impact *mens rea*-related attributions differently than other kinds of explanations. It is important to keep in mind that the means for participants' acceptance of defenses were generally below the midpoints of the scales, suggesting that participants did not perceive them to be *highly* applicable; however, the pattern of differences across conditions suggests that behavioural explanations do affect perceptions of criminal defenses. That is, across all three studies, genetic explanations reliably increased the perceived applicability of both the insanity and diminished capacity defenses compared to the environmental explanations involving one's rearing environment. Related to these findings, compared to environmental explanations, genetic explanations overall lowered one's perceptions of the perpetrator's conscious control over his actions as well as his perceived intention to harm the victim; but they do not impact perceptions of whether the perpetrator knew the outcome of his actions. Altogether, these results suggest that genetic explanations diminish one's agency – despite knowing that his actions could have killed the victim, neither was he able to control his behaviour, nor did he really intend to kill the victim. Both of these beliefs may serve to partially mitigate a person's perceived guilt. Unfortunately, Study 3 could not determine whether people understand genetic causes as impacting one's impulses or one's ability to inhibit their impulses. Additional research is needed to better understand how people actually conceptualize genotype-phenotype pathways.

Despite the impact of genetic explanations on general perceptions of *mens rea*, they do not appear to explicitly affect ultimate verdicts, nor do they reliably affect perceptions of criminal responsibility, compared to environmental explanations. Across the 3 studies, only Study 3 revealed lower levels of criminal responsibility in response to a genetic explanation compared to either an environmental explanation. Furthermore, participants' perceptions of final verdicts were not affected across the three studies, suggesting that genetic explanations may not affect ultimate legal judgments. Perhaps participants felt that there was insufficient information to arrive at an ultimate judgment, leading all participants to settle on a similar rating with which they were comfortable. Alternatively, there may be additional factors that play a role in the application of certain verdicts, and controlling for such factors may yet allow behavioural explanations to have an effect. Future work can examine these alternative interpretations and determine what additional factors should be considered.

The sentencing question sidestepped the issue of potential uncertainties by the participants with regard to applying different verdicts because they were provided with a verdict, and then they were asked to provide an appropriate prison sentence. While Studies 1 and 2 did not yield mean differences resulting from genetic and environmental explanations, the aggregate dataset shows an overall marginally significant effect. Furthermore, mediation and path analyses showed that this lack of a clear significant effect may have been due to different forces acting against each other. First, genetic, versus environmental, explanations led to lower perceptions of conscious behavioural control, which predicted lighter prison sentences. On the other hand, genetic explanations triggered greater concerns about recidivism than environmental explanations, subsequently predicting lengthier prison sentences. This potentially maps onto distinctions between retributive versus rehabilitative

punishment due to one's implicit theories of behaviour (Plaks, Levy, & Dweck, 2009). Thus, similar to what has been found in other domains such as mental health stigma (e.g. Phelan, 2005; Schomerus et al., 2014), genetic evidence may force people to concurrently consider and reconcile this opposing combination of mitigating and aggravating points.

2.2.   Study 4: Naturally Fast Metabolism – Genetic Essentialism and Body Weight

Aside from legal decision-making, another domain in which genetic causes have been heavily discussed is obesity, particularly given recent discoveries of genetic links and metabolic mechanisms underlying some individual differences in obesity (e.g. Claussnitzer, et al., 2015; Lim, et al., 2015). Media reports have heralded them as breakthroughs, but such reports have revealed people's genetic essentialist interpretations of the genetic links, with some headlines demonstrating an oversimplification of genetic effects. For example, some headlines suggested, "No need for diets and exercise? Scientists find obesity gene with off switch!" (RT News, 2015), and others said "Obesity gene can be switched off? New study may lead to better treatments" (Gutierrez, 2015). Even the National Institutes of Health Director's Blog discussed it as "Flipping a genetic switch on obesity?" (Collins, 2015). The impact of these cultural messages and their underlying genetic essentialist biases is important to understand because biological attributions of obesity are very common. For instance, a little over half of the teachers and school healthcare providers sampled in one Minnesotan school district attributed obesity to biological factors while less than half attributed it to lacking exercise (Neumark-Sztainer, Story, & Harris, 1999), with similar results from a large study of the general public in the United Kingdom (Ogden & Flanagan, 2008). This stands in contrast to medical opinions about the importance of genetics in obesity, with general

practitioners being only about half as likely as the general public to endorse biological factors as a cause for obesity, while greatly endorsing behavioural (e.g. inactivity) and structural (e.g. driving culture) factors (Foster, et al., 2003; Ogden & Flanagan, 2008). Furthermore, the aforementioned genetic causal factor for obesity described by Classnitzer et al. (2015) is found in 44% of European populations and only in 5% of African populations, and accounts only for an approximate increase of 1 kg for an adult between 160 to 180 cm in height (Dar-Nimrod et al., 2014; Speliotes, et al., 2010). Evidently, any given "obesity gene" is responsible for only a small amount of individual variability in body weight, despite the widespread belief that genes play an important role in obesity.

Understanding the nature of people's beliefs about obesity is important because people's conceptualization of obesity significantly predicts the types of social policies they endorse (Barry, Brescoll, Brownell, & Schlesinger, 2009). For example, perceiving obesity as an eating disorder or an addiction that is uncontrollable makes people more amenable to supportive policies such as requiring warning labels for food with high sugar or fat content; but this view of obesity also makes people less amenable to punitive policies such as the imposition of junk food taxes. Similarly, general practitioners who endorse genetics as an important causal factor for obesity are also more willing to endorse medical treatments for obesity (Ogden & Flanagan, 2008), suggesting that they believe that lifestyle changes are less effective at treating obesity if it had biological causes. Given such important implications, Study 4 attempted to unpack the consequences of attributing obesity to having a genetic etiology.

2.2.1.   Methods

2.2.1.1.   Participants

Study 4 involved 143 participants (25% males; $M_{age} = 20.50$, $SD = 3.88$) from the

University of British Columbia. The sample consisted of 34% Euro-Americans, 55% Asians,

7% Others, and 4% unspecified. Participants also provided their own height and body weight,

based on which a body mass index (BMI) was calculated. Given that one's own BMI has

been shown to be associated with one's obesity-related beliefs (Bleich, Bennett, Gudzune, &

Cooper, 2012), this was included as a covariate along with participants' age and sex.

2.2.1.2.   Materials

2.2.1.2.1.   Experimental Manipulation

Participants first encountered the experimental manipulation. Participants were

randomly assigned to read one of the three vignettes portraying Jeremy as a chef-in-training

who learns about the importance of metabolism given its links to obesity (Astrup, et al.,

1999), and discovers that he has high metabolism based on what he learns in this class (see

Appendix F). Similar to Studies 1-3, Study 4 contained a Genetic condition ($n = 30$), a

Control condition ($n = 30$), and another condition that served as a basis of comparison for the

Genetic condition. In this case, the comparison condition is the Breastfed condition ($n = 34$).

In the Genetic condition, Jeremy's faster metabolism was attributed to a genetic marker

based on obesity research (Haldar, et al., 2012). In the Breastfed condition, Jeremy's faster

metabolism was attributed to being breastfed rather than bottle-fed based on research by

Armstrong and Reilly (2002). The Control condition attributed Jeremy's faster metabolism to

general individual differences. In this study, participants were told that faster metabolism was

associated with being more capable of burning fat, turning it into energy.

2.2.1.2.2. Obesity-Related Beliefs

Participants then responded to a series of questions that examine their beliefs related to obesity. One question relates to their perceived control that Jeremy has over his weight on a 5-point scale (0 = No control at all, 4 = Complete control). Participants also indicated how likely they felt that Jeremy had a faster metabolism than the average person (0 = Not at all likely, 4 = Completely likely), and how easily Jeremy can burn calories from fat (-2 = Much harder than the average person, +2 = Much easier than the average person). Finally, an open-ended question asked participants to predict how much change in weight Jeremy would experience over the next five years, after having been told that people generally gain an average of one pound per year.

2.2.2.   Results

Multiple regression analyses similar to those used in Studies 1-3 were used to analyse data for Study 4, using participant sex (0 = male, 1 = female) and age as covariates. As mentioned above, analyses for this study also included participant BMI as a covariate. The Genetic condition served as the reference group against which the Breastfed and Control conditions were compared. Parallel regression models with the Breastfed condition as the reference group also allowed for the analysis of Breastfed-Control contrasts. Again, all continuous variables were standardised (i.e. age, BMI, and all criterion variables), yielding standardized regression coefficients that can be interpreted in the same way as Cohen's d's.

Participants in the Genetic group perceived Jeremy as having marginally less control over his weight ($M = 2.82$, $SE = 0.14$) compared to participants in the Breastfed condition ($M = 3.09$, $SE = 0.13$), $p = .061$, $\beta = 0.40$, $CI_{.95} = [-0.02, 0.80]$, but not to those in the Control

condition ($M = 2.84$, $SE = 0.13$), $\beta = 0.02$, $CI_{.95} = [-0.39, 0.42]$. The latter two conditions

differed marginally from each other, $p = 0.080$, $\beta = -0.38$, $CI_{.95} = [-0.79, 0.06]$ (see Figure 10).
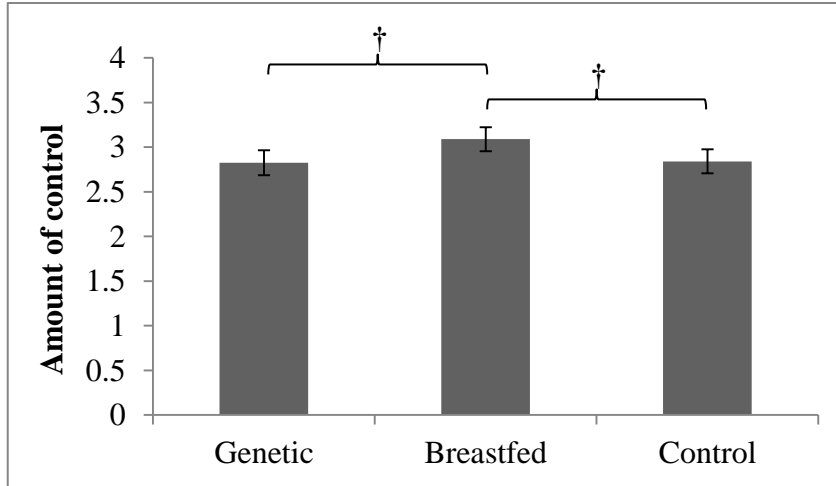
Figure 10. Perceived control over Jeremy's weight, by condition. Error bars
represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

Participants in the Genetic explanation felt that Jeremy had a higher likelihood of

having faster metabolism ($M = 2.86$, $SE = 0.14$), than was perceived by participants in the

Breastfed explanation ($M = 2.56$, $SE = 0.13$), despite them both being empirically supported,

$\beta = -0.43$, $CI_{.95} = [-0.79, -0.05]$. Conversely, participants did not differ between the Genetic

condition and the Control condition ($M = 2.74$, $SE = 0.13$), $\beta = -0.16$, $CI_{.95} = [-0.55, 0.21]$.

The Breastfed and Control conditions did not differ significantly, $\beta = 0.26$, $CI_{.95} = [-0.09,$

$0.61]$ (see Figure 11).

In line with other findings in this study, participants in the Genetic condition felt that

Jeremy had an easier time burning calories relative to the average person ($M = 1.21$, $SE =$

0.17) when compared to participants in the Breastfed condition ($M = 0.75$, $SE = 0.14$), $\beta = -$

0.64, $CI_{.95} = [-1.00, -0.25]$. The Genetic explanation also made burning calories easier than

simply attributing it to individual differences in the Control condition ($M = 0.77$, $SE = 0.15$),

$\beta = -0.61$, $CI_{.95} = [-1.00, -0.22]$. The Breastfed and Control conditions did not differ from each other significantly, $\beta = 0.03$, $CI_{.95} = [-0.33, 0.37]$ (see Figure 11).
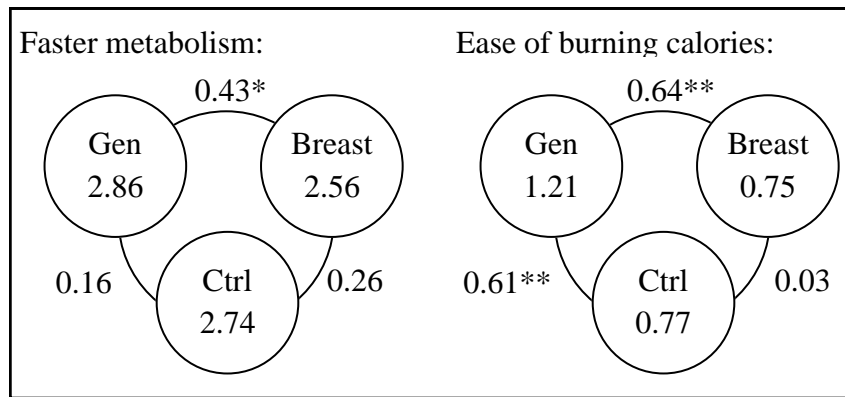
Figure 11. Adjusted means and absolute standardized effect sizes comparing between various perceptions of Jeremy between conditions. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

These perceptions appeared to have led participants to perceive that Jeremy would gain marginally less weight in the Genetic condition ($M = 3.63$, $SE = 0.85$) compared to the Breastfed condition ($M = 5.55$, $SE = 1.16$), $p = .093$, $\beta = 0.35$, $CI_{.95} = [-0.05, 0.80]$, but not compared to the Control condition ($M = 5.11$, $SE = 1.11$), $\beta = 0.27$, $CI_{.95} = [-0.12, 0.69]$. None of the other contrasts were significant, $|\beta|$'s < 0.30 (see Figure 12).

2.2.3.   Discussion

Study 4 yielded a similar result that was found in Studies 1 to 3, such that genetic causal explanations lead to lower perceived conscious control compared to environmental causal explanations. In general, when participants think that metabolism can be attributed to a gene, they perceive weight as being less due to one's control compared to attributing metabolism to one's environment while being nurtured as an infant. One alternative explanation may be that participants perceive Jeremy's genetic causes as being present throughout his lifespan, whereas his environment as an infant is no longer present given that he is now an adult and is presumably no longer feeding on his mother's bosom. Chapter 3

addresses this issue by examining how participants' ratings of causal stability in this study

vary according to their assigned condition.
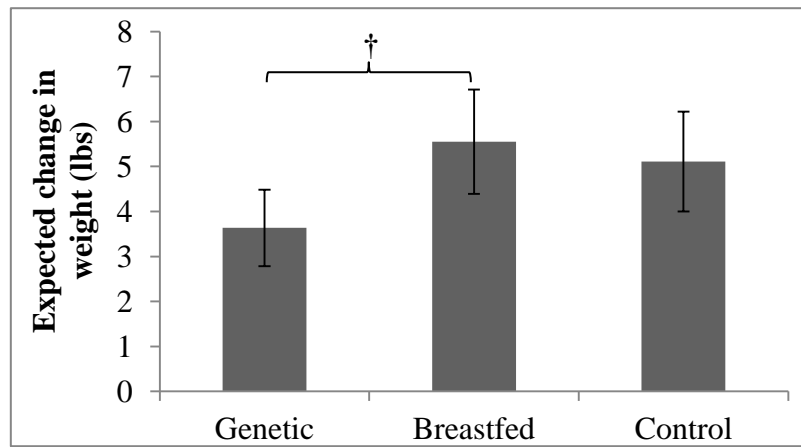
Figure 12. Expected change in weight for Jeremy over 5 years, in lbs, by condition. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

Interestingly, in all instances, participants' responses in the Control condition tended

to fall in-between the Genetic and Breastfed conditions, suggesting that their default

assumptions about individual differences in metabolism is a mix of genetic and

environmental attributions; however, given the stronger tendency for responses in the

Genetic condition to mimic those in the Control condition, there appears to be a slightly

greater endorsement of the genetic explanation as a baseline assumption. This may reflect

Neumark-Sztainer et al.'s (1999) findings discussed earlier, in which just over half of the

participants surveyed endorsed a biological explanation for obesity.

While the results of Study 4 did not reveal significant differences between conditions

in terms of expected weight change over time, the fact that participants perceived Jeremy as

having less control over their weight resulting from learning about a genetic explanation for

metabolism is still concerning. Given that perceived behavioural control is an integral

predictor of behavioural intentions based on the theory of planned behavior (Armitage &

Conner, 1999; Azjen, 1991), such beliefs may still have important implications for one's own health behaviours such as frequency of physical activity and regulation of eating behaviours (e.g. Dar-Nimrod et al., 2014). With the rate of obesity in the United States having more than doubled in the last fifty years (National Institutes of Health, 2012; Ogden, Carroll, Kit, & Flegal, 2012), genetic essentialist beliefs about obesity may end up exacerbating the problem.

2.3.   Study 5: I'm a Visual Learner – Genetic Essentialism in Learning Expectations

Beyond such important commonplace domains as criminal behaviour and obesity, genetic essentialism may also exert its influence on another lesser-discussed, but similarly contentious, topic – the construct of learning styles. Learning styles can be described as people's unique preferred ways of learning that best allow for the processing, absorption, and retention of new information (International Learning Styles Network, 2014). For almost fifty years, many educators have promulgated the importance and utility of recognizing individual learning styles in creating optimal learning outcomes (Butler & Gregorc, 1988; Dunn & Price, 1980; Sprenger, 2008). Over seventy models of learning styles have been described, altogether describing an impossibly large number of styles that often do not intersect (Coffield, Moseley, Hall, & Ecclestone, 2004). For instance, the VAK model (Barbe, Swassing, & Milone, 1979) posits that some people learn best by looking at the new material in the form of pictures or shapes (i.e. visual learning style), others learn best by listening (i.e. auditory learning style), while others learn best by using different body movements and active learning strategies to interact with new material (i.e. kinesthetic learning style). In comparison, another approach (Riechmann & Grasha, 1974) describes some people as those

who learn best by working with others (i.e. collaborative learning style), while others learn best by competing against others (i.e. competitive learning style).

According to proponents of learning styles, it is imperative for educators and parents to understand and respect the diversity that exists amongst children in terms of preferences for different ways of learning. The most important aspect of learning styles is the matching hypothesis – the assertion that learning outcomes for students are optimal when educators match their teaching styles to students' learning styles, creating a congruent learning environment (Dunn & Dunn, 1978). Evidence for this hypothesis has been lacking, and has been widely discredited within the psychological literature, as evidenced by several large-scale reviews that heavily criticized the construct and its multitudinous models (Coffield et al., 2004; Pashler, McDaniel, Rohrer, & Bjork, 2008; Riener & Willingham, 2010). Despite this paucity of evidence, learning styles continue to feature heavily in North America. For example, the National Association of Secondary School Principals ordered the creation of learning style inventories for use within the classroom (Keefe, 1988). Even the Canadian government promotes the use of learning styles in helping adults find suitable work (Service Canada, 2011). The continued focus on these models, and students' acceptance of such models, may put students at a disadvantage because an insistence on making pedagogical decisions based on a student's learning style can actually detract from their learning outcomes (Dembo & Howard, 2007).

Given how consequential the construct of learning styles is argued to be, and the resilience of the matching hypothesis amidst the lack of empirical support, Study 5 examined how perceiving learning styles as being genetic in etiology affects people's perceptions of learning styles and educational decision-making. Based on previous research, it is likely that

attributing learning styles to genetic causes would lead people to reify their beliefs in the validity of learning styles, and in the importance of the matching hypothesis. Specifically people's perceptions of learning styles in this study are broken down into four components: (a) their perceived control over one's learning style; (b) their perceived changeability of one's learning style; (c) their acceptance of learning styles; and (d) expectations of learning outcomes in relation to the matching hypothesis. Furthermore, Study 5 also sought to determine whether the educational decisions that people make might also be affected by one's etiological beliefs about learning styles. Based on results from the previous studies in this dissertation, this study tests several hypotheses: (a) stronger genetic etiological beliefs for learning styles is associated with less perceived control over one's learning style; (b) stronger genetic etiological beliefs for learning styles is associated with less perceived changeability in one's learning style; (c) stronger genetic etiological beliefs for learning styles is associated with greater acceptance of learning styles; (d) stronger genetic etiological beliefs for learning styles is associated with greater support for the matching hypothesis; and (e) stronger genetic etiological beliefs for learning styles is associated with stronger endorsement for educational decisions in line with the matching hypothesis.

2.3.1. Methods

2.3.1.1. Participants

Study 5 involved 300 participants (40% males; $M_{age} = 31.27$, $SD = 10.47$) from Amazon's MTurk. Participants received $1.25 for participating in the study.

2.3.1.2. Materials

2.3.1.2.1. Decision-Making Task

Participants first completed an educational decision-making task, during which they encountered a scenario depicting Patrick, a college student, who is choosing between two university courses based on online evaluations. He is described as having either a visual or a kinesthetic learning style. The courses are taught by two different professors, each has either a visual or a kinesthetic teaching style, and an overall rating of either 3.8 (lower rating) or a 4.3 (higher rating). Patrick's learning style, the professors' teaching styles and their overall ratings were fully randomized when the scenario was presented to the participant. Each dichotomous variable was dummy coded, such that visual learning style, visual teaching style, and having a lower rating were all coded as 0. Overall, this yielded a 2 (Learning style) × 2 (Professor 1 teaching style) × 2 (Professor 2 teaching style) × 2 (Professor 1 rating) × 2 (Professor 2 rating) within-subjects design. The participants then had to determine how much they would recommend each professor for Patrick on a 7-point scale (0 = Would completely not recommend it, 6 = Would completely recommend it).

2.3.1.2.2. Perceptions of Learning Styles

Participants rated the perceived cause of learning styles on a 6-point scale (0 = It's all due to genetics, 5 = It's all due to the environment), which served as the primary predictor for all analyses given our hypotheses. Participants then rated how much control they think people have over their learning styles on a 6-point scale (0 = No control at all, 5 = Complete control), and how likely a person can purposefully change their learning style on a 5-point scale (0 = Not at all likely, 4 = Completely likely).

Participants also expressed how much they believed in the matching hypothesis. To do this, they estimated the percentage (0% to 100%) of materials in a lecture that a visual learner can learn if the instructor teaches mainly in a) non-visual ways; versus b) visual ways.

The former (incongruent learning) was subtracted from the latter (congruent learning), such that a larger positive score denoted a greater acceptance of the matching hypothesis.

2.3.1.2.3. Learning Style Acceptance Questionnaire

Finally, participants responded to a 14-item questionnaire that was created for this study to examine the extent to which people generally accept the notion of learning styles and the matching hypothesis. Sample questions include "It is important that educators teach to a student's individual learning style" and "Everyone can be categorized into particular learning styles". Participants rated each item on a 5-point scale (0 = Strongly disagree, 4 = Strongly agree). The scale demonstrated high internal consistency (Cronbach's α = 0.89), with people generally accepting the construct of learning styles (M = 2.83, SD = 0.54).

2.3.2. Results

All regression models included participants' etiological beliefs about learning styles as the primary predictor, and also participant sex (0 = male, 1 = female) and age as covariates. All continuous variables were standardized so that the standardized coefficients can be interpreted as effect sizes on the same scale as Cohen's d's.

2.3.2.1. Decision-Making Task

Professor recommendation was analysed using multilevel modeling, with responses nested within participant numbers. All dichotomous variables were coded as 0 (lower rating; visual learning style; visual teaching style) and 1 (higher rating; kinesthetic learning style; kinesthetic teaching style).

The decision-making task revealed several interesting findings. Most intuitive is the finding that recommendation is higher for the professor who had the higher rating, β = 0.37,

p < .001. More germane to our purpose of examining participants' perception of the matching hypothesis, the data demonstrated several clear effects. Decision-making on the basis of the matching hypothesis should entail greater ratings for professors whose teaching style matches Patrick's learning style. Analyses supported this assertion. When holding Patrick's learning style constant at 0 (i.e. visual learning style), there was a significant effect of the professor's teaching style, $\beta = -0.87$, *p* < .001. In other words, when Patrick had a visual learning style, the kinesthetic professor received a lower recommendation. Similarly, when holding the professor's teaching style constant at 0 (i.e. visual teaching style), there was a significant effect of Patrick's learning style, $\beta = -0.99$, *p* < .001. In other words, when the professor used a visual teaching style, his recommendation was lower when Patrick had a kinesthetic learning style. This relation was further evidenced by a significant interaction between learning style and teaching style, $\beta = 2.06$, *p* < .001 (see Figure 13). This means that when Patrick's learning style was kinesthetic, the effect of the professor's teaching style was in the *opposite* direction, $\beta = 1.19$, *p* < .001. In other words, when Patrick had a kinesthetic learning style, the recommendation was significantly higher for the professor who used a kinesthetic teaching style, in accordance with a belief in the matching hypothesis. According to hypothesis (e) for this study, people's educational decision-making should be reified when people have stronger genetic etiological beliefs about learning styles. This suggests that the two-way interaction shown in Figure 13 should be even stronger for people who have strong genetic etiological beliefs. Counter to expectations, however, the predicted three-way interaction between learning style, teaching style, and perceived cause of learning styles was not significant, $\beta = 0.03$, *p* = .814.
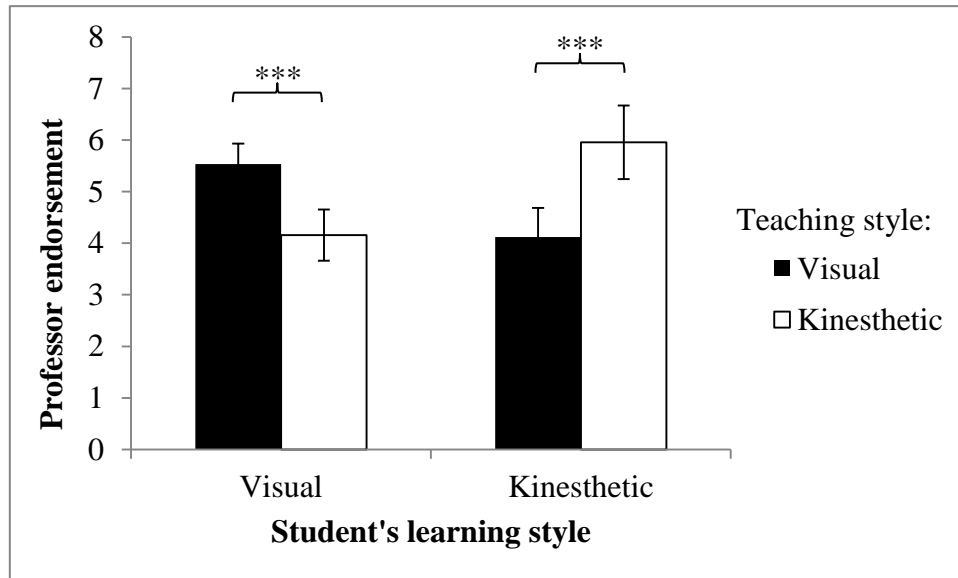
Figure 13. Endorsement of professors based on student's learning style and professors' teaching styles. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.3.2.2. Learning Style Beliefs

Perceived control, changeability, belief in the matching hypothesis, and scores on the learning styles questionnaire were all regressed onto participant sex, age, and perceived cause of learning styles. Results suggest that perceiving learning styles to be more due to genetics predicted less perceived control over one's learning style, $\beta = 0.39$, $CI_{.95} = [0.26, 0.51]$, less perceived changeability of learning styles, $\beta = 0.22$, $CI_{.95} = [0.09, 0.34]$, higher scores on the learning styles questionnaire, $\beta = -0.16$, $CI_{.95} = [-0.32, -0.02]$ (see Figure 14a), and greater belief in the matching hypothesis, $\beta = -0.17$, $CI_{.95} = [-0.28, -0.05]$ (see Figure 14b).
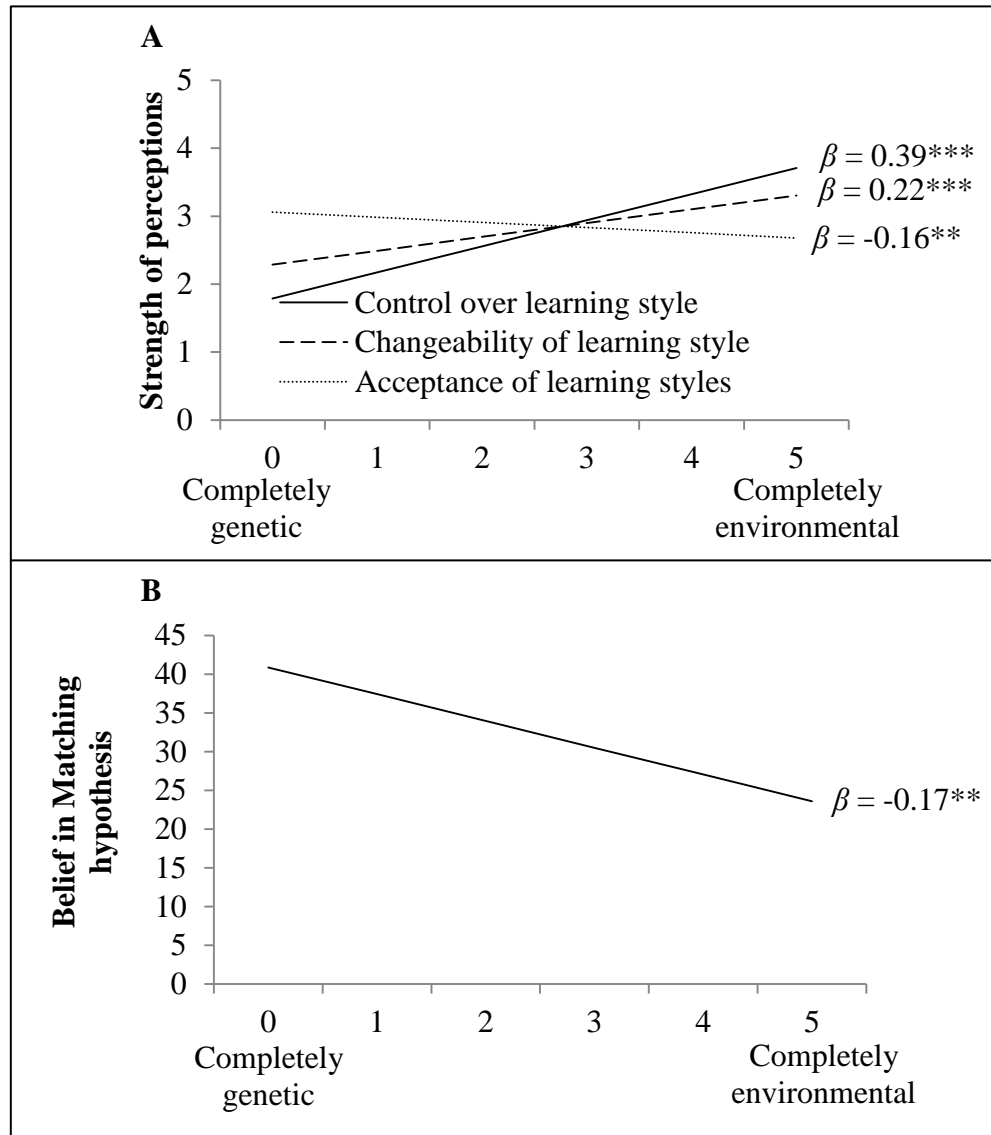
Figure 14. Panel A refers to various beliefs about learning styles. Panel B refers to people's belief in the advantage of the matching hypothesis. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.3.2.3. Mediation Analysis

In keeping with previous studies, a mediation analysis examined the role of perceived conscious control in mediating the relation between causal explanation and belief in the matching hypothesis. As reported previously, greater genetic ascription of learning styles

predicted less conscious control, $\beta = 0.39$, while greater control predicted less belief in the matching hypothesis, $\beta = -0.32$, $CI_{.95} = [-0.44, -0.20]$. The indirect effect was significant, $\beta = -0.12$, $CI_{.95} = [-0.19, -0.07]$. The significant non-mediated direct effect reported earlier, $\beta = -0.17$, became non-significant, $\beta = -0.05$, $CI_{.95} = [-0.17, 0.06]$, suggesting full mediation (see Figure 15a).

Another mediation analysis examined the role of perceived changeability in mediating the relation between causal explanation and belief in the matching hypothesis. As reported previously, greater genetic ascription of learning styles predicted less changeability, $\beta = 0.22$, while greater perceived changeability predicted less belief in the matching hypothesis, $\beta = -0.32$, $CI_{.95} = [-0.42, -0.21]$. The indirect effect was significant, $\beta = -0.07$, $CI_{.95} = [-0.12, -0.03]$. The significant non-mediated direct effect reported earlier, $\beta = -0.17$, became non-significant, $\beta = -0.09$, $CI_{.95} = [-0.19, 0.02]$, also suggesting full mediation (see Figure 15b).[7] Table 3 shows a correlation table for all variables in these mediation analyses.

	1	2	3	4	5	6
1. Etiology	1.00					
2. Perceived control	0.39***	1.00				
3. Expected changeability	0.22***	0.59***	1.00			
4. Belief in matching hypothesis	-0.21***	0.35***	-0.35***	1.00		
5. Age	-0.17**	0.02	0.04	0.14*	1.00	
8. Gender	-0.20***	-0.13*	-0.07	0.15**	0.24***	1.00

Table 3. Correlation matrix for all variables entered into the mediation analyses in Study 5. † p < .10 * p < .05, ** p < .01, *** p < .001

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[7] A path analysis determined that a model with both conscious control and changeability as simultaneous mediators of causal explanation and belief in the matching hypothesis had poor fit despite both mediators being significant in the model, CFI = 0.57, RMSEA = 0.60, SRMR = 0.11. As a result, it will not be discussed further.
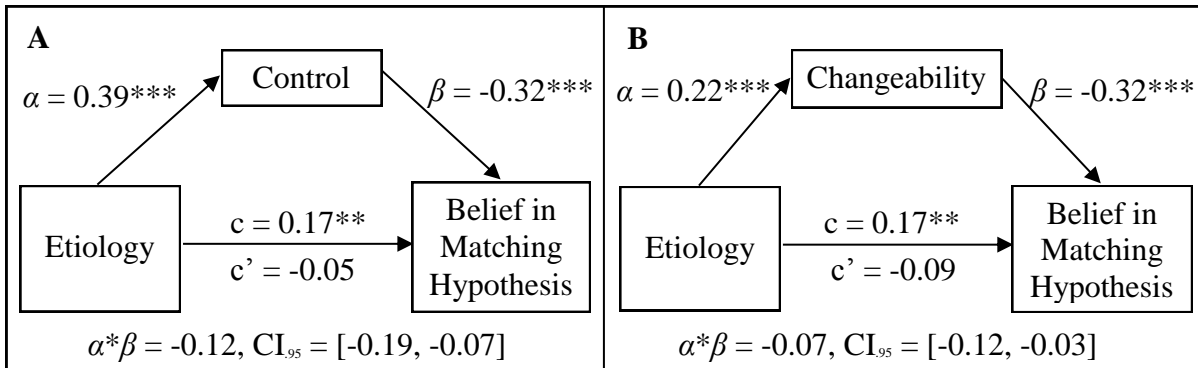
Figure 15. Mediation analyses involving perceived control over, and changeability of, learning styles in predicting belief in the Matching Hypothesis. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

2.3.3.   Discussion

Results from Study 5 suggest that there is something inherently genetic in people's acceptance of learning styles given that higher scores on the learning styles scale were also significantly associated with genetic ascriptions for learning styles. Such an etiological ascription was accompanied by a diminished perception of perceived agency, indicated by two findings. Specifically, a stronger tendency to ascribe learning styles to genetic factors was associated with less perceived conscious control over, and less perceived changeability of, learning styles. Likely wrapped up in these two beliefs is the idea of having an entity theory of the self and a malleable theory of the world (see Dweck, Chiu, & Hong, 1995; Yang & Hong, 2010). In other words, people feel that they cannot change their learning styles, which means that the external environment must adjust to the individual by necessitating teaching styles that create a congruent learning environment for learners. In accordance with this idea, ascribing learning styles to genetic causes was associated with a stronger belief in the matching hypothesis, such that people expected much better retention of lecture material when learning and teaching styles match compared to when they do not.

Taken together, these perceptions suggest that people view learning styles to be a persistent part of one's identity that guides their behaviour and expectations of the world.

Contrary to expectations, the relation between etiological ascription and belief in the matching hypothesis was not reflected in the decision-making task. The recommendations for the professors were in line with expectations purely based on the matching hypothesis, such that they were higher when teaching and learning styles matched than when they did not match; but this pattern was not exacerbated by a stronger belief in genetic etiology. The dissonance between participants' beliefs and actual recommendations may have been due to a ceiling effect. That is, people believe in the matching hypothesis so strongly, as evidenced by the large effect size, that it does not vary as a function of perceived etiology.

Despite the lack of a significant relation between one's etiological beliefs about learning styles and one's educational decision-making, this is still an important domain to explore. Given that people generally attribute learning styles more towards genetic factors than environmental factors, which was associated with a greater belief in the matching hypothesis, genetic ascriptions in this domain appears to reify the erroneous assertion that this construct is valid and consequential. This is particularly concerning given that teachers who believe in learning styles are also more likely to implement teaching strategies that match with students' different learning styles (Ballone & Czerniak, 2001). Given that the present study was a correlational study, future work should strengthen the relations found here. Importantly, additional research needs to determine whether having genetic perceptions of learning styles lead to certain behavioural outcomes. While the decision-making task here did not yield significant results, future research can examine other important behaviours such

as whether students insist on using studying methods based on some prescribed learning style despite their ineffectiveness.

2.4.   General Discussion

Five studies examined the impact of genetic essentialist thinking on subsequent cognitions in various domains, encompassing perceptions of criminal behaviour, obesity, and educational decision-making. Each domain revealed a unique set of findings that highlight the applicability of the genetic essentialism framework to explaining different important real-world phenomena. Furthermore, some consistent patterns emerged across all three domains that highlight ways in which many people tend to misinterpret and misunderstand the nature of genetic causes.

Some of the most compelling domain-specific findings emerged in Studies 1-3 on legal decision-making, whereby participants were more lenient towards a killer who was genetically predisposed to violence compared to a killer whose rearing environment predisposed him to violence. This tendency was indicated by the consistent finding that participants who learned about the genetic predisposition were more willing to endorse criminal defenses that either negated or mitigated one's *mens rea*. Furthermore, the aggregate data revealed that, in accordance with previous studies (Aspinwall et al., 2012; Dar-Nimrod et al., 2011), participants prescribed shorter prison sentences if they learned about the genetic predisposition. While these results suggest that there is a general tendency to be more amenable towards genetically-caused criminal behaviour, the same studies also revealed that a genetic predisposition also led to greater expectations for the perpetrator to reoffend compared to an environmental predisposition, highlighting the double-edged nature of genetic explanations in the context of legal decision-making. All of these results are in

accordance with expectations based on theoretical arguments made within legal studies (Levitt & Manson, 2007; Raine, 2008), and what other research data have suggested (Aspinwall et al., 2012).

Genetic perceptions of obesity and metabolism were also affected by the etiological explanation to which participants were exposed. Study 4 demonstrated that a metabolic rate that was rendered faster by genetic means was more efficient than a metabolic rate that was rendered faster by environmental means. This coincides with other research findings showing that people tended to eat more when they learned about a genetic cause for obesity rather than an environmental cause (Dar-Nimrod et al., 2014). Perhaps genetic causes led people to perceive more fatalism in their weight, which led them to feel justified in increasing food consumption.

Perceiving learning styles as genetic also had important consequences. Firstly, the results from Study 5 showed that the extent to which one accepts the construct of learning styles was strongly associated with one's tendency to attribute learning styles to genetic causes. Those who perceived learning styles to be genetic were more willing to believe that learning styles exist, that they are consequential, and that they would be difficult to change. Perhaps as a result of such perceptions, attributing learning styles to genetic causes was also associated with a stronger belief in the matching hypothesis, expecting more positive learning outcomes if learning and teaching styles match compared to when they do not. Within a sociocultural context that perpetuates the invalidated notion of learning styles at an institutional level (e.g. Service Canada, 2011), these results suggest that continued misunderstanding of genetics will only deepen the public's willingness to believe in this construct, potentially leading to unintentionally deleterious pedagogical practises.

The most important finding is that, despite the studies spanning several domains, one consistent pattern emerged – genetic explanations have the most impact on perceptions of control. Likely owing to the cognition of immutability and determinism discussed by Dar-Nimrod and Heine (2011), people generally perceived genetic causes as allowing for much less agency and control for the individual's likelihood to show a particular trait compared to environmental causes. This meant that ascribing behaviours and traits to genetic factors led to greater fatalism, perceiving people as having less control over the manifestation of those traits than would result from environmental ascriptions. This was demonstrated by the fact that participants who greatly attributed learning styles to genetic causes also expected greater difficulties in changing one's learning style. Furthermore, across the five studies, perceived conscious control generally mediated the relation between differences in etiological explanations and important outcomes. For instance, in the legal decision-making domain, genetic explanations led to weaker perceptions of control, which led to lower prison sentences. Also, in the education decision-making domain, a stronger tendency to ascribe learning styles to genetic causes was associated with less control over one's learning styles, which also predicted one's belief in the matching hypothesis. These results demonstrate that, across various domains, one of the most important effects of ascribing various aspects of human behaviour to genetic causes is the perception of weakened behavioural control, and an erosion of agency.

3.   **Chapter 3: Genetic Attributions**

The previous chapter highlighted the impact that genetic essentialist cognitions can have in various domains. More importantly, the results revealed that one consequence that appears to be inherent in attributing behaviours to genetic causes is having a lowered perception of conscious control, likely associated with the cognition of immutability and determinism discussed by Dar-Nimrod and Heine (2011). This chapter further develops that work by expanding the genetic essentialism framework to encompass additional cognitions, and one set of relevant cognitions is in understanding people's perceptions of genes as a causal agent. That is, rather than simply focusing on the consequences of genetic causes (e.g. traits that are caused by genes are immutable), the genetic essentialism framework may be expanded to include how people conceptualize the nature of genes.

Attribution theory (Abramson et al., 1978; Weiner, et al., 1971) provides a suitable supplementary framework to further explain people's perceptions of genes beyond what has been proposed by the genetic essentialism framework (Dar-Nimrod & Heine, 2011). As mentioned above, there are many reasons to expect genes to be associated with a particular suite of causal attributions. To recapitulate the previous discussion, Weiner's attribution theory proposes that people's causal attributions, or the ways in which people think about the nature of different causes, vary according to four dimensions – causal locus; causal stability; causal control; and causal specificity. Causal locus describes the extent to which one perceives the cause to be something that is internal or external to the target. Causal stability refers to whether or not the cause will continue to affect the target in the future. Causal control refers to how much control the target has over the specific cause. Causal specificity describes whether the cause affects multiple domains versus a specific domain.

Based on previous work, one would expect genetic causes to possess their own suite of causal attributions. While there is some uncertainty as to what kind of locus genes represent (Himelstein, Graham, & Weiner, 1991), it is reasonable to expect that because genes exist within an individual, they should be viewed as having an internal causal locus. Furthermore, as genes continue to exist within the individual throughout their lives, they should be seen as stable causes. In addition to this rationale, a body of research shows that greater stable attributions are associated with greater expected recidivism (Carroll, Galegher, & Wiener, 1982; Carroll & Payne, 1977). Given that the results from the aggregate dataset above demonstrated a general perception that a genetic cause of criminal behaviour leads to greater expected recidivism, genetic causes should also be seen as having stable attributions. Also, because people tend to perceive a deterministic relationship between genotypes and phenotypes (Castéra & Clément, 2014; Nelkin & Lindee, 1995), it would be reasonable to expect genetic causes to be associated with low causal control (Weiner, 2012). Finally, because contemporary studies generally portray candidate genes as particular kinds of genes (e.g. obesity gene, warrior gene), genetic causes should be seen as having high causal specificity.

The main purpose of this chapter is to determine whether or not genetic causes are associated with a certain set of causal attributions. As there are no guidelines for scores that constitute high versus low on each causal dimension, genetic causes were compared against environmental causes to determine their relative differences on the various causal dimensions. In order to do this, the research materials for Studies 2 and 3 in Chapter 2 included a measure of causal attributions in addition to the primary analyses. Specifically, these studies involved Study 2, which examined the impact of genetic and environmental causes on violent

behaviour; Study 3, which examined whether people thought genes affected behavioural impulses versus inhibition of impulses; and Study 4, which examined how people perceived obesity. Because these studies all relate to the same dependent variable measured using the same scale, discussion of these results will fall under one general discussion section at the end of this chapter.

3.1.   Studies 2-4: Perceptions of Genes as Causal Agents

3.1.1.   Methods

3.1.1.1.   Participants

As a review of the previous studies discussed in Chapter 1, Study 2 had 165 undergraduate students from the University of British Columbia (23% males; $M_{age} = 21.48$, $SD = 4.89$), with participant age, sex, and political orientation ($M = 2.47$, $SD = 0.98$) as a covariate. Study 3 had a sample size of 298 participants (42% males; $M_{age} = 34.62$, $SD = 11.26$), also with political orientation ($M = 2.63$, $SD = 1.05$) as a covariate. Study 4 had 143 undergraduate students from the University of British Columbia (25% males; $M_{age} = 20.50$, $SD = 3.88$), with participant age, sex, and BMI ($M = 21.47$, $SD = 2.95$) as a covariate.

3.1.1.2.   Materials

3.1.1.2.1.   Attributional Style Questionnaire

Participants from Studies 2 to 4 completed the Attributional Style Questionnaire (ASQ; Peterson, et al., 1982), which is a trait measure to determine the general tendency for individuals to draw certain types of causal attributions (i.e. internal, stable, domain-specific attributions). Given that one of the main purposes of these studies was to examine what people's causal attributions were of the cause specific to the scenarios in each study, the ASQ was adapted to each study as a state measure. That is, the participants were asked to respond

to each question by referencing the genetic cause discussed in the scenario in the Genetic condition, the rearing environment discussed in the scenario in the Environmental condition, or the original description their own generated cause for the control condition (i.e. "the cause of the individual's behaviour/faster metabolism"). The original ASQ contained questions that measured one's causal locus, causal stability, and causal specificity, but neglected causal control. As such, a question was created to address the missing causal control component ("Is the effect of the cause of Patrick's behaviour something that can be changed or corrected [i.e. such that the cause of Patrick's behaviour will no longer affect his behaviour?]")[8]. All questions were on a 1-7 scale, with higher scores indicating greater internal, stable, specific, and controllable attributions, separately.

3.1.1.2.2. Sentencing

Previous research has reliably demonstrated the robust link between internal attributions and other aspects of the criminal justice system, with greater internal attributions predicting greater punitiveness (Hartnagel & Templeton, 2012; Tam, Shu, Ng, & Tong, 2013) and less lenience towards prisoners (Na & Loftus, 1998). As a result, the sentencing data discussed in Chapter 2 were also used for analyses involving internal attributions for Studies 2 and 3. In particular, given that genetic causes should be seen as causally internal, and internal attributions are positively correlated with punitiveness, mediation analyses investigated the role of internal attributions in differences in prescribed sentence length between the Genetic and Environmental conditions.

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[8] A second question had also been created to test this; but participants expressed confusion over the question, and research assistants had trouble explaining the question. Thus, that question was not included in analyses.

3.1.1.2.3. Expected Recidivism

As discussed previously, there is a significant relation between stable attributions and recidivism (Carroll, Galegher, & Wiener, 1982; Carroll & Payne, 1977). Given that Chapter 2 demonstrated that genetic causes lead to greater expectations of recidivism, one may also expect genetic causes to be seen as causally stable. As such, causal stability should mediate differences between different kinds of causal explanations in expected recidivism. In order to examine this kind of relationship, previous data on expected recidivism were included in mediation analyses involving causal stability.

3.1.2.   Results

3.1.2.1.   Studies 2 and 3

3.1.2.1.1.   ASQ

Similar to the studies from Chapter 2, results for these studies were all obtained via multiple regression analyses, with the Genetic condition as the reference group and all continuous variables were standardised to generate the relevant effect sizes. Similar to Chapter 2, in the interest of simplicity, the data from Studies 2 and 3 were aggregated, contrasting the genetic explanation with the environmental explanation. Because the primary focus with this chapter was on the causal profile of a genetic cause compared to the environmental cause, the causal attributions of the other gene-based explanations in Study 3 are not discussed here. Graphs of the analyses from each study can be found in Appendix G.

Based on this aggregate dataset, within the context of explanations for violent behaviour, the genetic explanation was seen as a very different cause compared to the environmental explanation. For causal locus, the genetic explanation ($M = 5.65$, $SE = 0.16$) was seen as more internal than the environmental explanation ($M = 4.73$, $SE = 0.17$), $\beta = $ -

0.64, CI$_{.95}$ = [-0.87, -0.41]. Also, in accordance with expectations based on the effect of genetic explanations on perceived recidivism, the genetic cause was perceived as being more causally stable (M = 5.42, SE = 0.13) than the environmental cause (M = 5.10, SE = 0.14), β = -0.28, CI$_{.95}$ = [-0.52, -0.04]. Furthermore, causal specificity was higher in the Genetic condition (M = 3.06, SE = 0.14) than in the Environmental condition (M = 2.41, SE = 0.15), β = -0.50, CI$_{.95}$ = [-0.74, -0.27]. Finally, the genetic cause was seen as being less causally controllable (M = 4.43, SE = 0.16) than the environmental cause (M = 5.05, SE = 0.17), β = 0.44, CI$_{.95}$ = [0.20, 0.68] (see Figure 16).
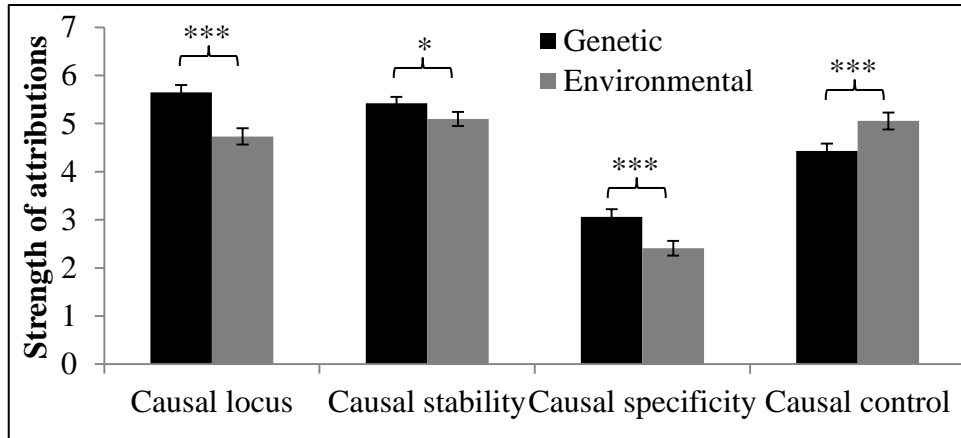
Figure 16. Different causal attributions for different conditions. Higher scores indicate more internal, stable, specific, and controllable attributions. Error bars represent standard errors. † *p* < .10 * *p* < .05, ** *p* < .01, *** *p* < .001

3.1.2.1.2. Mediation Analysis

Overall, a mediation analysis revealed the predicted indirect effect of behavioural explanations on sentencing through internal attributions (see Figure 17a). The results suggested that participants in the Environmental condition perceived the cause of Patrick's behavior to be less internal than participants in the Genetic condition, β = -0.64, CI$_{.95}$ = [-0.87, -0.41]. On the other hand, more internal causal attributions were associated with lengthier sentences, β = 0.19, CI$_{.95}$ = [0.07, 0.32]. The indirect effect was significant, β = -0.12, CI$_{.95}$ =

[-0.22, -0.04]. The non-mediated direct effect was marginally significant, $\beta = 0.17$, $p = .097$, $CI_{.95} = [-0.03, 0.38]$, while the mediated direct effect became larger and statistically significant, $\beta = 0.30$, $CI_{.95} = [0.07, 0.53]$, suggesting a suppression effect.

Another mediation analysis found the predicted indirect effect of behavioural explanations on expected recidivism through causal stability (see Figure 17b). The results suggested that participants in the Environmental condition perceived the cause of Patrick's behavior to be less stable than participants in the Genetic condition, $\beta = -0.28$, $CI_{.95} = [-0.54, -0.04]$. On the other hand, more stable causal attributions were associated with greater perceived recidivism, $\beta = 0.16$, $CI_{.95} = [0.03, 0.32]$. The indirect effect was significant, $\beta = -0.05$, $CI_{.95} = [-0.10, -0.00]$. The non-mediated direct effect was significant, $\beta = -0.31$, $CI_{.95} = [-0.56, -0.07]$, while the mediated direct effect was now marginally significant, $\beta = -0.26$, $CI_{.95} = [-0.53, 0.01]$
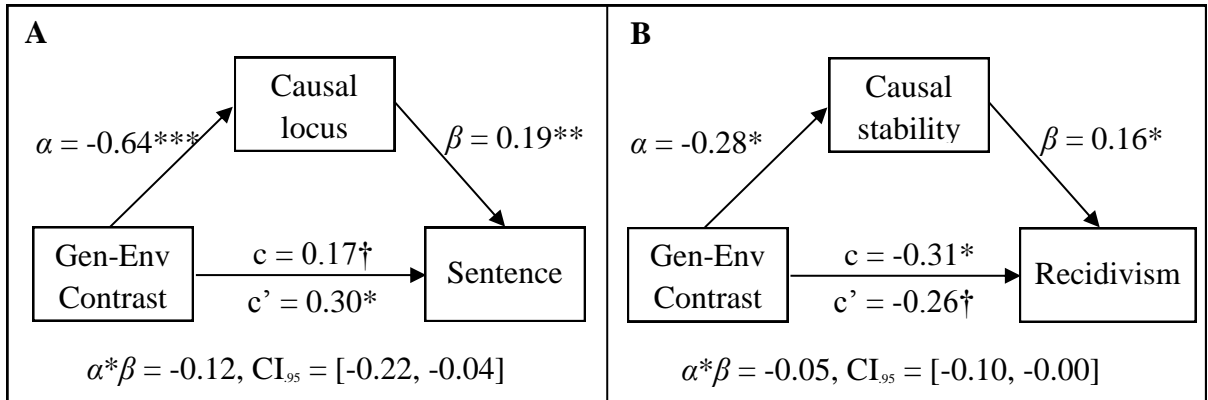
Figure 17. Mediation analyses examining causal locus and causal stability in modulating the relation between different conditions for sentencing and expected recidivism, respectively. All effect sizes are standardized. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

3.1.2.2.   Study 4

I further explored the applicability of attribution theory to the genetic essentialism framework by examining the causal attributions that people have for genetic explanations of variability in metabolism.

3.1.2.2.1. ASQ

The results of the ASQ from Study 4 can be seen in Figure 18. In terms of causal locus, the genetic explanation was seen as more internal ($M = 4.95$, $SE = 0.37$) than the breastfeeding explanation ($M = 3.54$, $SE = 0.33$), $\beta = -0.90$, $CI_{.95} = [-1.32, -0.48]$, but similar to the Control condition ($M = 4.51$, $SE = 0.29$), $\beta = -0.30$, $CI_{.95} = [-0.67, 0.10]$. The latter two conditions differed significantly from each other, with the breastfeeding explanation being seen as much more external than in the Control condition, $\beta = 0.60$, $CI_{.95} = [0.18\ 0.47]$.

The conditions were similarly causally stable, $|\beta|$'s < 0.20.

For causal specificity, the genetic explanation was perceived as being marginally more domain-specific ($M = 3.55$, $SE = 0.27$) than the breastfeeding explanation ($M = 2.97$, $SE = 0.32$), $p = .066$, $\beta = 0.41$, $CI_{.95} = [-0.02, 0.85]$, and the Control condition ($M = 3.02$, $SE = 0.31$), $p = .075$, $\beta = 0.37$, $CI_{.95} = [-0.06, 0.81]$. The latter two conditions did not differ from each other, $\beta = -0.04$, $CI_{.95} = [-0.44, 0.37]$.

Finally, causal control was different across conditions, with the genetic cause seen as being much less controllable ($M = 3.20$, $SE = 0.32$) than the environmental cause ($M = 4.26$, $SE = 0.35$), $\beta = 0.65$, $CI_{.95} = [0.23, 1.06]$, and the Control condition ($M = 3.91$, $SE = 0.35$), $\beta = 0.42$, $CI_{.95} = [0.01, 0.83]$. The latter two conditions did not differ significantly from each other, $\beta = -0.23$, $CI_{.95} = [-0.64, 0.20]$.
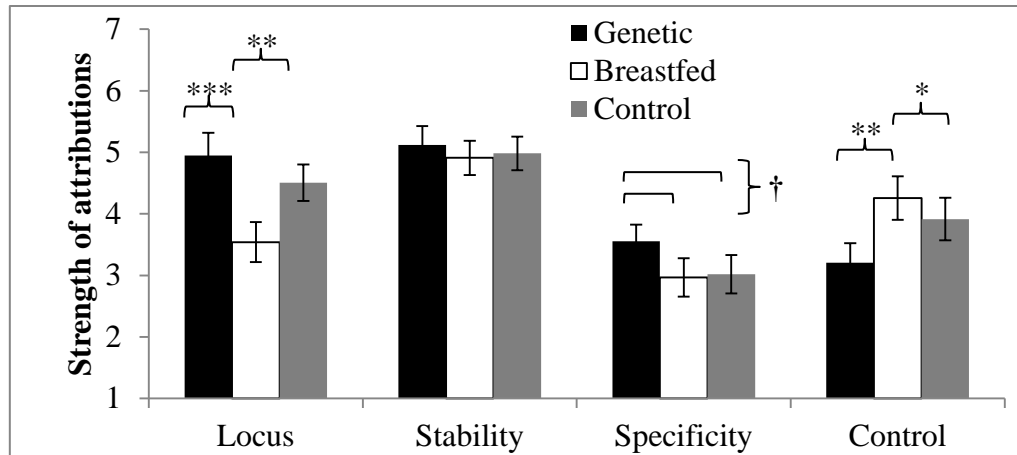
Figure 18. Causal attributions for different causal explanations of metabolism. Error bars represent standard errors. † *p* < .10 * *p* < .05, ** *p* < .01, * *p* < .001**

Overall, compared to the breastfeeding explanation for obesity, the genetic explanation for obesity was seen as being more internal but less controllable. Furthermore, it was seen as being marginally more domain-specific.

3.2.　　　General Discussion

Three studies examined the different types of causal attributions that people make for different kinds of behavioural explanations. Studies 2 and 3 examined the domain of criminal behaviour, while Study 4 examined the domain of obesity. Despite the variability in domains, genetic causes were consistently seen as being more internal, more domain-specific, and less controllable than environmental causes. These consistent differences may reflect fundamental attributions that people draw when they encounter genetic causal explanations, particularly when juxtaposed with environmental causal explanations. On the other hand, these causal explanations do not consistently differ in causal stability. Whereas the aggregate data on perceptions of criminal behaviour demonstrated that genetic causes were seen as being more causally stable than environmental causes, this did not manifest in the obesity data. One possibility is that this variation between domains is due to the particular comparisons being

made. The criminal behaviour scenario compares a genetic cause against an obvious environmental cause (i.e. the perpetrator's rearing environment), while the obesity scenario compares a genetic cause against another cause that is not as clearly environmental (i.e. being breastfed), the latter of which may conjure up unintended biological constructs such as the impact on hormonal development. This explanation suggests that the basis of comparison is a vital point of consideration for future research into understanding the causal attributions associated with genetic explanations.

Another important set of findings regarding participants' causal attributions relates to their role in mediating the effect of different behavioural explanations on important outcomes such as perceived recidivism and prescribed sentence length, particularly involving causal locus and causal stability. Much work has demonstrated the relation between causal locus on perceptions of criminal guilt, with internal attributions predicting lengthier prison sentences (Templeton & Hartnagel, 2012). Causal locus can also explain people's differential support for various forms of public policy, such that greater tendency to engage in internal attributions is associated with greater support for increased incarceration rather than rehabilitation for criminals (Thompson & Bobo, 2011). Reflecting such preferences for punitiveness, participants in Studies 2 and 3 perceived the genetic causes as being a more internal cause than environmental causes, and greater internal causal attributions were associated with longer prison sentences. This finding, in conjunction with the mediation analyses from Chapter 2, suggests that ascribing criminal behaviour to genetics activates multiple mechanisms that act in conjunction with, and in opposition to, each other in affecting people's punitive tendencies.

In addition to causal locus being a significant mediator, causal stability is also an important mechanism in explaining how genetic ascriptions affect the legal decision-making process. As discussed in Chapter 2, previous research has demonstrated that people also make legal decisions based on expectations about a perpetrator's likelihood to reoffend, whereby higher expectations of recidivism predicts greater dangerousness of an offender (Sanderson, Zanna, & Darley, 2000), and lower likelihood of granting parole (Carroll & Payne, 1977). This concern about recidivism may be associated with the presumption that the criminogenic factor continues to impact the individual in the future. This suggests that a cause that is causally stable should also lead to higher expectations of recidivism, such that causal stability should be a significant mediator between different explanations and expected recidivism. Indeed, the aggregate data demonstrated that causal stability significantly mediates the impact of various behavioural explanations on expected recidivism. Specifically, participants perceived genetic causes as being more stable than environmental causes, and greater causal stability was associated with greater expected recidivism. These causal attributions were also added to the path analysis model discussed in Figure 13 of Chapter 2. The new model, seen in Figure 19, demonstrates decent fit, CFI = 0.93, RMSEA = 0.12, SRMR = 0.04, and shows the numerous significant pathways that explain how genetic causes can affect sentencing in various ways.

Based on previous research by Sanderson et al. (2000), these results suggest that people should support rehabilitative efforts to reform criminals if the criminal behaviour was explained as having an environmental etiology. On the other hand, perceiving criminal behaviour to be due to genetic causes should lead people to support incarceration as the preferable punishment for criminals. Thus, future work should examine how genetic

behavioural explanations impact people's support for various social policies in different domains including the criminal justice system.
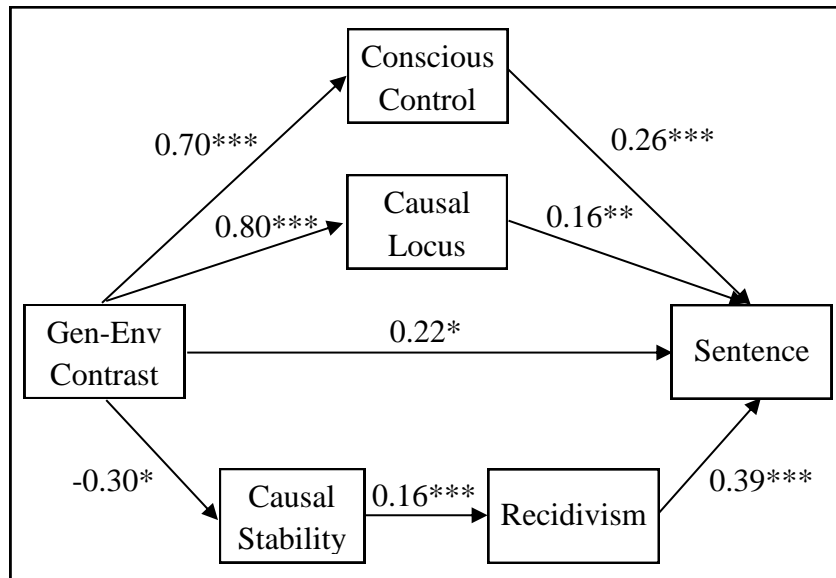
Figure 19. Path analysis of all theoretically predicted pathways in predicting sentencing. All effect sizes are standardized. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

Overall, the findings in this chapter demonstrate two important ideas. First, genetic causes have a certain profile of causal attributions, such that they are seen as being causally internal, stable, and uncontrollable; however, there are methodological issues to consider because there are no clear guidelines as to what scores on the ASQ should be taken as being high or low on the causal dimensions. Profiles of causal attributions are only interpretable if the genetic cause has a meaningful basis of comparison. The studies described in this chapter generally found that causal profiles differed between genetic and environmental causes; but it is unclear whether the same results would emerge if the basis of comparison were non-environmental and non-genetic (e.g. a neurological explanation). While no work has currently examined this issue, there is some research suggesting that neurological causes of behaviour may be seen as similar to genetic causes, and thus are not likely to yield significant differences in causal attributions relative to genetic causes (Appelbaum et al., 2015;

Schweitzer, et al., 2011). This may affect one's ability to discern a unique profile of causal attributions for genetic causes.

The second major point that this chapter illustrates is the importance and utility of causal attributions as social cognitive mechanisms in explaining how different behavioural explanations may impact important outcomes. For instance, rather than seeing a simple relation between different explanations and prescribed sentence length, causal locus and stability can be identified as important mediators that explain this relation in conjunction with other countervailing social cognitive mechanisms, providing a much richer understanding of the varied nature of different underlying cognitive processes. Future work should examine a wider set of behavioural and attitudinal outcomes, and determine the unique predictive role of different causal attributions.

4.   Chapter 4: Mitigating Genetic Essentialism

The two preceding chapters provided much evidence that people tend to essentialize genes. Owing to people's tendency to associate genes with perceptions of determinism and fatalism, they also ascribe less conscious control and agency to individuals when different traits and behaviours are discussed as having genetic, rather than environmental, causes. Moreover, genetic causes are associated with certain types of causal attributions compared to environmental causes. As discussed previously, such deterministic perceptions of genetic effects are often unwarranted, given that effect sizes of specific genes are extremely small (e.g. Bertram & Tanzi, 2005; Hinney, et al., 2006), accounting for a small proportion of variance for any given trait. All of these genetic factors likely act in conjunction with each other and with other environmental forces to produce resultant phenotypes, although how these different factors all fit together to predict outcomes is still highly speculative (Kraemer, Stice, Kazdin, Offord, & Kupfer, 2001; Rutter, 2009). These processes highlight the fact that there is no simple, deterministic link between genetic factors and any subsequent characteristics.

Given that the general perception of genetic effects is highly simplistic and inaccurate, subsequently leading to genetic essentialist biases, it is important to find ways to mitigate those biases. The fact that people are demonstrating an essentialized perception of genes despite the objectively complex mechanisms behind genetic effects leads to two possible explanations. The first is that people are unfamiliar with the complexity of genetic effects, believing that genes do affect outcomes in a deterministic fashion. This may be explained by the fact that there is a demonstrable lack of knowledge about genetics amongst the general public (e.g. Lanie, et al., 2004). Another explanation may be that people *are* aware of the role

of complex interactions involving genetic factors and other factors; but it may be cognitively less demanding to rely on a simpler heuristic of reasoning based on singular causal factors. Indeed, concerns about public scientific literacy often involve discussions about the public misapprehending complex scientific ideas, ultimately leading scholars to fall back on more intuitive and simplistic metaphors (for a review, see Ratto, 2006). In a culture that finds genetic effects so compelling due to oversimplified messages about them (Mcinerney, Bird, & Nucci, 2004; Nelkin & Lindee, 1995), it may be that people ignore the more complex explanations such that their genetic essentialist biases are activated simply because such complex explanations partially involve genetics. As such, having an accurate apprehension of, and appreciation for, the complex picture of how genotypes affect phenotypes should mitigate genetic essentialist biases. Few studies have examined the impact of emphasizing the combined effect of biological and environmental factors in attenuating the impact of genetic essentialism. One such study investigated this issue in relation to stigma on mental illness, in which Boysen and Gabreski (2012) found that combining environmental and biological explanations did not consistently impact mental health stigma more than biological explanations alone. These findings seem to support the second explanation posited above given that participants were made aware of a more complex causal explanation, thus negating the applicability of the first explanation. It appears that even after more complex and nuanced explanations have been presented, people simply revert to a simplified, essentialized perspective of genetic effects.

Against this backdrop of a discrepancy between the objective complexity of genetic causes and the public's overly simplistic assumptions about genetic effects, it is prudent to examine ways to mitigate such biased views. One important direction to explore in terms of

mitigating genetic essentialist cognitions is to determine whether certain characterizations of the complex relationship between genotypes and phenotypes are more effective than others. By making participants more aware of the different forms of complex relationship between genotypes and phenotypes, it may be possible to determine the most effective framing of genetic effects that can effectively mitigate genetic essentialist biases. As such, the purpose of the subsequent studies discussed in this chapter was to examine how exposure to different types of complex characterizations of genetic effects can affect people's genetic essentialist cognitions. Given the robust findings that were reported in Chapter 2 regarding legal decision-making, Studies 6 to 8 used the same general scenario from Studies 1 to 3, with some relevant changes, to serve as a case study with which to examine the impact of various mitigating strategies on genetic essentialist cognitions. In particular, the three studies examined the effect of framing the complex genetic effects using a) an interactionist account; b) a monogenic versus polygenic account; and c) a weak versus strong genetic account.

4.1. Study 6: Genes Interacting With the Environment

4.1.1. Methods

4.1.1.1. Participants

Study 6 involved 412 participants (54% males; $M_{age} = 35.14$, $SD = 11.86$) from MTurk. The sample consisted of 74% Euro-Americans, 7% African-Americans, 5% East Asians, 13% Others, and 1% unspecified. As with previous studies, participants indicated their political orientation using a 5-point scale (1 = Very liberal, 5 = Very conservative; $M = 2.56$, $SD = 1.08$). Participants received $0.50 for participating in the study.

4.1.1.2.   Materials

The experimental manipulation consists of the same Genetic ($n = 131$) and Environmental ($n = 140$) conditions as those used in Studies 1 and 2 (i.e. Patrick had committed a murder either due to his genes or his rearing environment). In addition to those conditions, this study also included an extra Interaction condition ($n = 153$), which described the cause of Patrick's increased likelihood of engaging in violent behaviour as an interaction between genetic and environmental factors. This was based on research demonstrating that people with a certain genetic variant, and who grew up in an environment of childhood maltreatment, were more likely to engage in violence in adulthood (Caspi, et al., 2002). The Environmental condition was kept in this study because it was important to determine whether people considered interactionist behavioural explanations to be more similar to genetic explanations or environmental explanations. Similar to the Genetic and Environmental conditions, the Interaction condition described the cause as being responsible for a fourfold increase in Patrick's likelihood of being violent (see Appendix H).

All dependent variables used in this study were same as the trimmed down set that was used in Study 3. This entails questions about the perceived applicability of different criminal defenses, prison sentence length, and various perceptions of Patrick as a criminal, including perceived conscious control, criminal intent, and expected recidivism. This study also included the Attributional Style Questionnaire.

4.1.2.   Results

All regression results in this study were performed having dummy coded each of the conditions, with the Interaction condition being the reference group. In order to obtain the effect size of the Genetic-Environmental contrast, separate regression models were analyzed

with the Genetic condition as the reference group. Again, participant age, sex, and political orientation were included as covariates, and all continuous variables were standardized to generate effect sizes that can be interpreted on the same scale as Cohen's d's. This design allowed for analyses into the impact of interactionist accounts relative to genetic and environmental accounts.

4.1.2.1.   Defense Claims

Results for the defense claims can be seen in Figure 20. There was a main effect of Explanation on the applicability of the insanity defense. The Interaction condition led to similar degrees of perceived applicability compared to the Genetic condition, but significantly higher than the Environmental condition. In particular, endorsement was higher in the Interaction condition ($M = 3.25$, $SE = 0.18$) than in the Environmental condition ($M = 2.32$, $SE = 0.23$), $\beta = -0.47$, $CI_{.95} = [-0.69, -0.25]$, but not in the Genetic condition ($M = 3.36$, $SE = 0.24$), $\beta = 0.05$, $CI_{.95} = [-0.19, 0.31]$. The latter two conditions differed significantly from each other, with endorsement being higher in the Genetic condition than in the Environmental condition, $\beta = -0.52$, $CI_{.95} = [-0.76, -0.30]$.

The same pattern emerged with the perceived applicability of the diminished capacity defense, wherein participants preferred it more in the Interaction condition ($M = 4.64$, $SE = 0.17$) compared to those in the Environmental condition ($M = 3.69$, $SE = 0.23$), $\beta = -0.48$, $CI_{.95} = [-0.71, -0.26]$, but not to those in the Genetic condition ($M = 4.54$, $SE = 0.24$), $\beta = -0.05$, $CI_{.95} = [-0.29, 0.19]$. The latter two conditions differed significantly from each other, with endorsement being higher in the Genetic condition than in the Environmental condition, $\beta = -0.43$, $CI_{.95} = [-0.67, -0.18]$.

The intoxication defense was seen as less applicable in the Interaction condition ($M =$ 1.37, $SE = 0.18$) than in the Environmental condition ($M = 1.72$, $SE = 0.13$), $\beta = 0.37$, $CI_{.95} =$ [0.11, 0.64], but not compared to the Genetic condition ($M = 1.26$, $SE = 0.09$), $\beta = -0.12$, $CI_{.95} = [-0.30, 0.08]$. In a departure from earlier studies, the latter two conditions differed significantly from each other, with endorsement being higher in the Environmental condition than in the Genetic condition, $\beta = 0.48$, $CI_{.95} = [0.24, 0.73]$.
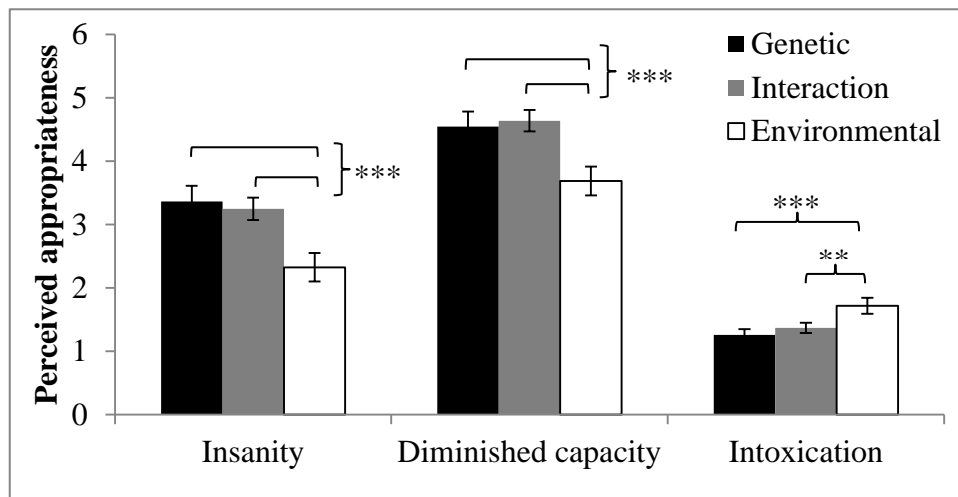
Figure 20. Effect of different causal explanations of perceived applicability of different defense claims. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.1.2.2.   Sentencing

Sentencing did not differ between the different conditions, $|\beta|$'s < 0.10 (see Figure 21).
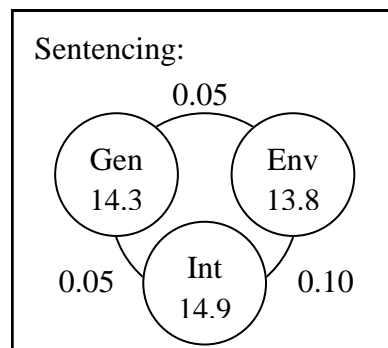
Figure 21. Adjusted means and absolute standardized effect sizes comparing sentencing between conditions. Gen = Genetic, Env = Environmental, Int = Interaction † p $< .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.1.2.3. Perceptions of the Perpetrator

Results regarding perceptions of the perpetrator can be seen in Figure 22. Explanation had a weak effect on the extent to which participants felt that Patrick had conscious control over his violent behaviour, with the responses of participants in the Interaction condition being intermediate between responses in the Genetic and Environmental conditions. In particular, participants in the Interaction condition perceived marginally greater control ($M = 4.90$, $SE = 0.10$) than those in the Genetic condition ($M = 4.63$, $SE = 0.15$), $p = .081$, $\beta = -0.23$, $CI_{.95} = [-0.46, 0.02]$, but marginally less control than those in the Environmental condition ($M = 5.15$, $SE = 0.14$), $p = .089$, $\beta = 0.20$, $CI_{.95} = [-0.02, 0.42]$. The latter two conditions differed significantly from each other, being lower in the Genetic condition than in the Environmental condition, $\beta = 0.41$, $CI_{.95} = [0.17, 0.68]$.
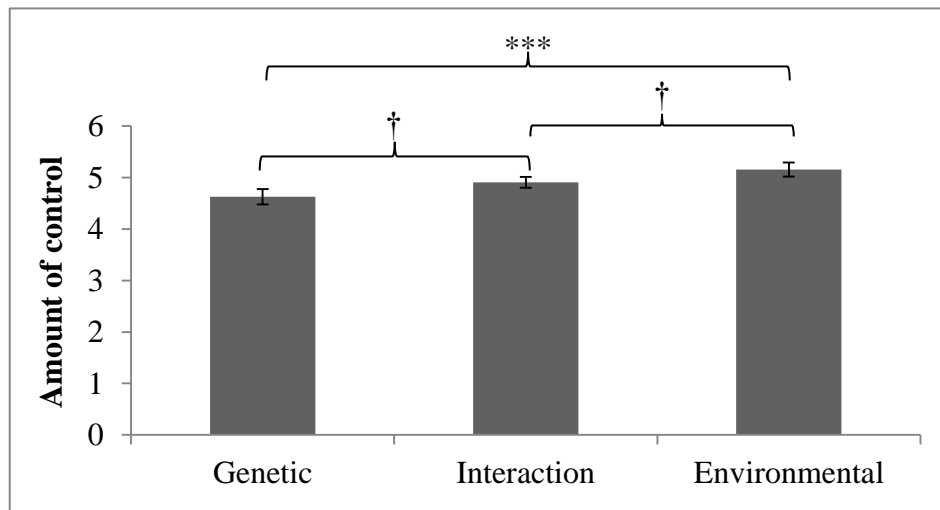
Figure 22. Differing amount of perceived control based on condition. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

Perceived intention to kill was marginally affected by Explanation, with intention being marginally higher in the Interaction condition ($M = 3.98$, $SE = 0.15$) than in the Genetic condition ($M = 3.61$, $SE = 0.21$), $p = .058$, $\beta = -0.23$, $CI_{.95} = [-0.48, 0.02]$, but not different from the Environmental condition ($M = 3.95$, $SE = 0.18$), $\beta = -0.02$, $CI_{.95} = [-0.24,$

0.20]. The latter two conditions were marginally different from each other, with greater

perceived intention in the Environmental condition than in the Genetic condition, $p = .089$, β

$= 0.21$, $CI_{.95} = [-0.05, 0.46]$ (see Figure 23).

The Interaction condition did not differ significantly from the other two conditions in

terms of criminal responsibility, $|\beta|$'s < 0.20; however, participants in the Genetic condition

perceived less criminal responsibility ($M = 7.09$, $SE = 0.13$) than those in the Environmental

condition ($M = 7.44$, $SE = 0.12$), $\beta = 0.33$, $CI_{.95} = [0.07, 0.56]$ (see Figure 23).

The perceived recidivism in the Interaction condition was in-between the Genetic and

Environmental conditions, such that it was marginally higher in the Interaction condition (M

$= 4.82$, $SE = 0.11$) than in the Environmental condition ($M = 4.56$, $SE = 0.15$), $p = .079$, $\beta = -$

0.21, $CI_{.95} = [-0.44, 0.02]$, but not different from the Genetic condition ($M = 4.73$, $SE = 0.15$),

$\beta = -0.08$, $CI_{.95} = [-0.32, 0.16]$. The two latter conditions did not significantly differ from

each other, $\beta = -0.13$, $CI_{.95} = [-0.38, 0.11]$ (see Figure 23).
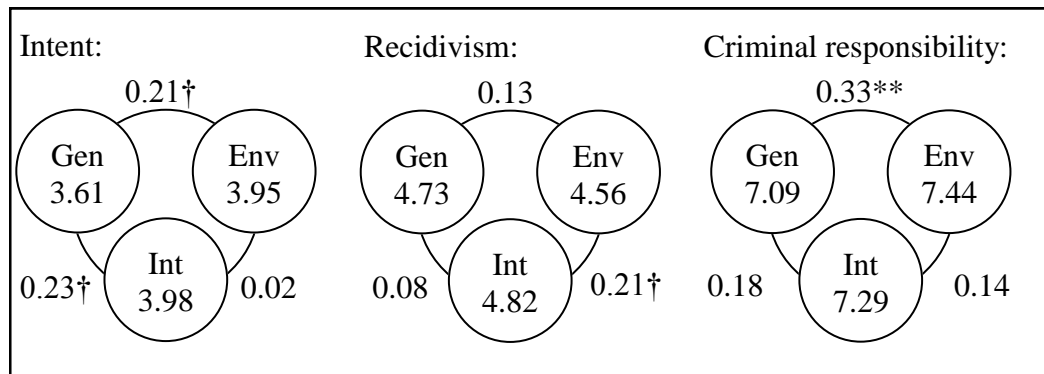
Figure 23. Adjusted means and absolute standardized effect sizes for comparing various perceptions of the perpetrator between conditions. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.1.2.4.   ASQ

All results regarding the ASQ can be seen in Figure 24. In terms of causal locus, the

Interaction condition was seen as less internal ($M = 4.95$, $SE = 0.14$) than the Genetic

condition ($M = 5.69$, $SE = 0.17$), $\beta = 0.48$, $CI_{.95} = [0.26, 0.70]$, but similar to the

Environmental condition ($M = 4.96$, $SE = 0.19$), $\beta = 0.01$, $CI_{.95} = [-0.23, 0.24]$. The latter

two conditions differed significantly from each other, being more internal in the Genetic

condition than in the Environmental condition, $\beta = -0.47$, $CI_{.95} = [-0.70, -0.24]$.

With regard to causal stability, the Interaction condition was seen as being more

causally stable ($M = 5.85$, $SE = 0.10$) than both the Environmental ($M = 5.44$, $SE = 0.13$), $\beta = -0.35$, $CI_{.95} = [-0.57, -0.12]$, and Genetic conditions ($M = 5.33$, $SE = 0.14$), $\beta = -0.44$, $CI_{.95} = [-0.67, -0.21]$. The latter two conditions did not differ significantly from each other, $\beta = 0.10$, $CI_{.95} = [-0.16, 0.35]$.

For causal specificity, the Interaction condition was perceived as being in-between

the Genetic and Environmental conditions. In particular, domain-specificity was higher in the

Genetic condition ($M = 3.21$, $SE = 0.18$) than in the Interaction condition ($M = 2.85$, $SE = 0.14$), $\beta = 0.25$, $CI_{.95} = [0.00, 0.50]$, but it was higher in the Interaction condition than in the

Environmental condition ($M = 2.21$, $SE = 0.16$), $\beta = -0.45$, $CI_{.95} = [-0.66, -0.24]$. The Genetic

and Environmental conditions differed significantly from each other, being more domain-

specific in the Genetic condition than in the Environmental condition, $\beta = -0.71$, $CI_{.95} = [-0.94, -0.48]$.

Finally, causal control was lowest in the Interaction condition ($M = 4.12$, $SE = 0.16$),

which did not differ significantly from the Genetic condition ($M = 4.28$, $SE = 0.20$), $\beta = 0.10$, $CI_{.95} = [-0.15, 0.34]$, but it was significantly lower compared to the Environmental condition

($M = 4.81$, $SE = 0.19$), $\beta = 0.43$, $CI_{.95} = [0.20, 0.66]$. The Genetic and Environmental

conditions also differed significantly from each other, being higher in the Environmental condition than in the Genetic condition, $\beta = 0.33$, CI$_{95}$ = [0.09, 0.58].

Overall, compared to the environmental cause, the interactionist cause was seen as being more causally stable and domain-specific, but less controllable.
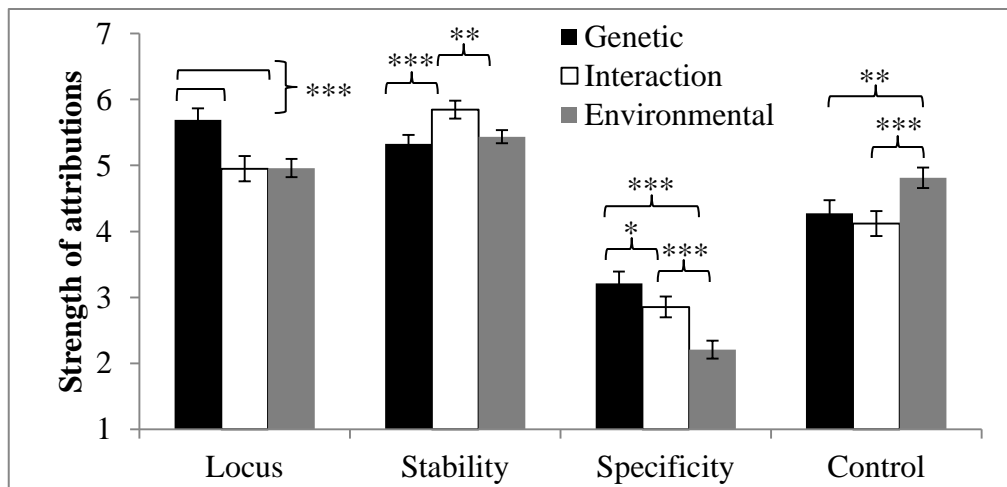
Figure 24. Causal attributions for various causal explanations. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.1.3.   Discussion

Study 6 examined whether framing genetic effects in more complicated terms can mitigate genetic essentialist biases. In particular, this study focused on portraying genes as interacting with an environmental factor, subsequently leading Patrick to develop his violent tendencies.

In terms of criminal defense claims, this study replicated findings from Chapter 2, such that the perceived applicability of both the insanity and the diminished capacity defenses were higher when the causal explanation was genetic rather than environmental. More importantly, the interactionist account did not differ from the genetic explanation on

either of these ratings, but led to greater perceived applicability than the environmental explanation.

While the findings in relation to the defense claims seem to suggest that interactionist accounts were perceived in similarly essentialist terms as genetic explanations, the findings regarding people's perceptions of the perpetrator provided a murkier pattern. The first set of findings demonstrated that the interactionist account led to an intermediate response between genetic and environmental explanations. For instance, the interactionist account led to less perceived control than the environmental explanation, but greater perceived control than the genetic explanation. A similar finding evinced with criminal responsibility. Although the interactionist account did not differ significantly from the genetic and environmental accounts, the latter two explanations did differ significantly, with the interactionist account situated in the middle. Indeed, the environmental explanation led to the highest level of criminal responsibility ($\beta = 0.15$ when contrasted to the interactionist account), while the genetic explanation led to the lowest level of criminal responsibility ($\beta = -0.18$ when contrasted to the interactionist account), although neither of these effects were significant.

The remaining patterns saw the interactionist account resemble one of the component explanations. For example, perceived intention to kill was similar between the interactionist account and the environmental explanation, while both of these explanations were marginally different from the genetic explanation. Conversely, expected recidivism was similar between the interactionist and the genetic accounts.

This inconsistent pattern involving the interactionist account was also found amongst the ASQ items. The interactionist explanation was intermediate for domain-specificity, such

that the genetic explanation was the most domain-specific while the environmental explanation was the most domain-general, and the interactionist explanation being situated in-between them. In comparison to this pattern, the interactionist explanation was considered to be as causally external as the environmental explanation; but it was just as causally uncontrollable as the genetic explanation.

One point of interest is that the findings in this study mimicked what was found by Boysen and Gabreski (2012). Whereas they characterised their study as not having found that an interactionist account affected people's stigma against mental health conditions differently than genetic and environmental explanations alone, careful perusal of their data suggest that they simply found that people responded to the interactionist account in the same three ways that were found in the present study. That is, sometimes the interactionist response was in-between the genetic and environmental responses, while other instances had the interactionist response resembling responses from either of the component explanations. In a similar vein, the perceived effectiveness of a mental health treatment that combined efforts to change one's environment and involved biological interventions such as pharmacological treatments to be intermediately effective compared to each component treatment alone (Deacon & Baird, 2009). These suggest that people are likely to respond to interactionist accounts of behaviours similarly across different domains.

The findings in Study 6 suggest that framing genetic effects as being complicated by interactions with environmental factors may mitigate genetic essentialist tendencies by pushing people's perceptions to some intermediate response between genetic and environmental explanations alone. And while there are some instances in which the interactionist explanation leads to responses that mimic the environmental explanation's

responses (i.e. perceived recidivism and causal locus), the remainder of the responses generally resembled genetically-based responses (i.e. insanity defense, diminished capacity defense, perceived intention to kill, and causal control). Taken together, people appear to interpret interactionist accounts appropriately as a combination of the two component explanations, which did mitigate some level of genetic essentialist responses; however, it cannot fully mitigate genetic essentialist biases as it still tends to lead to responses that mimic those elicited by genetic explanations.

4.2. Study 7: One Gene vs. a Set of Genes

Study 6 presented a more complicated account of genetic effects by giving participants an interactionist explanation. The present study examined the impact of another complicated account of genetic effects – multiple genes acting in conjunction with each other to lead to the expression of a trait. For example, a large-scale genome-wide association study discovered that thousands of genetic alleles, each accounting for a small effect, contribute to the risk of developing schizophrenia and manic depressive disorder (The International Schizophrenia Consortium, 2009). In other words, schizophrenia and manic depressive disorder are polygenic traits rather than monogenic traits that only involve one genetic mutation. The present study used the same genetic mechanism to explain one's aggressive behaviour.

4.2.1. Methods

4.2.1.1. Participants

Study 7 involved 494 participants (55% males; $M_{age} = 32.76$, $SD = 10.36$) from MTurk. The sample consisted of 78% Euro-Americans, 6% African-Americans, 8% East Asians, 8% Others, and 1% unspecified. As with previous studies, participants indicated their

political orientation using a 5-point scale (1 = Very liberal, 5 = Very conservative; $M = 2.59$, $SD = 1.04$). Participants received \$0.50 for participating in the study.

4.2.1.2. Materials

The main purpose of this study was to examine whether one's genetic essentialist responses can be affected by framing genetic effects as a polygenic process, and whether people reason about monogenic and polygenic traits differently. To that end, this study consisted of three conditions: a) the same Genetic condition as in Study 6 ($n = 166$); b) a Polygenic-All condition in which Patrick's violent behaviour was described as having a polygenic component consisting of three different alleles, of which Patrick had all three ($n = 165$); and c) a Polygenic-One condition in which Patrick was described as only having one of the three different alleles that lead to violent behaviour ($n = 163$; see Appendix I). In particular, the effect size was equalized across all conditions, such that the allele in the Genetic condition, the full set of three alleles in the Polygenic-All condition, and the one out of three alleles in the Polygenic-One condition, all were described as being responsible for an 80% increase in the likelihood that Patrick would act aggressively. Participants were randomly assigned to read one of the three conditions before completing the same dependent variables used in Study 6.

4.2.2. Results

All regression results in this study were performed having dummy coded each of the conditions, with the Genetic condition being the reference group. In order to obtain the effect size of the Polygenic-All-Polygenic-One contrast, separate regression models were analyzed with the Polygenic-One condition as the reference group. Again, participant age, sex, and

political orientation were included as covariates, and all continuous variables were standardized.

4.2.2.1. Defense Claims

Across the three conditions, there were no differences in the perceived applicability of the insanity defense, $|\beta|$'s < 0.15. The perceived applicability of the diminished capacity defense was higher in the Polygenic-All condition ($M = 3.69$, $SE = 0.21$) than in the Genetic condition ($M = 3.22$, $SE = 0.17$), $\beta = 0.24$, $CI_{.95} = [0.02, 0.47]$. No other significant differences emerged, $|\beta|$'s < 0.15. The three conditions also did not differ in terms of the perceived applicability of the intoxication defense, $|\beta|$'s ≤ 0.01 (see Figure 25).
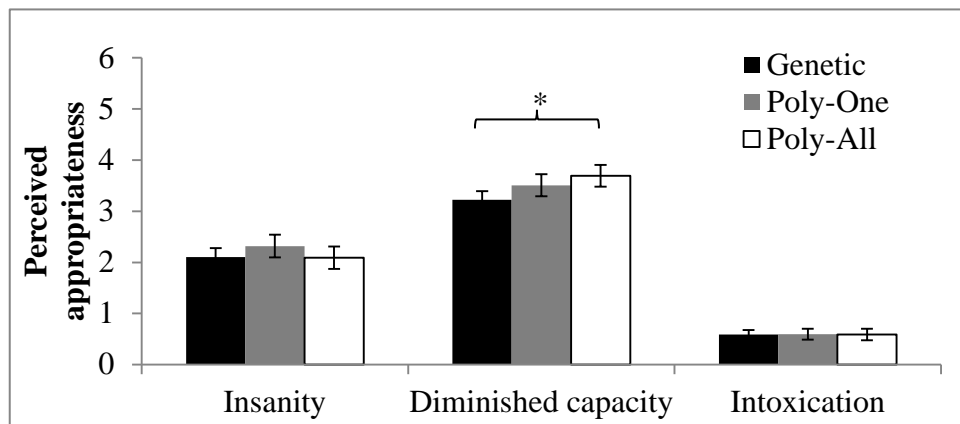
Figure 25. Perceived appropriateness of each defense claim by condition. Genetic = Genetic, Poly-One = Polygenic-One, Poly-All = Polygenic-All † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.2.2.2. Sentencing

No differences emerged across the different conditions, $|\beta|$'s < 0.20 (see Figure 26).

4.2.2.3. Perceptions of the Perpetrator

Perceived control was weaker in the Polygenic-All condition ($M = 4.53$, $SE = 0.14$) compared to the Genetic condition ($M = 4.83$, $SE = 0.12$), $\beta = -0.24$, $CI_{.95} = [-0.46, -0.02]$. The remaining contrasts were not significant, $|\beta|$'s < 0.20 (see Figure 27). There were also no

perceived differences between the different conditions in terms of perceived intent, criminal

responsibility, and expected recidivism, $|\beta|$'s < 0.20 (see Figure 28).
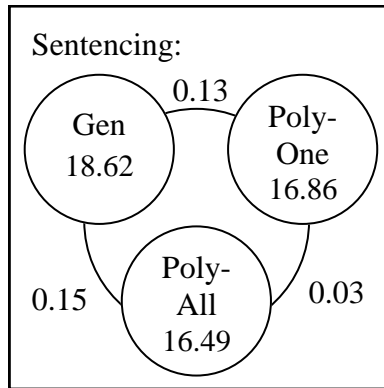
Figure 26. Adjusted means and absolute standardized effect sizes for comparing sentencing between condition. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$
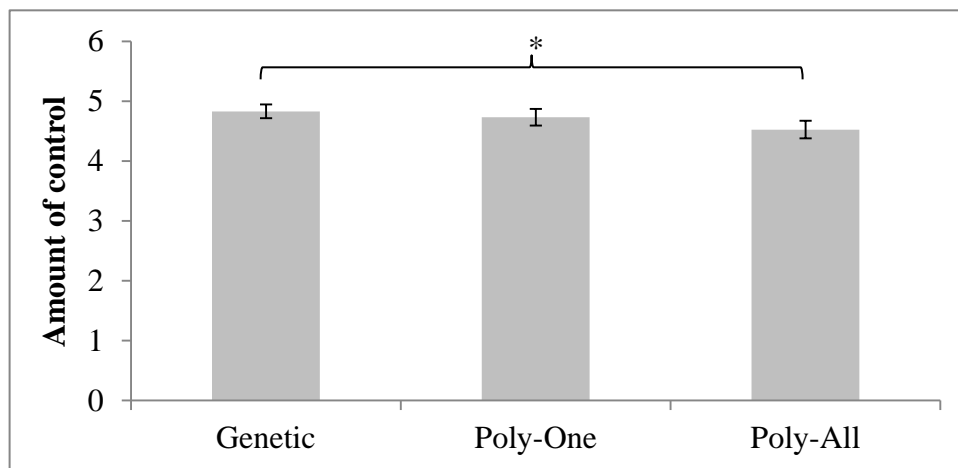
Figure 27. Different levels of perceived control by condition. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$
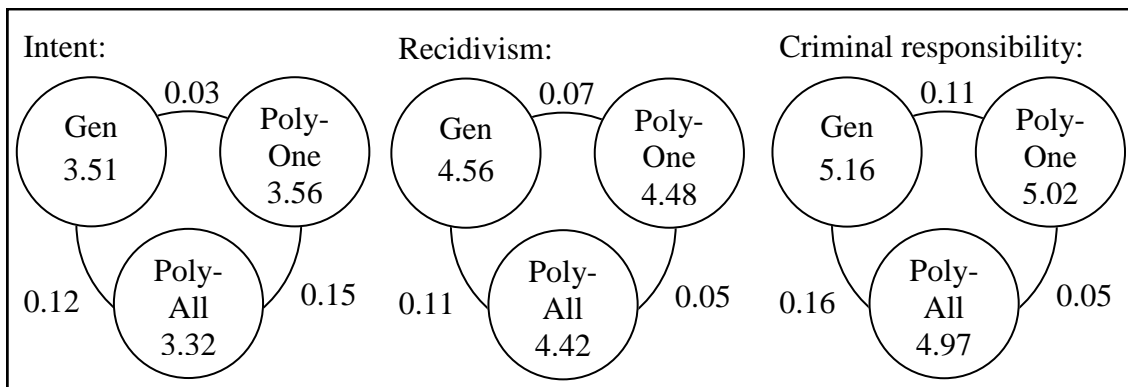
Figure 28. Adjusted means and absolute standardized effect sizes for comparisons of various perceptions of the perpetrator between conditions. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.2.2.4. ASQ

No significant contrasts emerged across the conditions, $|\beta|$'s < 0.20 (see Figure 29).
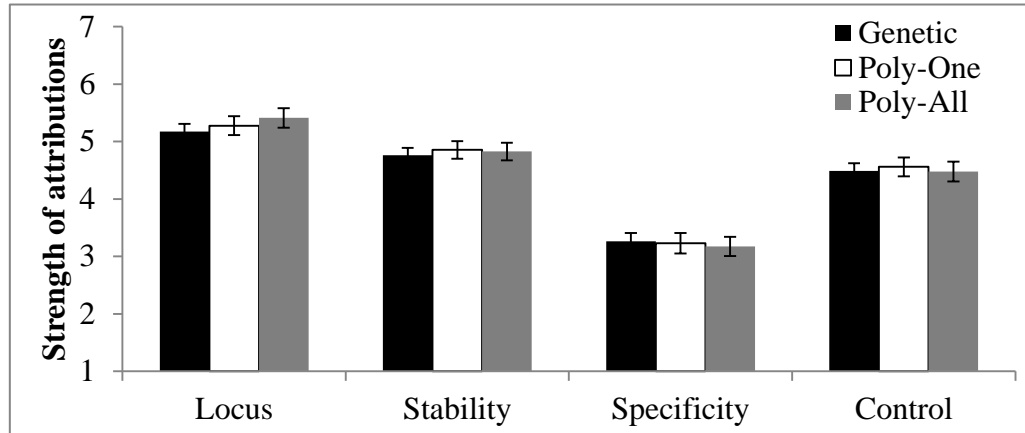
Figure 29. Causal attributions for different causal explanations. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.2.3. Discussion

Study 7 sought to determine whether people are sensitive to the nature of genetic involvement in the expression of a trait. In particular, participants learned that Patrick acted violently because he had a single genetic variant, one of three genetic variants, or all three required genetic variants. There were some instances in which having all three genetic variants exacerbated genetic essentialist biases, such that people felt that the diminished capacity defense was more appropriate, and perceived the perpetrator as having even less behavioural control, than the generic condition in which only one genetic variant was implicated. Beyond these specific patterns, however, people did not reason differently about Patrick's behaviour based on the nature of genetic effects. In general, people held similarly essentialist biases regardless of whether the perpetrator had one or many genetic variants. This suggests that when the only focal point of a causal explanation is on genetic factors, people's genetic essentialist biases become activated without being adjusted in response to

how many genes are implicated. This lack of sensitivity to the role of other genes means that efforts to downplay people's deterministic thoughts about genetic causes by introducing the role of other genes will likely be unsuccessful. In fact, given the pattern observed for perceived conscious control, certain versions of these explanations may exacerbate the issue. This exacerbation of genetic essentialist biases suggests that that with every additional genetic mutation that is involved, genetic essentialist biases also become incrementally stronger, leading people to ascribe even less behavioural control to the individual. Future research should examine whether or not one's genetic essentialist biases are sensitive to the amount of genetic mutations involved in a given expression. Nonetheless, this effect appears only in an extreme minority of cases, and stands against the backdrop of an overwhelming general lack of sensitivity to genetic complexity.

Another point of consideration is that the similar responses between the generic monogenic causal explanation (i.e. the original genetic explanation) and the more complex explanations may be further evidence that people have only vague conceptualizations about genes. That is, one interpretation of these results is that laypeople lack the understanding that genetic effects of varying degrees of complexity lead to different probabilistic relations between genes and trait expression. Instead, they may see genetic effects as operating in the same deterministic way, insensitive to the degree of genetic complexity. This supports Conrad's (1999) proposed "one gene one disease" perception that was briefly described in Chapter 1. In other words, as research has repeatedly demonstrated, people have a poor understanding of genetics (Lanie, et al., 2004; Moffitt, Caspi, & Rutter, 2006), which may contribute to the existence of genetic essentialism, and its insensitivity to different

probabilistic relationships. If there is to be any sensitivity, it seems to have more to do with the exacerbation of genetic essentialist biases than the mitigation thereof.

4.3. Study 8: Weak Genes, Strong Genes

The previous two studies examined different ways of mitigating genetic essentialist cognitions by portraying genetic effects in more complicated ways that more closely approximate how genes actually operate. In Study 8, the effect sizes of genetic causes were manipulated to determine whether people's genetic essentialist biases are sensitive to information about the strength of genetic causes. In other words, genetic alleles vary in terms of how much impact they have on the manifestation of a given trait. For instance, researchers have found that over 30 genetic alleles can significantly predict BMI, with some alleles being more predictive than others (Speliotes, et al., 2010). In particular, the FTO gene had the strongest effect size, accounting for 0.49% of individual variation in BMI, while KCTD15 accounted for less than 0.01% of that individual variation. Due to concerns about participants not being able to comprehend effect sizes in terms of the amount of variance explained, genetic effects were described as leading to varying levels of increase in the likelihood of the phenotype becoming manifest, as described below.

4.3.1. Methods

4.3.1.1. Participants

Study 8 involved 218 participants (49% males; $M_{age} = 34.26$, $SD = 11.22$) from MTurk. The sample consisted of 78% Euro-Americans, 8% African-Americans, 10% East Asians, and 4% Others. As with previous studies, participants indicated their political orientation using a 5-point scale (1 = Very liberal, 5 = Very conservative; $M = 2.45$, $SD = 1.00$). Participants received $1.25 for participating in the study.

4.3.1.2.   Materials

As with Studies 6 and 7, the experimental manipulation related to the same murder vignette. Because the purpose of this study was to determine whether people's genetic essentialist biases are sensitive to information about the strength of genetic effects, the experimental manipulation varied in terms of the increased likelihood of someone acting violently if they shared the same particular genetic variant as Patrick. Specifically, this study contained three conditions: a) the same Genetic condition as in Study 6, without presenting the effect size ($n = 72$); b) a Low condition that conveyed the Genetic condition as leading to a 25% increase in the likelihood of acting violently ($n = 71$); and c) a High condition that conveyed the Genetic condition as leading to a 400% increase in the likelihood of acting violently ($n = 71$; see Appendix J). Participants also responded to the same set of dependent variables as the ones used in Studies 6 and 7.

4.3.2.   Results

All regression results in this study were performed having dummy coded each of the conditions, with the Genetic condition being the reference group. In order to obtain the effect size of the Low-High contrast, separate regression models were analyzed with the Low condition as the reference group. Again, participant age, sex, and political orientation were included as covariates, and all continuous variables were standardized.

4.3.2.1.   Defense Claims

Across the three conditions, there were no differences in the perceived applicability of the insanity defense, $|\beta|$'s < 0.15. On the other hand, the perceived applicability of the diminished capacity defense was higher in the original genetic condition ($M = 3.84$, $SE = 0.25$) than the Low condition ($M = 3.08$, $SE = 0.31$), $\beta = -0.41$, CI$_{.95}$ = [-0.73, -0.06], but

similar to the High condition ($M = 3.80$, $SE = 0.30$), $\beta = -0.02$, $CI_{.95} = [-0.34, 0.30]$. The latter two conditions differed significantly from each other, $\beta = 0.39$, $CI_{.95} = [0.07, 0.73]$.

The intoxication defense was endorsed marginally more in the Low condition ($M = 0.69$, $SE = 0.20$) than the Genetic condition ($M = 0.36$, $SE = 0.12$), $p = .074$, $\beta = 0.30$, $CI_{.95} = [-0.05, 0.66]$, and significantly more than the High condition ($M = 0.30$, $SE = 0.15$), $\beta = -0.38$, $CI_{.95} = [-0.72, -0.02]$. The latter two conditions did not differ significantly from each other, $\beta = -0.06$, $CI_{.95} = [-0.32, 0.20]$. These results can be seen in Figure 30.
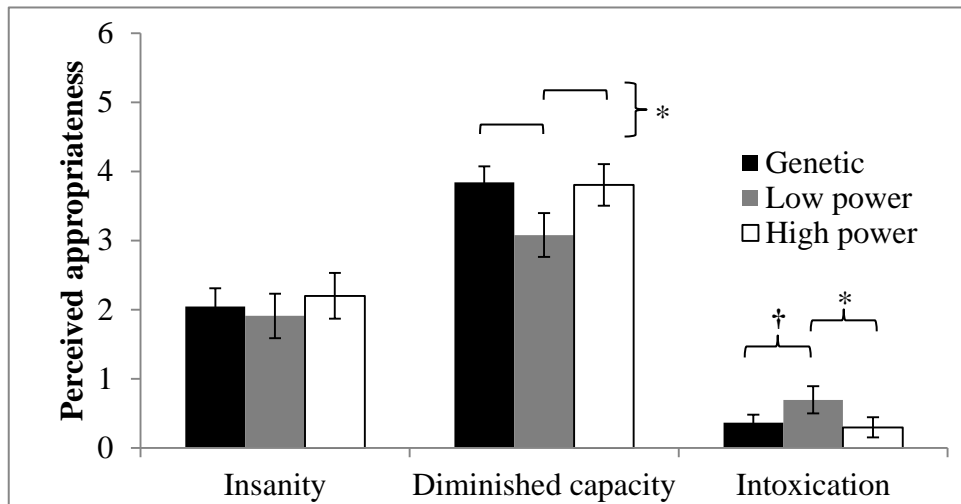
Figure 30. Perceived appropriateness of various defense claims by condition. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.3.2.2.   Sentencing

No differences emerged across the different conditions in terms of prescribed sentence length, $|\beta|$'s < 0.05. These results can be seen in Figure 31.

4.3.2.3.   Perceptions of the Perpetrator

Participants did not differ significantly from each other between conditions in terms of perceived conscious control, $|\beta|$'s < 0.25 (see Figure 32), perceived intention, $|\beta|$'s < 0.10, criminal responsibility, $|\beta|$'s < 0.15, and expected recidivism, $|\beta|$'s < 0.15 (see Figure 33).
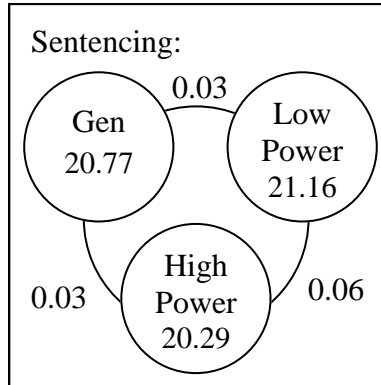
Figure 31. Adjusted means and absolute standardized effect sizes for comparisons of sentencing between conditions. † *p* < .10 * *p* < .05, ** *p* < .01, *** *p* < .001
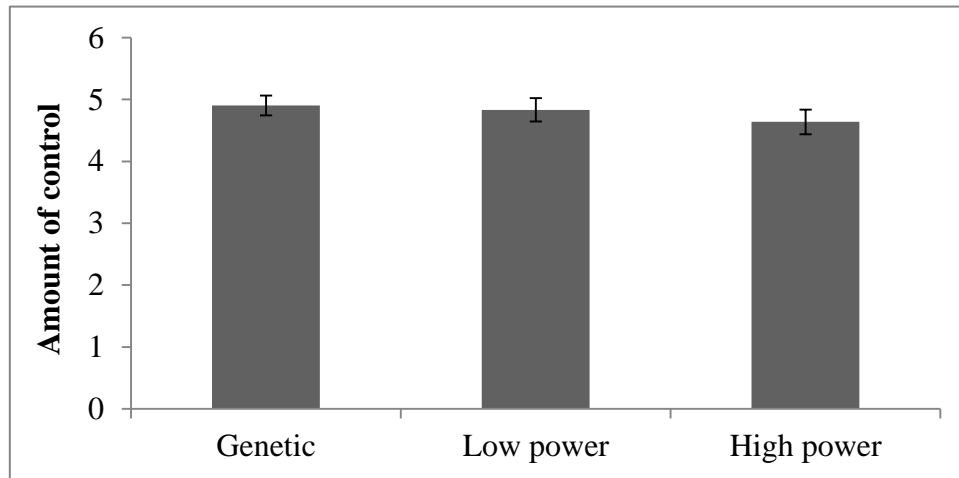
Figure 32. Amount of perceived control by condition. Error bars represent standard errors. † *p* < .10 * *p* < .05, ** *p* < .01, *** *p* < .001
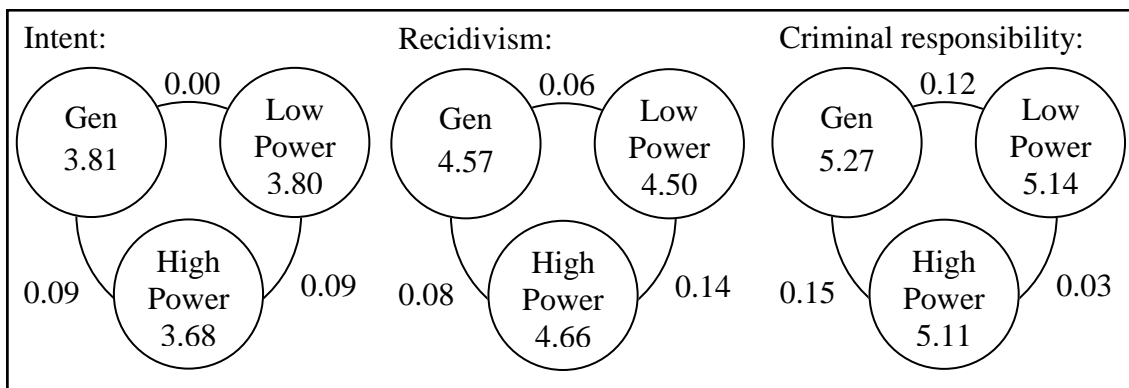
Figure 33. Adjusted means and absolute standardized effect sizes comparing various perceptions of the perpetrator between conditions. † *p* < .10 * *p* < .05, ** *p* < .01, *** *p* < .001

4.3.2.4.　ASQ

The conditions did not differ from each other in terms of causal locus, $|\beta|$'s < 0.15, and causal control, $|\beta|$'s < 0.25. On the other hand, the High condition was seen as being marginally more stable than the Genetic condition, $p = .075$, $\beta = 0.31$, $CI_{.95} = [0.01, 0.63]$, and significantly more stable than the Low condition, $\beta = 0.42$, $CI_{.95} = [0.09, 0.77]$. The Genetic and Low conditions did not differ from each other, $\beta = -0.12$, $CI_{.95} = [-0.45, 0.23]$. The High condition also led to marginally lower perceptions of specificity compared to the Low condition, $\beta = -0.30$, $CI_{.95} = [-0.66, 0.02]$. No other contrasts were significant, $|\beta|$'s < 0.20. All results for the ASQ can be seen in Figure 34.
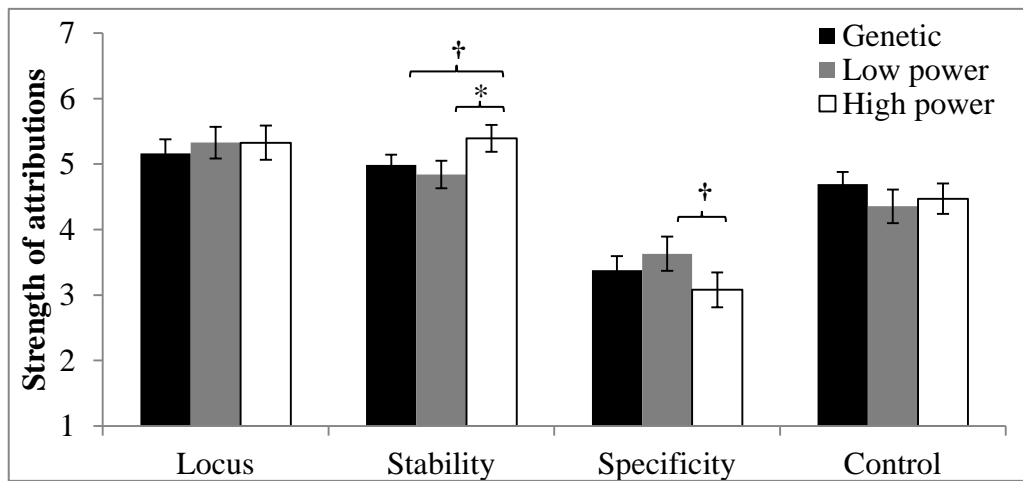
Figure 34. Causal attributions for various genetic causal explanations. Error bars represent standard errors. † $p < .10$ * $p < .05$, ** $p < .01$, *** $p < .001$

4.3.3.　Discussion

Study 8 examined the impact of another kind of genetic explanation on one's genetic essentialist biases. Specifically, people learned about genetic effects of varying effect sizes to determine the sensitivity of people's genetic essentialist biases to such information. Similar to the results of Studies 6 and 7, there was no consistent pattern in terms of the impact of effect size. For most of the dependent variables, responses across the three conditions did not

significantly differ from each other, particularly regarding people's perceptions of the perpetrator; however, the power of the genetic cause had an impact on the perceived appropriateness of different criminal defenses. Specifically, presenting genetic causes as being low in power led participants to endorse the diminished capacity defense much less, and to endorse the intoxication defense more, than presenting genetic causes as being high in power.

Some interesting findings also emerged in relation to people's causal attributional profile associated with each kind of genetic cause. While the regular genetic cause was seen as having similar causal attributions compared to the low-powered genetic cause, the high-powered genetic cause was seen as being more stable than both other genetic causes, while being *more* domain-general. The stable perception of powerful genes is in accordance with expectations based on earlier studies—if essentializing genes means that they will be seen as deterministic and immutable, then more powerful genes should also be seen as being even more stable. The domain-general perception of powerful genes, on the other hand, was unexpected. If genetic ascriptions lead to a certain attributional profile, then exposure to a strong genetic explanation should lead people to consider the same attributional profile, but to a greater extent. The obtained pattern, however, is the opposite of what had been expected. The adoption of a more domain-general causal attribution in this case suggests that when genes appear to be excessively powerful, people perceive that such genes can bleed into other domains. The rationale for such a perception is unclear. One potential explanation may be that when genes are seen as being so powerful, they are also seen as granting a general, stable identity that lies at the heart of many different phenotypes as opposed to a stable identity based on a specific phenotype. For instance, people perceive that food that has been

genetically modified to include "human genes" is wrong because that is akin to eating humans (Shaw, 2002). Similarly, people have also reasoned that mixing animal genes with plants appear to be particularly unsettling. By inserting a fish gene into corn, people perceived it as combining *a fish* with corn; and similar processes would entail having "a dog in [one's] peach or plum," or putting "a hog into [one's] corn" (Knight, 2009, p. 185). In these cases, people associated particular genes as representing whole biological kingdoms, such that even taking one gene from a dog meant inserting a general dog essence into another object. Thus, it may be that when genes are perceived as being particularly powerful, they can be seen as taking on greater portions of an individual's general identity. Future research should further investigate whether or not people over-ascribe the scope of a gene's impact if a gene is portrayed as being very powerful.

Overall, participants' responses were similar across the different conditions for most dependent variables, with people generally feeling that the power of genetic explanations lie somewhere between the low-power and high-power genetic causes by default. The simplest interpretation is that there did not appear to be strong evidence that people were consistently sensitive to the effect size of a genetic cause as there was no uniform pattern of statistical significance based on effect sizes. This, however, accounts for only parts of the results. The lack of statistical significance across various dependent variables, particularly in contrasting the low- versus high-power genes, may suggest a lack of sensitivity; but an alternate explanation may be that participants' sensitivity might be nonlinear. In other words, purported effect sizes may still impact people's cognitions; but people are sensitive to such effect sizes only below a certain threshold, such that results end up resembling a logarithmic curve. Below the threshold, people's responses may be sensitive to different effect sizes.

Above the threshold, *any* reference to genetic causes will activate one's essentialist biases. Different variables may have different thresholds, which may account for the variability in the patterns obtained from Study 8. One other interpretation of the data is that, rather than a nonlinear effect, there is actually a linear effect, and the effect sizes used in this study were too coarse to reveal this effect. Which relationship(s) most accurately characterize(s) the real state of affairs can be elucidated in the future by examining participants' responses as a function of a greater number of purported effect sizes, particularly small effect sizes that more closely mimic the effect sizes of currently-identified candidate genes (e.g. Hinney, et al., 2006; Schaefer, et al., 2009). It is important to note that these interpretations assume that participants understood a 25% increase in likelihood of engaging in violence was actually low-powered, and 400% increase was actually high-powered. It would be ideal in future research to incorporate a wider spectrum of effect sizes.

4.4.   General Discussion

Three studies attempted to determine whether framing genetic causes in different ways can impact the extent to which people will respond in accordance with genetic essentialism. Specifically, Study 6 complicated the role of a genetic cause by suggesting that it acts in conjunction with an environmental cause; Study 7 situated a genetic cause within the context of a larger set of genes; and Study 8 discussed a genetic cause as being either low-powered or high-powered. These portrayals of genetic causes were within the context of the criminal decision-making process first described in Study 1 because Studies 1 to 3 provided a set of robust findings to draw upon, which aided in determining what greater/less genetic essentialist responding should resemble. Furthermore, using the same context for all

three studies in this chapter allowed for the interpretation of commonalities and differences across the three studies.

Across Studies 6 to 8, several important findings emerged. The first finding is that manipulating and complicating the framing of genetic explanations does not have a uniform impact on people's responses, and is generally ineffective at mitigating genetic essentialist cognitions. In particular, Study 6 found that the effect of interactionist behavioural explanations resemble genetic explanations for some responses, and resemble environmental explanations for others, without any discernible patterns. Study 7 found that complicating genetic explanations by introducing the idea of polygenic effects also does not consistently impact responses such that genetic causes, regardless of their monogenic versus polygenic nature, yielded similar responses. The exceptions were two analyses revealing that perceiving someone having multiple genetic mutations may exacerbate genetic essentialist responses by engendering an even weaker ascription of behavioural control. Study 8 found that portraying a genetic mutation as being more versus less powerful also does not consistently affect how people think about someone who possesses such a gene. Some responses suggested low-powered genes might temper genetic essentialist biases by decreasing one's endorsement of the diminished capacity defense, and some responses suggested high-powered genes might exacerbate such biases by increasing one's perceived causal stability. On the other hand, the majority of responses did not yield significant differences across the different conditions. These results generally resemble those of Boysen and Gabreski's (2012), who found that complicating genetic accounts by emphasizing the interactionist nature of such effects did not yield consistent patterns of responding. In particular, the overwhelming pattern that can be seen across Studies 6 to 8 is that, with the exception of a small number of findings, the vast

majority of the results clearly demonstrate that genetic essentialist biases are insensitive to most contextual information or increased complexity of genetic behavioural explanations.

The second consistent finding was that endorsement of criminal defenses was the only dependent variable that was consistently affected by the framing of the genetic cause. This was an unexpected finding because, legally, the criminal defenses provided in these studies rely on constructs that did not yield significant differences across conditions, such as perceived behavioural control and intention. Perceived behavioural control was affected in two of the three studies in this Chapter, suggesting that it is still an important component of people's decisions about the applicability of criminal defenses; however, the fact that other perceptions of the perpetrator, such as intent, were not consistently affected, while endorsement of criminal defenses was, indicates two possibilities. One possibility is that behavioural control was the only matter of concern to participants in determining the applicability of different defense claims. Alternatively, another possibility is that there were additional considerations that concerned participants beyond the factors that the law deemed to be relevant. For example, participants may have considered implications of genetic explanations on one's neural development, which may also impact perceived criminal guilt (Appelbaum, 2009; Brown & Murphy, 2010). Indeed, neurological evidence has previously been shown to significantly impact the types of defense claims endorsed by mock jurors (Gurley & Marcus, 2008; but see Schweitzer, et al., 2011), with greater acceptance of neurological evidence being associated with greater endorsement of insanity defenses. This suggests that other considerations, such as possible perceived pathways by which genetic causes lead to behaviours (particularly neurological pathways), may play a role in legal decision-making. Based on these findings, future research should explore a broader set of

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perceptions that may be affected by genetic explanations in order to best determine appropriate predictors of the endorsement of defense claims.

Focusing on the contrasts that were significant yields another interesting finding in relation to people's default assumptions about genetic effects. Study 6 showed that the significant contrasts differentiated the interactionist account from both the genetic and the environmental explanations. In Study 7, the significant contrasts highlighted the consequences of having the complete set of multiple requisite genetic mutations. In contrast, having only one of multiple requisite genetic mutations was perceived similarly to simply learning that one has a genetic predisposition. Study 8 yielded a series of significant contrasts that indicated mitigated genetic essentialist biases when the genetic mutation is portrayed as being low in power, and exacerbated genetic essentialist biases when it is portrayed as being high in power. Taken together, these contrasts suggest that people's default assumptions about genetic effects do not generally involve the role of environmental factors, likely involve other genetic factors, and are moderately powerful. Understanding how people comprehend genetic effects is important for crafting effective methods of mitigating genetic essentialist biases. Therefore, future research should more definitively examine people's understanding of genetic causes. For instance, researchers should adopt qualitative methodologies such as interviews and written accounts (see Joseph & Guerra, 2015; Lewis, Leach, & Wood-Robinson, 2000; Monterosso et al., 2005). These can guide future formulations of questions to address how people understand genetic causes, particularly in relation to other behavioural causes.

5. **Chapter 5: Overall Discussion and Conclusions**

The purposes of this dissertation were threefold. First, it set out to investigate how genetic essentialism manifests in various domains. Two, it endeavoured to expand on the current genetic essentialism framework by incorporating people's causal attributions about genetic causes. Lastly, it sought ways to mitigate people's genetic essentialist biases.

5.1. **Consequences of Genetic Essentialism**

Overall, regardless of whether people are considering genetic causes for violence, metabolism, or learning styles, one robust consequence of genetic essentialism is that people perceive genes as entities that reduce one's ability to exercise control over their traits and behaviours, particularly in comparison to environmental causes. As an extension of this perspective, people also perceive traits that are caused by genes to be less changeable. This can have important implications. For instance, across all studies that involved comparing a genetic cause to an environmental cause for violence, people consistently found the defenses that mitigated *mens rea* to be more applicable when the perpetrator was portrayed as having had the genetic predisposition. In other words, these were the defense claims that argued for a lack of willful intent to commit the crime. This is important as these defenses reflect a perception that the perpetrator was unable to refrain from acting in contravention to the law. The results from these studies are in line with findings from previous research showing that reducing beliefs in free will leads to reduced tendency to engage in retributive punishment, or punishment with the intent to censure the perpetrator (Shariff, et al., 2014). Despite the fact that a significant difference in sentencing between genetic and environmental explanations only emerged in Study 3 and in the aggregate dataset, the general pattern is consistent across all studies – thinking that the perpetrator was genetically compelled to commit a crime led to

an overall shorter prescribed sentence than if he had environmental reasons. This was further supported by the path analyses from the aggregate dataset, which demonstrate that perceived control over one's behaviour is a key point of consideration in predicting the length of prescribed prison sentences for a perpetrator. These findings are in line with other studies who similarly found that genetic explanations generally lead to more lenient punishments (Aspinwall et al., 2012; Dar-Nimrod et al., 2011; Monterosso et al., 2005). In particular, the mediation and path analyses involving perceived behavioural control replicate Dar-Nimrod et al.'s (2011) results, suggesting that this is a robust and reliable effect. This justifies concerns that many have previously raised regarding the impact of the use of behavioural genetics in the courtroom. Numerous legal scholars had already expressed concerns that the presentation of behavioural genetics may lead to an expectation that one has little behavioural control, which is problematic given the preponderance of free will and volition assumed by the criminal justice system (Coffey, 1993; Nelkin, 1992), particularly given how little we know about the role of specific genes in leading an individual to show particular traits.

Another interesting consequence of genetic essentialist thinking in relation to perceiving little conscious control over one's criminal behaviour is the double-edged perception of genetic causes. As many researchers have discussed (e.g. Aspinwall et al. 2012; Nelkin, 1992), the mitigating interpretation of genetic causes taking away one's conscious control is often accompanied by the aggravating interpretation that the perpetrator will continue to be a threat to society. Indeed, the present data suggest that genetic explanations of violence do lead to greater expectations of recidivism, which the path analyses also showed to be an important predictor of one's punitiveness. These perceptions of genetic causes of criminal responsibility might extend into future work examining the rationale

behind punishing a perpetrator, and the type of punishment that one might recommend. For instance, punishment can be meted out for several reasons: specific deterrence and rehabilitation (to deter the criminal from reoffending in the future); general deterrence (to deter others from committing similar crimes); incapacitation (ensuring that the perpetrator cannot harm the public again); and just deserts (punishment the offender regardless of concerns about reoffending or copycat crimes from others; Carlsmith, Darley, & Robinson, 2002; Pinker, 2011). Given the present evidence for the countervailing forces impacting sentencing, it may be worthwhile to examine whether people's endorsement of the above reasons for punishment are also affected. Perhaps just deserts, rehabilitation, and both forms of deterrence would become less appropriate reasons to exercise punishment given that the perpetrator ostensibly had no control over his actions, which would suggest that deterrence is ineffective. On the other hand, incapacitation would be perceived as being the most appropriate reason to consider given expectations that the perpetrator is likely to reoffend. It is unknown whether it is warranted to draw a connection between the perceptions of the perpetrator in Studies 1 to 3 and the above reasons for punishment. Future research should examine such links, and determine whether these links have implications for actual sentencing practises.

The perception of lessened conscious control also applies to people's conceptualization of metabolism, such that attributing metabolism to genetic causes, relative to being breastfed as a child, led to lower expectations that one could control one's own metabolism. One result that follows from this lower expectation of control is that people expected that someone with a genetic cause for fast metabolism would gain less weight over time. This fatalistic perception likely has some important implications, such as leading

people to eat more (Dar-Nimrod et al., 2014). This is problematic given that currently known "obesity genes" can explain only a very small amount of variability in one's weight (Speliotes, et al., 2010). Thus, increasing food consumption as a result of genetic ascriptions of obesity-related constructs can have detrimental effects on one's physical health. One particular concern regarding this issue is the potential for anyone to fall into the trap of engaging in a self-fulfilling prophecy (Merton, 1948). In other words, the belief that one has a gene related to obesity may lead one to eat more, thereby causing weight gain, eventually fulfilling the erroneous original expectation. Such concerns have previously been expressed within the context of mental health treatment (Phelan, Yang, & Cruz-Rojas, 2006), math performance (Dar-Nimrod & Heine, 2006), and as a general consequence of genetic etiological beliefs (Vineis, 2004). As such, it would be important to further explore other consequences of genetic essentialist biases in the realm of obesity.

Another important consequence to consider in terms of having strong genetic essentialist beliefs about obesity is that they may affect the perceived efficacy of different treatments of obesity. Taking the perspective of the theory of planned behavior (Azjen, 1991), one's perceived control over their weight strongly predicts one's intention to lose weight, as well as their actual weight loss over time (Godin, Valois, & Lepage, 1993; Palmeira, et al., 2007; Schifter & Azjen, 1985). The findings here regarding genetic causes for metabolism leading to less perceived control over one's weight suggests that people who learn about such explanations are less likely to attempt to lose weight. This may have implications for the form of treatment that one would attempt, such that genetic explanations of obesity would lead to greater preferences for treatments that signify less exercising of personal control (e.g. pills, surgery), while decreasing preferences for treatments that require greater personal

control (e.g. exercise, diet). Indeed, patients who were given a genetic diagnosis for high cholesterol – a condition that is often comorbid with obesity – felt that changing their diet would be less effective, and taking medication would be more effective, at reducing cholesterol than patients who were given a non-genetic diagnosis for high cholesterol (Marteau, et al., 2004). Furthermore, in anticipation of genetic essentialist tendencies even amongst physicians, some have argued that physicians may feel that treating obesity is a pointless endeavour given the monogenic mutations that have been associated with obesity (Hainer, Toplak, & Mitrakou, 2008). Future work should examine these cognitions simultaneously within the framework of the theory of planned behavior to better understand the health and behavioural consequences of attributing obesity to genetics, with a particular focus on the forms of treatment that people are willing to seek out. Moreover, it is also important to examine the impact of genetic explanations of obesity in terms of stigma and discrimination. With mounting evidence that biological explanations of obesity, such as a perceived genetic etiology, lead to lowered perceptions of self-efficacy in losing weight (e.g. Dar-Nimrod et al., 2014; Pearl & Lebowitz, 2014), this should lead to less blame and greater sympathy (Suhay & Jayaratne, 2013). While this pattern has been found for certain conditions such as smoking and anorexia nervosa (Bannatyne & Abel, 2015; Dar-Nimrod, Zuckerman, & Duberstein, 2014), the evidence has been inconsistent with regard to mental illness (Kvaale, Haslam, & Gottdiener, 2013; Phelan, 2005). Given that obesity-related stigma is a strong predictor for anxiety disorders, depression, and further exacerbation of one's obesity (Puhl & Heuer, 2010), it is important for researchers to better understand how the increasingly geneticized basis of obesity may contribute to subsequent stigma and discrimination.

Lastly, resembling the patterns seen in other domains, perceiving learning styles as being due to genetics also led to less perceptions of conscious control over what one's dominant learning style is, as well as the changeability of this learning style. This finding is important because of the consistent lack of empirical support for the concept of learning styles and the measures that examine such a concept (Coffield et al., 2004; Freedman & Stumpf, 1978; 1980). In particular, there is no scientific basis for the proposed argument that learning outcomes are optimal when learning styles match the teaching styles that are used to deliver the content. Yet, recent decades have seen a push for more inclusive learning environments that take into consideration supposed variability of learning styles amongst learners (Lage, Platt, & Treglia, 2000; Rassool & Rawaf, 2007), predicated on the presumption that learning styles are immutable. As a result, it is seen as being incumbent upon educators to tailor their teaching styles to students' learning styles. On the other hand, when learning styles are framed as being a malleable construct, it engenders a push for students to expand on their repertoire to encompass more learning styles (e.g. Smith, 2002). Some researchers have argued that a mismatch between learning and teaching styles can have serious consequences, including lack of interest in course material, and fatalism about one's own capabilities (Borg & Shapiro, 1996; Rassool & Rawaf, 2007). Furthermore, data from Study 5 also suggest that people expect academic performance to be poorer for situations in which there is a mismatch of styles; but if this problematic reticence to change one's learning style to adapt to different subjects and teaching styles is based on the perceived immutability of learning styles, then the present data suggest that perceiving learning styles as being genetic in etiology may contribute to, or exacerbate, this reticence. Just as in the case of obesity, this may result in a self-fulfilling prophecy, such that perceiving learning styles to

have a genetic etiology may lead students to perceive their learning styles as immutable, therefore expecting poor performance in courses in which the teaching styles do not match their individual learning styles. As a result, they may disengage from such courses, resulting in poor academic performance. This would then reify their belief in the importance of matching teaching styles with one's learning style. Given the important implications that students' belief in learning styles has on their learning outcomes, it is also important to better understand the consequences of perceiving learning styles to be genetic in etiology. While perceived etiology did not predict behavioural differences in terms of instructor recommendations in the present data, there may be other important variables that were not incorporated, including motivation factors such as engagement with the course (Becker, McCaleb, & Baker, 2015), and behavioural variables such as the amount of time spent studying (Darolia, 2014). Both of these have been associated with better academic performance, and can contribute to a better understanding of the impact that perceived etiology of learning styles can have.

5.2.   Causal Attributions of Genetic Causes

There has been little work examining the attributions that people have when considering genes as causal agents. Previous conceptualizations of genetic causal attributions have primarily focused on causal locus and controllability, positing that genetic causes should be seen as internal and uncontrollable (Rees, Ingledew, & Hardy, 2005). Across multiple studies, the causal attributions that people drew for genetic causes were consistent, spanning different domains. The results bore out predictions based on previous work, such that, relative to environmental causes, genetic causes are seen as more internal, and less controllable. In addition, genetic causes were also seen as being more domain-specific. The

one inconsistency regarding genetic causal attributions that emerged related to causal stability. While genes were seen as being more causally stable than the environment in relation to perceptions of criminal responsibility, the same result was not found within the context of metabolism and obesity. This inconsistency may have to do with the fact that the Attributional Style Questionnaire is scored on a continuum. As such, it would not be possible to make absolute judgments about whether a cause is "internal" versus "external"; instead, it is best to understand the nature of causal attributions in relative terms. Such relative judgments are sensitive to the particular comparisons being made. The fact that the environmental factors differed between the two domains examined here may have led to the inconsistency that emerged.

Understanding the causal attributional profile of genetic causes is important because it allows for the examination of relations between causal attributions and important outcomes. Some results in this dissertation have already conceptually replicated previous research findings. For instance, previous work had demonstrated that stronger internal attributions were associated with stronger punitive tendencies and less lenient parole decisions (Carroll & Payne, 1977; Tam et al., 2013). Mediation and path analyses found that genetic causes were seen as being more internal than environmental causes, leading to lengthier sentences in turn. With regard to obesity, previous work has found that the general public tends to perceive obesity and metabolism as being due to internal and uncontrollable causes (Ogden, et al., 2001). This type of cause has also been associated with greater intentions to avoid future dieting efforts in the future, and weaker expectations of future success in weight loss (Burnette, 2010). A focus on causal attributions involving genetic causes can guide future work to examine whether a perceived genetic cause for obesity leads to weaker motivations

to diet, and weaker expectations of success in weight loss efforts. More importantly, causal beliefs about body weight has been associated with actual changes in body weight over time, with greater increases in body weight amongst people who more strongly believe that body weight is due to genetic causes (Jeong, 2007; McVay, Steinberg, Askew, Kaphingst, & Bennett, 2015). Future work can investigate links between how genetic etiological beliefs affect dieting and eating behaviours, and determine whether such effects may be due to the causal attributions that people have for genes.

5.3. Mitigating Genetic Essentialism

Most of the aforementioned cognitions that people have with regard to genetic causes do not reflect how genes actually affect behaviour. In contrast to the highly probabilistic nature of how genes are understood to affect behaviour, the cognitions found in the present studies portray a conceptualization that is much more simplistic – people appear to believe that genes greatly diminish one's behavioural control, making all associated traits more immutable. This effect was consistent across several studies, and was applicable to aggression, metabolism, and learning styles. One remaining question that this dissertation sought to address was the possibility of mitigating these perceptions by manipulating the portrayal of genetic causes in ways that highlight the complex nature of genetic effects.

The three primary ways in which genetic causes were portrayed involved discussing an interactionist account that included both a genetic and an environmental cause; discussing genetic causes within the context of other relevant genetic mutations; and discussing genetic causes as being either low- or high-powered. Across all of these portrayals, there was no consistent pattern to suggest that genetic essentialist cognitions can be easily mitigated. The interactionist account appeared to be the most effective out of the different portrayals, such

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that it led to the greatest number of significant contrasts relative to the original genetic explanation, although these contrasts were still infrequent. Nonetheless, this suggests that mitigating genetic essentialist responses may require a disproportionately strong environmental counterpoint to rebut a simple genetic explanation. While no work has examined this hypothesis directly, there is emerging evidence that strongly highlighting external procedures (e.g. applying sunscreen) that can control the likelihood that underlying genetic risks will be expressed (e.g. melanoma) is effective in convincing people to engage in such preventative measures (Taber & Aspinwall, 2015). Short of changing the genetic sequence itself, it appears that the most effect way to mitigate genetic essentialist biases is by providing tangible ways to mechanically prevent the genes from expressing themselves. This may be a promising avenue of future research.

In contrast to using interactionist accounts, complicating the original genetic explanation by adding the role of other genetic mutations did little to mitigate genetic essentialist responses. In fact, suggesting that one has multiple genetic mutations for a certain trait exacerbated some responses such that people perceived even less behavioural control. Finally, portraying genetic causes as being low- or high-powered also did not mitigate genetic essentialist responses, suggesting that people do not appear to be responsive to such information about genetic causes. Additional work is required to determine people's sensitivity to the effect size of genetic effects. The finding that manipulating effect sizes did not lead to significant differences in comparison to the original genetic account may not indicate a lack of sensitivity. Instead, people may be sensitive to this information until the effect size reaches a certain threshold, beyond which effect sizes are seen as *effectively* equally strong despite being different in magnitude. The current design may be too coarse to

reveal this difference; but it does suggest the difficulties of mitigating genetic essentialist responses.

5.4. Future Directions

Based on the results obtained across the eight studies in this dissertation, there are numerous future directions for subsequent research. Some of these have already been discussed given that they followed directly from the relevant results. Other future directions can be divided into several broad themes, relating to prejudice and discrimination; support for eugenic ideas; comparing genes to other biological causes; and understanding one's own genetic predisposition.

5.4.1. Prejudice and Discrimination

One important genetic essentialist cognition that Dar-Nimrod and Heine (2011) originally proposed is the perception of homogeneity and discreteness. In other words, people who are seen as carrying the same genetic material are perceived as being fundamentally similar to each other; but they are also seen as being fundamentally different from people who do not share the same genetic material. This has important implications for intergroup interactions, particularly with regard to prejudice and discrimination, because categorical thinking is an integral part of prejudice (Allport, 1954; Quadflieg, Mason, & Macrae, 2010; Tajfel, 1978). It is, thus, reasonable to expect that perceiving clear and strong boundaries between in- and out-groups as a result of genetic essentialism would similarly lead people to have strong prejudices.

Race is one of the most highly essentialized categories, making people perceive strong boundaries between different racial groups (e.g. Cosmides, Tooby, & Kurzban, 2003;

Chao, Chen, Roisman, & Hong, 2007; Haslam, Rothschild, & Ernst, 2000; Jayaratne, et al.,

2006). The consequences of such essentialism of race have consistently been negative.

Greater racial essentialism has been associated with more prejudice against out-group

members (Haslam, Rothschild, & Ernst, 2002; Roets & Van Hiel, 2011), greater reliance on

stereotypes of outgroup members (Paulker, Ambady, & Apfelbaum, 2010), and less support

towards immigrants and asylum seekers (Pehrson, Brown, & Zagefka, 2009). Conversely, the

tendency for people to identify with all of humanity as one in-group is associated with

holding less prejudiced views and engaging in more prosocial behaviour with others (Buchan,

et al., 2011; McFarland, Webb, & Brown, 2012). All of these examples highlight the myriad

of ways in which essentialism can impact intergroup relations.

If people perceive genes as the underlying entity that gives rise to identities and group

membership, then perceiving genes to be the underpinning of race and racial differences

should also lead to greater levels of prejudice. More recent research has found that perceiving

race as being biologically determined is associated with less interest in interracial interactions,

and greater acceptance of existing racial inequities (Williams & Eberhardt, 2008). Perceiving

race as a socially constructed concept did not lead to the same results. More germane to

perceptions of genes, European Americans who felt that genes underlie racial differences

between them and African Americans scored higher on various measures of anti-Black

racism, and also had stronger endorsement of negative stereotypes against African Americans

(Sheldon, Jayaratne, & Petty, 2007). Furthermore, when people perceive racial groups as

being very different from each other due to a lack of genetic overlap, they more readily

categorize Black-dominant mixed-race faces as Black, and White-dominant mixed-race faces

as White (Plaks, Malahy, Sedlins, & Shoda, 2012), suggesting a more categorical perception

of different racial groups. Overall, these results strongly suggest that perceiving race as being based in genetics likely leads to the perception that different racial groups are highly discrete, leading to greater levels of prejudice.

Based on the extant research on genetic essentialism and prejudice, subsequent work can focus on whether emphasizing the social constructionist nature of race, or the immense genetic relatedness shared by geographical populations around the world (Smedley & Smedley, 2005), can decrease interracial prejudice and weaken people's endorsement of racial stereotypes. I am currently running a series of studies examining the extent to which people are willing to ascribe different racial stereotypes to genetic causes based on whether they learn that human populations share much of their genetic material, or that people who come from different geographical locations have distinct genetic profiles (Cheung & Heine, 2015). The data suggest that portraying humans as being genetically similar leads to a reduction in genetic ascriptions for different racial stereotypes, $\beta = -0.56$, $p < .001$, $CI_{.95} = [-0.89, -0.23]$. In terms of raw scores from the study, this translates to a 20% reduction in expected genetic contribution to racial stereotypes. Additional work can examine whether such portrayals can also impact people's willingness to endorse stereotypes, motivations to engage in interracial interactions, and desire for social distance from an interaction partner who appears to be from another race. Given that prejudice against other racial groups constitutes only one type of prejudice, it would also be of interest to determine whether such interventions can impact other forms of prejudice as well, such as gender discrimination and prejudice between social classes (Haslam et al., 2000; Kraus & Keltner, 2013).

5.4.2.   Support for Eugenics

As discussed previously, being prejudiced and engaging in discriminatory behaviour against others is a manifestation of one's tendency to mentally separate different groups (Allport, 1954; Tajfel, 1978). The eugenic ideas that were so pervasive amongst the scientific community in the early twentieth century were arguably the cruel manifestations of some very extreme forms of prejudice and discrimination. This era saw legalized and codified prejudice against "undesirable" members of society, including those with mental illnesses, people with low intelligence, criminals, and people with low socioeconomic statuses (Lynn, 2001). For example, it was legal in many countries to forcibly sterilize criminals and people who were mentally disabled or had mental illnesses, and to curtail benefits for women on welfare to discourage them from having children. Conversely, people who were considered to be desirable in terms of possessing certain traits, such as high intelligence and strong moral character, were financially incentivised to have children. Reproductive policies such as these were based on the assumption that these traits are all based on genetics, and that artificially manipulating the gene pool of a nation would lead to a nation of intelligent, moral, and successful citizens (Kevles, 1985). This perspective is particularly indicative of the genetic essentialist cognition that Dar-Nimrod and Heine (2011) referred to as specific etiology. That is, the prevailing perception was that genetic causes were the sole causal agent that determined traits such as one's intelligence and moral character. The reverse inference would also have been made, such that someone who was intelligent would have been assumed to possess the appropriate genes. In fact, couples who had a highly intelligent child were also incentivised to have additional children (Lynn, 2001).

While forced sterilization is no longer legal in most countries owing to its status as a crime against humanity by the Rome Statute of the International Criminal Court (United Nations General Assembly, 1998), advances in genetic biotechnology have led to procedures that some have considered to be a part of a new eugenics movement (Lynn, 2001). For example, prospective parents can select specific embryos for implantation during *in vitro* fertilisation based on an analysis of the embryonic genomes. More recently, scientists have successfully used a procedure called CRISPR to edit a small number of specifically targeted genes, leading to the prospects of widening the scope of this engineering process to include a larger set of genes (Hendel, et al., 2015). Many academics have argued that the increasing focus on genetic discoveries such as genetic associations of various health conditions is leading to a greater perception that genes lie at the heart of individual differences (Duster, 2003; Phelan, Link, & Feldman, 2013). Combining these ideas with the new genetic technologies that are available to scientists creates fertile grounds for a revival of eugenic ideas. This has become a particularly contentious issue with regard to CRISPR, with many perceiving it as signalling the return of eugenic agendas (Krishan, Kanchan, & Singh, in press; Pollack, 2015), calling for an international interdisciplinary conference to discuss the ethics of genetic modification.

Despite the emergent concerns about the reality of eugenic technology, there is currently no extant empirical literature on causal factors that affect support for eugenic technology and policies; however, if the increase in genetic discoveries is leading to a greater perception of the role that genes play in various traits, it may lead to greater genetic essentialism and support for genetic modification procedures that appear to directly address those traits. I am currently involved in several projects examining whether learning about the

genetic factors underlying intelligence, relative to environmental factors, affect one's support

for eugenic policies, such as sterilization and tax incentives for certain segments of society to

have children. The current results suggest that the genetic discussion leads to greater support

for eugenic policies, $\beta = -0.17$, $p < .05$, $CI_{.95} = [-0.35, 0.01]$, potentially validating concerns

by academics that genetic essentialism may be a backdoor to eugenics (Phelan, Link, &

Feldman, 2013; Ream, Cheung, & Heine, 2015). Additional work is needed to replicate these

findings and better understand the consequences of supporting such eugenic policies.

5.4.3.   Genetic Causes vs. Other Biological Causes

Many of the studies in this dissertation involved a comparison between genetic causes

and variations of environmental causes to determine the impact of genetic essentialism;

however, the gene is not the only example of specific essentialism. As discussed previously,

genetic essentialism is representative of a more general tendency for people to essentialize

categories in their world, and this general tendency can be instantiated within different

entities. While the gene has served as a very appealing and popular essence, other biological

entities may also be candidates for essences. One prominent alternative to the gene is the

brain, leading to neurological essentialism (Corrigan & Watson, 2004). Rather than

suggesting that traits such as intelligence are genetic, the basis of such traits and mental

conditions are seen as being due to differences in the brain. O'Connor, Rees, and Joffe (2012)

elucidated several ways in which the public understands neuroscience, and their discussion

demonstrates significant overlap with how people understand genetics. For instance, the

authors discuss the general perception that the brain is seen as an index of difference, the

entity that underlies differences between people who belong to different categories –

resembling the homogeneity and discreteness cognition discussed previously. Similarly, the

brain is seen as the ultimate basis for a particular trait, which can normalize behaviours that may otherwise be considered pathological or abnormal. In other words, when people think about neurological causes of behaviour, they also engage in the genetic essentialist cognitions of specific etiology and the naturalistic fallacy. These points clearly suggest that people view genetics and the brain in fundamentally similar ways. This relationship has been of particular concern within the criminal justice system because, similar to anticipated results of genetic evidence, many have expressed concerns about how neurological evidence may be used to unduly mitigate defendants' criminal responsibility (Appelbaum, 2009; Batts, 2009). Indeed, previous work have shown that explaining criminal behaviour as being due to hormonal levels in the brain or brain lesions as evidenced by neurological images led to more lenient perceptions of the perpetrator (Gurley & Marcus, 2008; Monterosso et al., 2005; Steinberg, 2013).

Both genes and the brain constitute different forms of biological essentialism (Haslam, 2011; Lebowitz, Ahn, & Nolen-Hoeksema, 2013; O'Connor & Joffe, 2013), and people tend to perceive both genetics and neuroscience to be similarly valid explanations of human behaviour (van Staden, 2003). Following this line of reasoning, some studies have directly compared the impact of neurological versus genetic explanations, generally finding no differences. This suggests that people essentialize the brain and genes in similar ways. For example, Appelbaum et al. (2015) found that portraying a criminal's behaviour as being genetic versus neurological in nature led people to perceive the perpetrator in similar ways. Extending beyond this domain, biological explanations of obesity (e.g. genetics, neuroscience) tend to be similarly associated with less perceived self-efficacy over, and changeability of, one's body weight, as well as less self-blame (Pearl & Lebowitz, 2014).

With the growing use of both neurological and genetic evidence in criminal trials, particularly to support claims of insanity or diminished capacity (Pyun, 2015), there is also an increasing need to better understand the biased ways in which both kinds of evidence can impact people's ways of thinking.

5.4.4.   Understanding Our Own Genes

The majority of the discussion in this dissertation relates to variables within an interpersonal context such as perceiving someone else's criminal responsibility or expected weight gain over time; but intrapersonal constructs are important to consider as well. With the immense popularity and affordability of direct-to-consumer (DTC) genetic testing services such as 23andMe (McGuire, Diaz, Wang, & Hilsenbeck, 2009), it is imperative that researchers better understand the impact of learning about one's own genetic predispositions within the context of people's genetic essentialist cognitions. Indeed, many academics have expressed concerns over the usage of DTC tests, often questioning whether the general public is able to rationally interpret the genetic results appropriately (Hauskeller, 2011; Hudson, Javitt, Burke, & Byers, 2007; Wright & MacArthur, 2012).

As my colleagues and I have highlighted elsewhere (Cheung, Dar-Nimrod, & Gonsalkorale, 2014), one important application of the genetic essentialist framework within this area is the fact that people may reason fatalistically about their own genetic predispositions just as they do when engaging in interpersonal judgments. Previous research has demonstrated that when people thought about having a genetic risk factor for various health conditions, they consistently perceived such conditions to be less preventable than attributing them to an unspecified cause (Senior, Marteau, & Weinman, 2000). Similarly, learning that one was genetically predisposed to develop alcoholism led to the perception that

one's own drinking behaviour was not under one's control, ultimately leading to greater negative affect and less positive affect (Dar-Nimrod, Zuckerman, & Duberstein, 2013). These demonstrate that the perception of weakened behavioural control applies to both interpersonal judgments and to intrapersonal perceptions, and may impact one's emotional well-being as well.

Perceiving that one has little control over one's own health conditions has immense behavioural implications. On the one hand, it appears that people engage in more medical screening behaviours when they learn that they are genetically susceptible to a certain condition. For instance, people who were provided with a positive test result for a genetic predisposition for melanoma engaged in more regular skin examinations than people who were given a negative test result (Aspinwall, Taber, Leaf, Kohlmann, & Leachman, 2013b). Similarly, women who learned that they were genetically predisposed to developing breast and ovarian cancers were much more likely to get prophylactic mastectomies and oophorectomies (Lynch, et al., 2006). These appear to support the expectations of certain proponents of DTC genetic tests that such tests would bring health benefits to users (e.g. Collins, Green, Guttmacher, & Guyer, 2003). On the other hand, using such tests may also perpetuate the medicalization of medical conditions, and shift responsibilities away from individual effort. Many studies have shown that learning about one's own genetic predispositions do not generally change one's engagement in preventative behaviours (Audrain, et al., 1997). Despite people being told that they were genetically at risk for lung cancer, it did not compel them to cease smoking (Lerman, et al., 1997). In a similar vein, being told that one was genetically predisposed to having high cholesterol did not increase dieting or exercising behaviours (Marteau et al., 2004). Particularly concerning is the finding

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that one's belief in the genetic etiology of one's own obesity was associated with less reported physical activity levels and consumption of fruits and vegetables (Wang & Coups, 2010). Taken together with the increased willingness to engage in screening behaviours, these studies suggest that genetic predispositions lead people to have a perception of weakened control over the development of their conditions. As such, they are not likely to engage in effortful, continuous behaviours that serve as preventative measures to protect against developing such conditions. Instead, they more readily rely on medical surveillance procedures to detect when such conditions will surely manifest, and prophylactic surgeries to stave off anxieties about eventually developing the condition. Furthermore, people also view this weakened control to extend into future generations, such that knowledge of one's genetic at-risk status compels people to decide against having children (Meiser, et al., 2007), demonstrating the strong fatalistic expectations that people have about genetic causes.

The DTC genetic testing industry was partly predicated upon the optimistic assumption that people are able to use this new, individualized genetic information to make better, more informed medical decisions (Helgason & Stefánsson, 2010). The evidence profiled throughout this dissertation suggests that such an expectation may not be realistic, and the current evidence is equivocal at best. On a more optimistic note, some of the negative effects of personal genetic tests discussed here have not been demonstrated when test results were discussed with genetic counsellors (Aspinwall, Taber, Leaf, Kohlmann, & Leachman, 2013a), highlighting the importance of discussing one's test results with medical professionals to appropriately interpret the data. Understanding how genetic counsellors discuss such results with clients may also inform future work on ways to mitigate genetic essentialism. More generally, subsequent research should further examine the psychological

and behavioural effects of DTC genetic tests as that will allow for a better understanding of variables that will lead to a greater likelihood of engaging in preventative measures and adherence to screening procedures.

5.5.   Limitations

There are several issues that limit the conclusions that can be made from the data presented in this dissertation. The first relates to the nature of the studies on the legal decision making process, which took up a bulk of the studies in this dissertation. In particular, the procedure asked participants to individually make determinations regarding the perpetrator's level of guilt. This, however, is not representative of how criminal trials are run in reality. Instead, these decisions are made within the group setting of jury deliberations, subject to a variety of group processes (Saks, 1977; Sommers, 2006). For example, shifting of opinions and group polarization have been found in simulated jury studies, such that sentences that were decided as a group tended to differ significantly from those that were decided privately (Izzett & Leginski, 1974). Furthermore, groupthink has also been implicated in jury deliberations, with a recognition that very specific and methodical procedures are necessary to stave off the impactful influence of the pressures inherent in jury deliberations (Neck & Moorhead, 1992). Both of these processes also suggest the importance of considering the influence that people with high status have within such group settings, such that their private perceptions of genetic and environmental explanations of aggression may come to greatly influence the decisions of others (e.g. Cheng, Tracy, Foulsham, Kingstone, & Joseph, 2013). The studies on legal decision-making in this dissertation did not take any of these processes into consideration, and should be included in future research.

Another limitation is with concerns about the external validity of the results. This is particularly important given the potential cultural differences that may exist regarding one's tendency to engage in genetic essentialist biases. The majority of the samples in the dissertation studies are European Americans, and there is much evidence showing that people from this cultural background are more likely than East Asians to focus on, and refer to, one's dispositions in explaining one's behaviour (Choi, Nisbett, & Norenzayan, 1999). European Americans are also more likely to be entity theorists, perceiving that people's traits are stable and unchangeable (Heine, et al., 2001). Hence, despite the fact that essentialism is a universal phenomenon (e.g. Gil-White, 2002), there are reasons to expect cultural differences in the extent to which one would exhibit genetic essentialism, leading to concerns about the generalizability of the results described in this dissertation. Examining cultural differences may reveal two potential outcomes. The first likely finding is that quasi-experimental studies should reveal East Asian participants to engage in less genetic essentialism in general, which can be measured using various scales that examine genetic essentialist cognitions. Some examples of such measures include the Belief in Genetic Determinism scale (Keller, 2005), and the Genetic Essentialist Tendencies questionnaire that I am currently helping to develop (Dar-Nimrod, Ruby, Cheung, Tam, & Murray, 2014). The second likely finding is that the explicit discussion of genetic causes would increase the salience of new, genetic information to East Asian participants, leading them to be particularly affected by such information. In accordance with this expectation, Phelan (2005) found that ascribing mental illness to genetic causes has a particularly pronounced effect on Asian American participants, leading to even less blame and anger towards someone with mental health issues compared to European American participants. These hypotheses should

be examined in the future to determine the generalizability of genetic essentialism and the impact of genetic ascriptions.

At a statistical level, all of the studies in this dissertation rely on the use of single-item questions as dependent variables. As such, there may be concerns about the stability of results, leading to issues regarding reliability (see Wanous & Reichers, 1996). While this may be an issue, many single-item questions have been used to replace lengthier scales while retaining good psychometric properties, such as attitudes towards specific product brands (Bergskvist & Rossiter, 2007) and self-esteem (Robins, Hendin, & Trzesniewski, 2001). Furthermore, the single-item questions that were used across multiple studies have mostly replicated (both significant and non-significant results), and the results have been generally consistent with theoretical expectations. These findings suggest that the single-item questions in these studies meaningfully measured the intended constructs.

Finally, it is important to consider that the vignette used in the legal decision-making studies involved a very extreme case in which the perpetrator demonstrated an immense level of violence and aggression against the victim. The relatively rare and extreme nature of that vignette may have rendered the legal decision-making questions was partially responsible for the genetic essentialist cognitions that participants exhibited. More mundane crimes such as petty theft or break-in and entering could have led to different responses. I have data from a recent study addressing this issue, showing that the seriousness of a crime does not appear to lead to significantly different cognitions. Specifically, this newer study examined the impact of genetic versus environmental behavioural explanations for a serious crime (i.e. murder) versus a less serious crime (i.e. an assault), reflecting a two-factor research design. In general, the effect sizes and directions of the contrasts between the genetic and environmental

conditions in this study were consistent with the results that were profiled above in Studies 1-3, and interactions involving the seriousness of the crime and different behavioural explanations were generally not significant (Byun, Cheung, & Heine, 2015). For example, one of the most consistent findings across the original legal decision-making studies was that a genetic explanation led people to perceive the perpetrator as having had less behavioural control compared to an environmental explanation, $\beta = 0.63$. In this newer study, the effect size of the same contrast between genetic and environmental conditions for perceived behavioural control was comparable, $\beta = 0.67$, $CI_{.95} = [0.39, 0.95]$. The interaction involving this contrast and the seriousness of the crime was not significant, $\beta = 0.34$, $CI_{.95} = [-0.05, 0.74]$. These results suggest that genetic essentialist biases are likely unaffected by the seriousness of the crime, although this interpretation can be bolstered by further examining different types of crimes of varying degrees of seriousness.

5.6.   Conclusions

An important factor that affects how people make interpersonal judgments is the causal explanation that they have of another's behaviour or traits. Genetic explanations are one such type of causal factor. For a variety of reasons, people tend to think about genes in unrealistically simplistic terms by assuming that they are the primary determinants of behaviour, and that their effects are immutable. This leads people to believe that behaviours and traits that are caused by genetics exist outside the realm of volition, and that there is little that one can do to prevent such behaviours and traits from manifesting. Across eight studies, this dissertation found that these perceptions apply to various domains, and have implications for important outcomes such as sentencing within a legal context, and eating behaviours when obesity is discussed. Furthermore, the studies determined that people draw specific

causal attributions. In particular, based on Weiner's theory of attribution, genetic causes are seen as being more internal, more domain-specific, and less controllable compared to environmental causes, and potentially more causally stable. Finally, data from several studies found that genetic essentialist biases are difficult to mitigate. Complicating the contribution of individual genetic factors by discussing the role of other genes or manipulating the effect size of genetic effects did not appear to affect how people reasoned about genetic causes, suggesting that people generally do not reason deeply about how genes affect behaviours. There is some evidence that emphasizing the role that environmental factors play may be a fruitful direction to explore in the future, but additional work is needed in this regard.

In general, genetics are seen as powerful entities that seemingly have the ability to give rise to behaviours over which one has little control. With the large number of gene-association studies being covered in the media, it is imperative that researchers understand how people interpret genetic information because it would have immense implications for a variety of fields of study including psychology, sociology, political science, public policy, and public health. Given the current understanding of people's reasoning regarding genes, we can now better interpret Donald Trump's quote from the beginning. In particular, it appears that he was making the inference that it is only natural for him to be successful because his genes had determined his success in life, fundamentally separating him from people who are not successful. In other words, because of his genes, he was born to be, and forever will be, a winner, unlike all the other losers.

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Appendix A

Study 1: Vignette common to participants in all conditions:

<u>Case briefing</u>

Defendant information

File No.: DA213-1206

Name: Patrick ███████

Birth date: 08/11/1990

Age: 21

Gender: Male

　　　Patrick ███████████ is a 21-year-old male who was attending Woffordson University as a 3rd-year student. At approximately 5:35 pm on December 14, 2010, Mr. ███████████'s neighbour reportedly saw him leave his house to take his regular walk in Virginia Park two blocks away. At approximately 5:40 pm, a witness living across the street to the north of the park, Mrs. ██████████, reportedly saw Mr. ███████████ arrive at the park as usual. His demeanour appeared to be the same as on any other day. There was no one else at the park at the time. According to Mrs. ███████████'s statement to the police, approximately ten minutes into Mr. ███████████'s walk, the victim, Mr. Donald ██████, arrived at Virginia Park, and walked onto the sidewalk to the east of the park. The tipster saw the victim walking with his head down, and appeared to be having trouble with what seemed like an iPod in his hand. About three minutes later, Mrs. ██████████ reportedly saw the victim, trying to fix his iPod, bump into Mr. ███████████ after their paths crossed, which led to a verbal argument between them. When the two began shoving each other, Mrs. ██████████ called the police. This was confirmed at the 9-1-1 call centre, with a call from Mrs. ██████████ being logged at 5:54 pm. Mr. ███████████ then allegedly pulled out a pocket knife and stabbed the victim multiple times. According to the coroner's report, the victim received three stab wounds to the lower abdomen, one stab wound to the chest, as well as two cuts on both of his hands. A police unit that was 8 blocks away responded to Mrs. ██████████'s call and arrived at 5:57 pm, and arrived within minutes. Mr. ███████████ was immediately taken into police custody.

Genetic condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in murder trials, the medical examiner took samples of Mr. ████████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ████████ has a genetic mutation known as SSHL-L. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, this SSHL-L mutation affects the concentration of important neurotransmitters in the brain. This has been associated with increased impulsivity, aggression, and violent tendencies. Recent studies estimate that the existence of this mutation leads to a 4-fold increase in the likelihood of responding to a provocation with violence. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ████████. The evidence is under review by the Crown prosecutors.

Environmental condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in murder trials, a court-appointed social worker created an in-depth profile of Mr. ████████'s life. The social worker discovered that Mr. ████████ had lived in adverse environmental conditions throughout his life. He grew up in poverty, was harshly beaten as a child by his single mother, and lived in a gang-infested neighbourhood. While he never joined any gangs, he had, and still has, many friends with gang associations. According to research done by criminologists Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, the above factors have all been associated with increased impulsivity, aggression, and violent tendencies. Recent studies estimate that these factors lead to a 4-fold increase in the likelihood of responding to a provocation with violence. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ████████. The evidence is under review by the Crown prosecutors.

Control condition:

No additional information was provided to participants.

Appendix B

The basic vignette seen by all participants is the same as what is available in Appendix A.

Genetic condition:

The information was the same as the genetic condition from Appendix A.

Gene-Impulse condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in trials, the medical examiner took samples of Mr. ███████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ██████ has a genetic mutation known as SSHL-L. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, this SSHL-L mutation affects the concentration of important neurotransmitters in the brain. This has been associated with increased impulsivity, aggression, and violent tendencies. Recent studies estimate that the existence of this mutation leads to an 80% increase in the likelihood of responding to a provocation with violence. This relationship is a result of an increased impulse to behave aggressively as a result of this gene. The SSHL-L mutation targets and strengthens inherent aggressive impulses so that the individual is impelled to respond with violence. Therefore, while all individuals have similar impulses to react with violence, this genetic mutation intensifies these urges. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ██████. The evidence is under review by the Crown prosecutors.

Gene-Impulse Control condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in trials, the medical examiner took samples of Mr. ███████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ██████ has a genetic mutation known as SSHL-L. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, this SSHL-L mutation affects the concentration of important neurotransmitters in the brain. This has been associated with increased impulsivity, aggression, and violent

tendencies. Recent studies estimate that the existence of this mutation leads to an 80% increase in the likelihood of responding to a provocation with violence. This relationship is a result of an individual's lack of restraint over aggressive behaviour as a result of this gene. The SSHL-L mutation reduces one's ability to exercise control over inherent aggressive impulses, thus leading individuals to respond with violence. Therefore, while all individuals have similar impulses to react with violence, this genetic mutation diminishes the control an individual has over them. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ███████. The evidence is under review by the Crown prosecutors.

Appendix C

Labels and definitions of legal terms provided to participants:

Defense claims:

- *Insanity defense*: The killing of a human being where a mental disorder or defect rendered the accused incapable of (a) distinguishing between right and wrong during the killing; OR (b) acting in accordance with the law; OR (c) understanding the nature/consequences of his/her actions
- *Diminished capacity defense*: The killing of a human being, where general malice existed (i.e. intention to do wrong), but the accused had a mental illness or defect that hindered his/her ability to (a) premeditate and plan the killing; OR (b) have the intention to kill; OR (c) know that his/her conduct could result in another's death
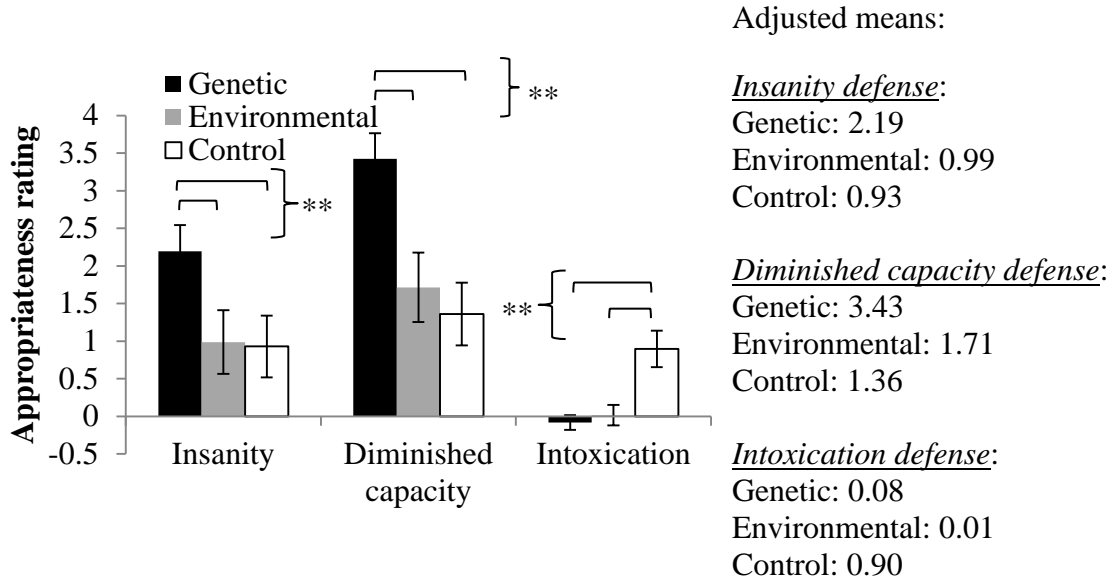- *Intoxication*: The killing of a human being, where general malice existed (i.e. intention to do wrong), but the accused was intoxicated, which hindered his/her ability to (a) premeditate and plan the killing; OR (b) have the intention to kill; OR (c) know that his/her conduct could result in another's death

Verdicts:

- *First degree murder*: The killing of a human being, where the killing is "planned and deliberate"; or the killing is done, deliberately or accidentally, during the commission of a felony (e.g. aggravated assault, arson, burglary, rape, robbery, etc.)
- *Second degree murder*: The killing of a human being, where the killing is not "planned and deliberate," often occurring at the "spur of the moment." There was an intent to kill, which occurred just prior to the "spur of the moment"
- *Manslaughter*: The killing of a human being, where the killing is not "planned and deliberate," and there is no intention to kill. This usually occurs in the heat of passion, OR out of recklessness and negligence
- *Not guilty*: There is (a) **insufficient evidence** that the defendant killed the victim; OR (b) **sufficient evidence** that the defendant did not have the intention and forethought to kill the victim, and did not have knowledge that his/her actions could lead to one's death
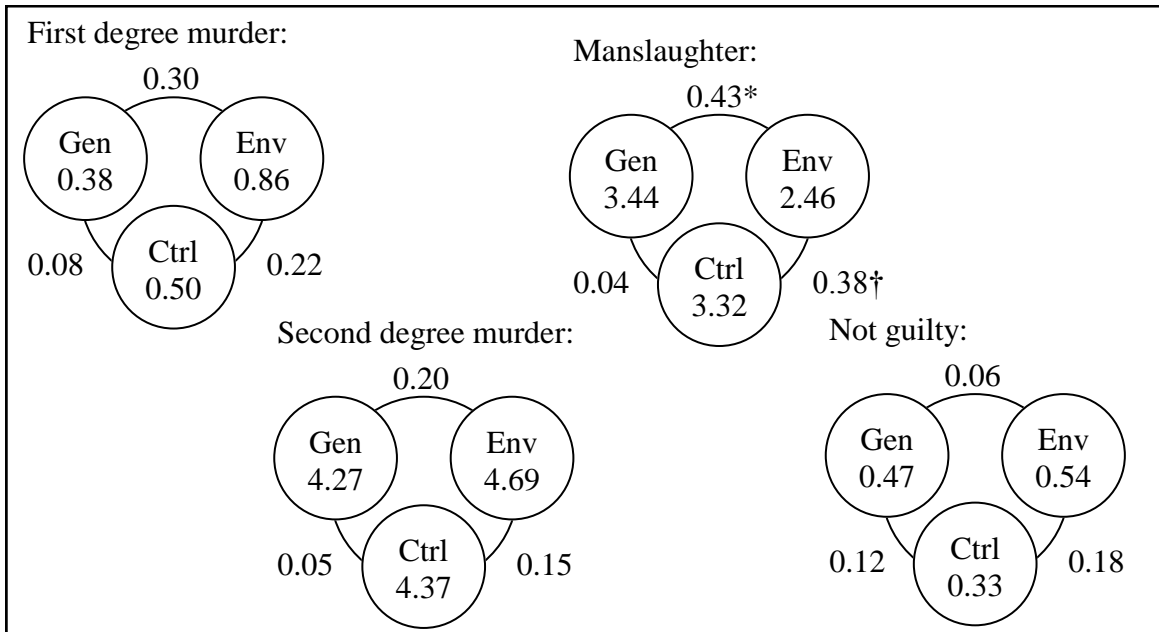
Appendix D

Study 1 results: All error bars on graphs represent standard errors; † *p* < .10 * *p* < .05, ** *p* < .01, *** *p* < .001
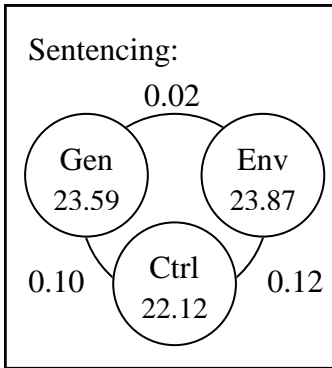
Appropriateness of defense claims:

Adjusted means:

Insanity defense:
Genetic: 2.19
Environmental: 0.99
Control: 0.93

Diminished capacity defense:
Genetic: 3.43
Environmental: 1.71
Control: 1.36

Intoxication defense:
Genetic: 0.08
Environmental: 0.01
Control: 0.90
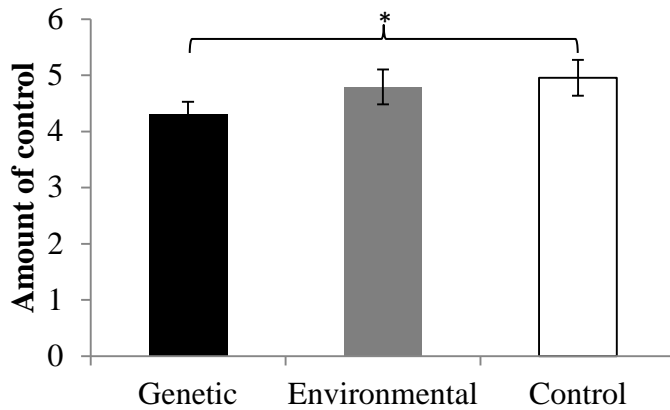
Appropriateness of verdicts:

Adjusted means and absolute effect sizes comparing different verdicts between conditions. Circled numbers represent adjusted means for each condition. Numbers between circles are the absolute standardized effect sizes from comparing between adjusted means. Gen = Genetic, Env = Environmental, Ctrl = Control.

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Sentencing:

Sentencing:

0.02

Gen
23.59

Env
23.87

0.10

Ctrl
22.12

0.12

Adjusted means and absolute effect sizes for sentencing. Circled numbers represent adjusted means for each condition. Numbers between circles are the absolute standardized effect sizes from comparing between adjusted means. Gen = Genetic, Env = Environmental, Ctrl = Control.
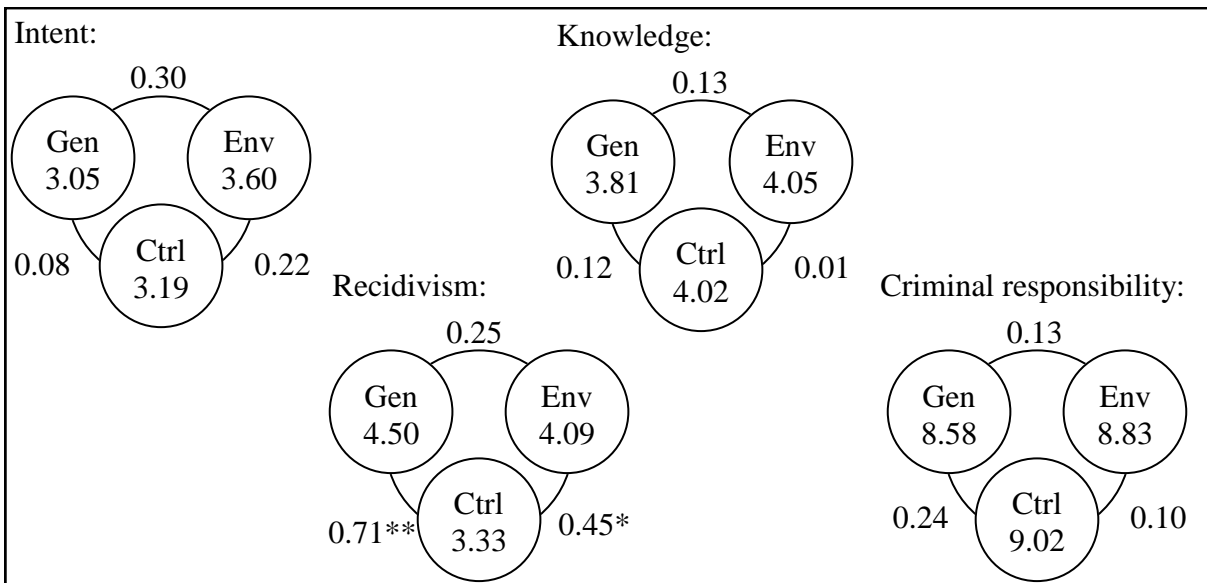
Perceived conscious control:

Adjusted means:

Genetic: 4.31
Environmental: 4.79
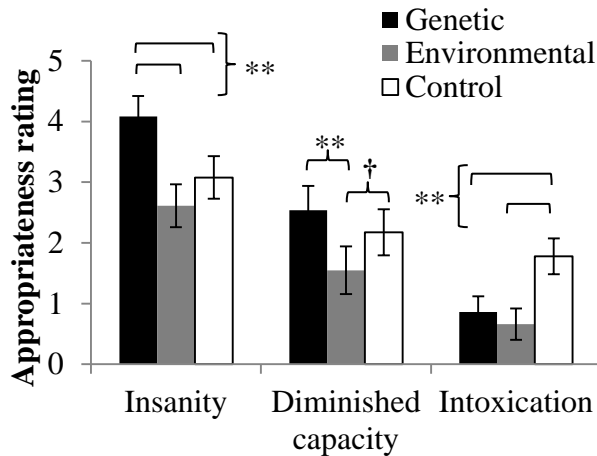Control: 4.96

Other perceptions of the perpetrator:

Intent:

0.30

Gen
3.05

Env
3.60

0.08

Ctrl
3.19

0.22

Knowledge:

0.13

Gen
3.81

Env
4.05

0.12

Ctrl
4.02

0.01

Recidivism:

0.25

Gen
4.50

Env
4.09

0.71**

Ctrl
3.33

0.45*

Criminal responsibility:

0.13

Gen
8.58

Env
8.83

0.24

Ctrl
9.02

0.10

Adjusted means and absolute effect sizes for various perceptions of the perpetrator.

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Appendix E

Appropriateness of defense claims:

Adjusted means:

Insanity defense:
Genetic: 4.08
Environmental: 2.61
Control: 3.08

Diminished capacity defense:
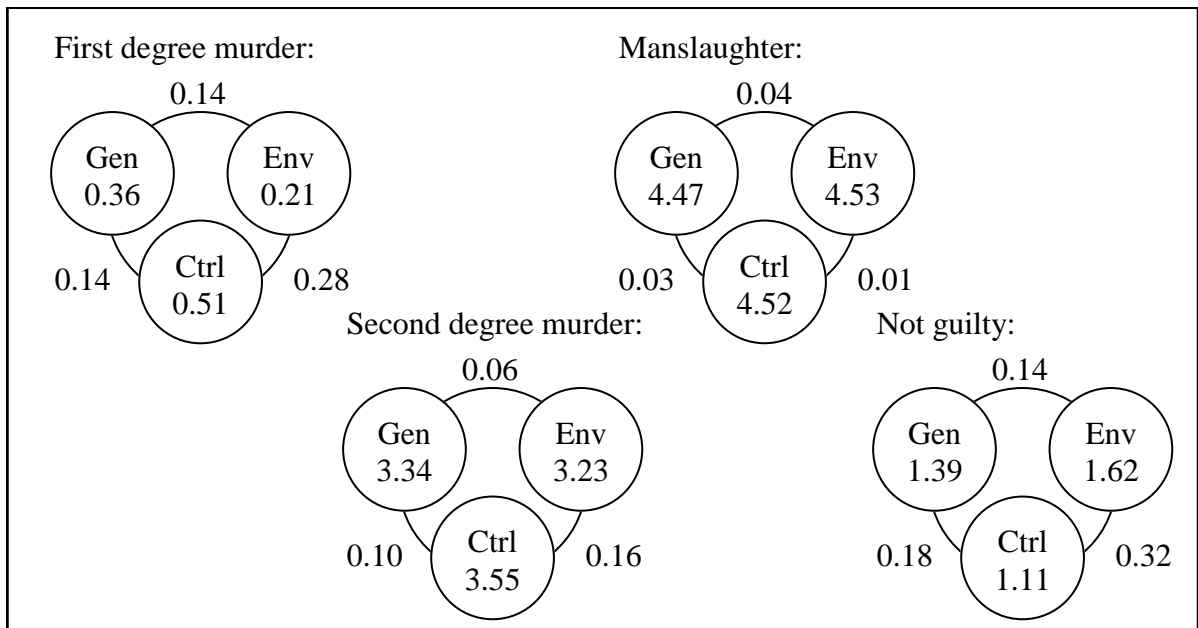Genetic: 2.54
Environmental: 1.55
Control: 2.17

*Intoxication defense*:
Genetic: 0.86
Environmental: 0.66
Control: 1.78
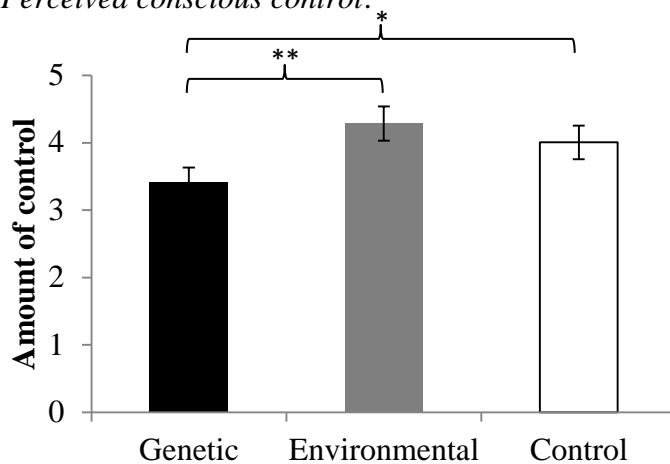
Appropriateness of verdicts:

Adjusted means and absolute effect sizes comparing different verdicts between conditions. Circled numbers represent adjusted means for each condition. Numbers between circles are the absolute standardized effect sizes from comparing between adjusted means. Gen = Genetic, Env = Environmental, Ctrl = Control.

Sentencing:
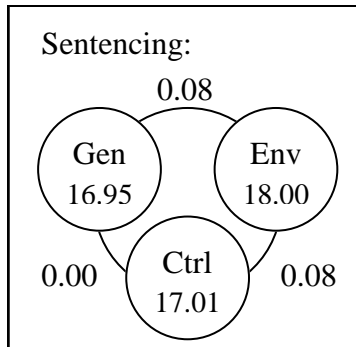
Adjusted means and absolute effect sizes for sentencing. Circled numbers represent adjusted means for each condition. Numbers between circles are the absolute standardized effect sizes from comparing between adjusted means. Gen = Genetic, Env = Environmental, Ctrl = Control.

Perceived conscious control:

Adjusted means:

Genetic: 3.42
Environmental: 4.29
Control: 4.01

Perceptions of the perpetrator:

Adjusted means and absolute effect sizes for various perceptions of the perpetrator. Circled numbers represent adjusted means for each condition. Numbers between circles are the absolute standardized effect sizes from comparing between adjusted means. Gen = Genetic, Env = Environmental, Ctrl = Control.
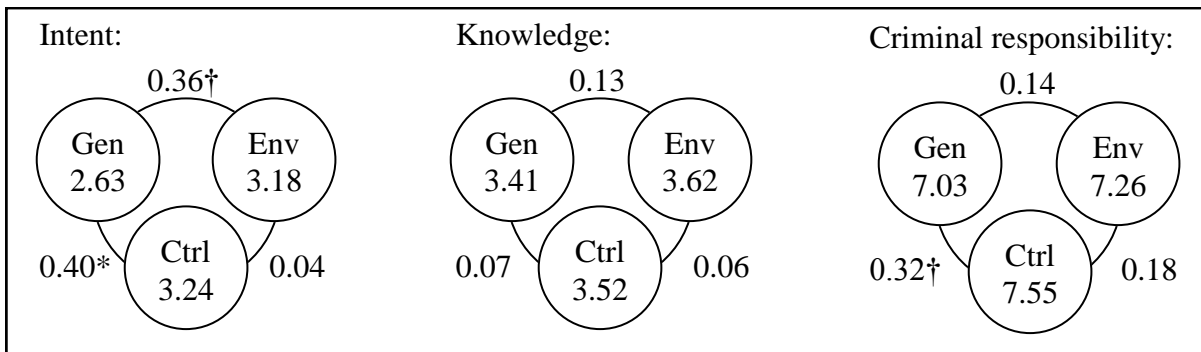
Appendix F

<u>Vignette common to participants in all conditions</u>:

25-year-old Jeremy Cooper is training to be a chef at his local Culinary Arts Institute. One of the policies of the school is that students must be educated on the importance of food and physical health as part of their curriculum. As part of their unit on metabolism, they must learn about what metabolism is, and what causes rates of metabolism to be faster or slower.

<u>Genetic condition</u>:

As part of this learning process, it is the goal of the administration for each student to know their own metabolism so that they are more aware of the food that they create, and the ingredients that they will use. The administration hopes to achieve this by discussing a cause of variability in metabolic rates, borrowing from a new trend in the culinary field – molecular gastronomy, which is the study of how to make use of physical and chemical reactions in cooking. The exception is that, for this unit, the administration wants to shift the focus from the food to the students, focusing on the biochemical interactions between our bodies and the food we eat. This will be accomplished by taking advantage of the cheap genetic testing technology that has emerged on many genetic testing websites. After each student obtains their result, the instructor will depict the distribution of the relevant gene amongst the students. The administration hopes that this will allow students to have a broader appreciation of the variability in metabolism in the population, as shown by the distribution of the gene.

Jeremy's results show that he does, in fact, have the KLF15 gene, a gene that has been associated with faster metabolism, especially of fats, according to a recent journal

article by researchers at the Case Western University (Haldar et al., 2012). In other words, this gene is associated with the ability to more quickly burn off calories from fats, with strong implications for keeping off excess weight and a lower prevalence of cardiovascular disease.

Environmental condition:

As part of this learning process, it is the goal of the administration for each student to know their own metabolism so that they are more aware of the food that they create, and the ingredients that they will use. The administration hopes to achieve this by discussing a cause of variability in metabolic rates. They brought in Dr. Guy Putet, a professor of Pediatrics from the French Paediatric Society. Speaking about a journal article that he published in 2011, he lectured the students about the key role that being breastfed as an infant plays in affecting metabolic rates in adulthood. He suggests that there is something about the nurturing that occurs during nursing of an infant that sets the initial conditions for developing his or her metabolism. Specifically, being breastfed is associated with higher metabolism rates than being bottle-fed. In other words, being breastfed is associated with the ability to more quickly burn off calories from fats, with strong implications for keeping off excess weight and a lower prevalence of cardiovascular disease. Dr. Putet encouraged each student to consider their own upbringing, and depicted the distribution of the frequency of breastfeeding amongst students during infancy. The administration hopes that this will allow students to have a broader appreciation of the variability in metabolism in the population, as shown by the distribution of other students' experiences.

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Jeremy considered his own upbringing, and brought up the fact that he was breastfed as a child.

Control condition:

As part of this learning process, it is the goal of the administration for each student to know their own metabolism so that they are more aware of the food that they create, and the ingredients that they will use. The administration just wanted them to be aware of the variability in metabolism rates in the people that they will eventually have to cook for. Thus, the students should be wary of the types of ingredients they use – not everyone has the high metabolic rate to facilitate burning away of calories from fats quickly.

Jeremy thought about his own metabolism and, through chatting with his other classmates, decided that he may have relatively higher metabolic rate compared to others, but he isn't quite sure. He understands, though, that he still needs to be aware of others' dietary needs.
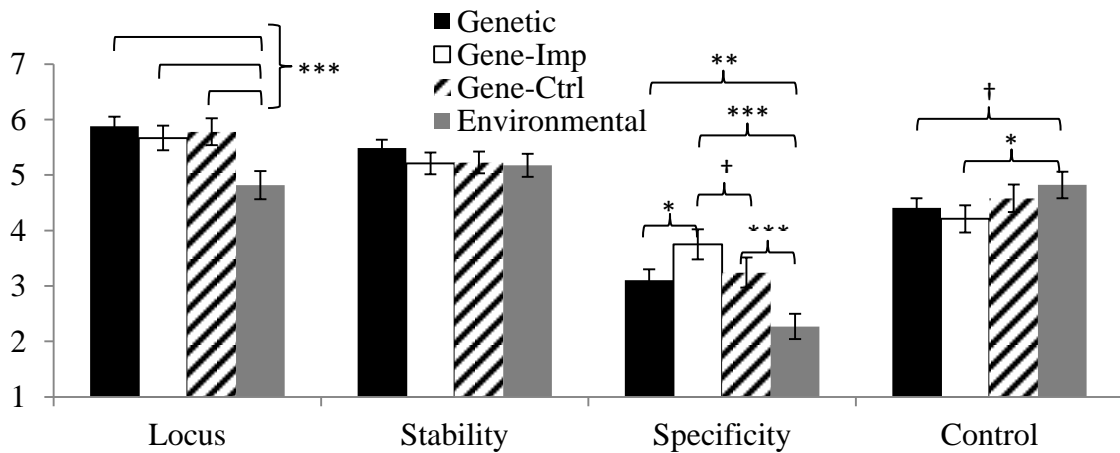
Appendix G

Study 2's causal attributional profiles. All error bars on graphs represent standard errors; † *p < .10 * p < .05, ** p < .01, *** p < .001*
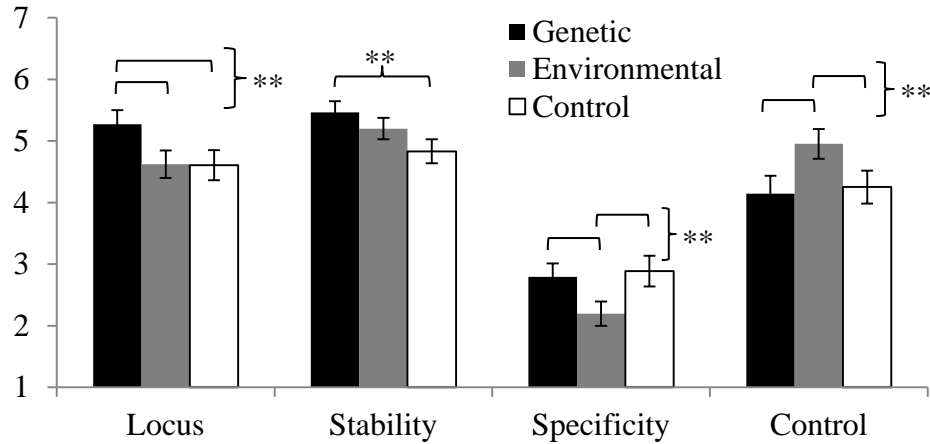
	Causal locus	Causal stability	Causal specificity	Causal control
Genetic	5.27	5.46	2.79	4.14
Environmental	4.62	5.20	2.20	4.95
Control	4.61	4.83	2.89	4.25

Study 3's causal attributional profiles. All error bars on graphs represent standard errors; † *p < .10 * p < .05, ** p < .01, *** p < .001*

	Causal locus	Causal stability	Causal specificity	Causal control
Genetic	5.88	5.49	3.10	4.41
Gene-Impulse	5.67	5.17	3.75	4.21
Gene-Control	5.78	5.22	3.24	4.58
Environmental	4.82	5.17	2.27	4.82

Appendix H

The basic vignette seen by all participants is the same as what is available in Appendix A.

Genetic condition:

The information was the same as the genetic condition from Appendix A.

Environmental condition:

The information was the same as the genetic condition from Appendix A.

Interactionist condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in murder trials, the medical examiner submitted samples of Mr. ███████'s blood to two independent labs for genetic testing, and arranged to have a court-appointed social worker to create an in-depth profile of Mr. [redacted]'s life. Both genetic labs confirm that Mr. ███████ has a genetic mutation known as SSHL-Ls. Moreover, the court-appointed social worker discovered that Mr. ███████ had lived in adverse environmental conditions throughout his life. He grew up in poverty, was harshly beaten as a child by his single mother, and lived in a gang-infested neighbourhood. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, they estimate that when both adverse environmental factors and the SSHL-L mutation are present, the interaction between them leads to an 80% increase in the likelihood of responding to a provocation with violence. Neither the genetic mutation, nor the environmental factors alone are sufficient in increasing this likelihood to this degree. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ███████. The evidence is under review by the Crown prosecutors.

Appendix I

The basic vignette seen by all participants is the same as what is available in Appendix A.

Genetic condition:

The information was the same as the genetic condition from Appendix A.

Polygenic-One condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in trials, the medical examiner took samples of Mr. ████████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ████████ has an important mutation on a gene known as SSHL-L. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, mutations on three genes (SSHL-L; MDHA3; and R1N4) work together to affect the concentration of important neurotransmitters in the brain. These mutations, together, have been associated with increased impulsivity, aggression, and violent tendencies. Out of these three mutations, people with the SSHL-L mutation alone have an 80% increase in the likelihood of responding to a provocation with violence, and Mr. ████████ has the SSHL-L mutation (but not the other two mutations). The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ████████. The evidence is under review by the Crown prosecutors.

Polygenic-All condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in trials, the medical examiner took samples of Mr. ████████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ████████ has important genetic mutations on three genes: SSHL-L; MDHA3; and R1N4. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, these mutations together affect the concentration of important neurotransmitters in the brain. These mutations, together, have an increased impulsivity, aggression, and violent tendencies, making them 80% more likely to respond to a provocation with violence. The defence intends

to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ███████. The evidence is under review by the Crown prosecutors.

Appendix J

The basic vignette seen by all participants is the same as what is available in Appendix A.

Genetic condition:

The information was the same as the genetic condition from Appendix A, with one difference between the removal of information about the effect size of the genetic mutation.

Low power condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in trials, the medical examiner took samples of Mr. ██████████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ██████████ has a genetic mutation known as SSHL-L. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, people with this mutation are 15% more likely to have increased impulsivity, aggression, and violent tendencies, making them more likely to respond to a provocation with violence. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ██████████. The evidence is under review by the Crown prosecutors.

High power condition:

As part of the District Attorney's office's new initiative to collect and retain more relevant evidence in trials, the medical examiner took samples of Mr. ██████████'s blood, and submitted them to two independent labs for genetic testing. Both labs confirm that Mr. ██████████ has a genetic mutation known as SSHL-L. According to research pioneered by Dr. William Bernet and Dr. Stephen Montgomery at Vanderbilt University, people with this mutation are 4 times more likely to have increased impulsivity, aggression, and violent tendencies, making them more likely to respond to a provocation with violence. The defence intends to summon Dr. William Bernet and Dr. Stephen Montgomery as expert witnesses to testify for Mr. ██████████. The evidence is under review by the Crown prosecutors.