Not all Explanations are Equal: Only Explanations Invoking a Change from the true self Mitigate Responsibility

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Abstract

When do people believe that biological explanations, such as genes, brain damage, or abnormal hormones, mitigate punishment for crimes? We propose the way in which biology is viewed as impacting the *true self* of the actor—who the actor really is, deep down—is the key element for predicting biologically-based mitigation. Across four preregistered studies, 4,066 American adults learned of different biological explanations for crimes and judged punishment, fault, and blame. We show, while some biological explanations mitigate punishment and responsibility, general ascriptions to genes do not. Participants viewed external events like traumatic brain injury as a disconnect between the *true self* and the peripheral self, whereas they did not for genetic ascriptions. People interpret general genes-based explanations for crimes as indicating that the perpetrators *true self* was violent or dangerous, while other biological explanations did not impugn the actor's *true self*. Only when genetic bases are described as mutations do genes become mitigating factors. In these latter cases, the actor is not judged as harshly because the violent crime could be attributed to aspects peripheral to the *true self*. Therefore, biological explanations differ in the extent to which they implicate the *true self*. These results help to clear up when biological explanations do and do not appear to mitigate responsibility for crimes: When the aspect of biology in question is viewed as part of the *true self*, it will not mitigate blame; but when the biological aspect is viewed as more peripheral, the blame seems to be mitigating.

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When someone commits a crime, we tend to hold them responsible for that crime. All of our behavior, including criminal behavior, has biological causes. These causes can include mechanical capability (e.g. physical movement), brain and neuronal structure and function, neurochemical activity, hormonal balances, genetic propensity. Drawing attention to such underlying biological causes has been argued to mitigate punishment for crimes. In a study of sitting U.S. judges, simply providing a biological mechanism (low MAOA activity, atypical amygdala function, etc.) for an assailant who repeatedly pistol-whipped a victim (*Mobley vs. The State of Georgia*, 1995) led to shorter sentences (Aspinwall et al., 2012). Furthermore, lawyers are more and more often invoking biology as a defense (Denno, 2011), with anecdotal evidence that the judges are reducing the severity of punishment for crimes such as murder when biology used as a defense (Ferro, 2009; see also Tabb et al., 2018).

Other studies have shown, however, that biological explanations do not mitigate punishment or responsibility. For example, both Cheung & Heine, (2015) and Kim et al., (2015) found the punishment that participants suggested for someone who stabbed and killed another man during a fight was not affected by whether they were told the perpetrator had a biological predisposition to engage in antisocial behavior. Similarly, Scurich & Applebaum (2015) observed that learning a perpetrator had a biologically based propensity towards violence did not affect participants' assignment of punishment for assault with a baseball bat (see also Costa et al., 2017). Finally, Berryessa, (2017) found that participants were unswayed in their opinion that a convicted murderer should remain on death row after learning that there was a biological basis for his mood and anger imbalances.

This disconnect across studies may come from an over-broad lumping-together of 'biological explanations'(see Fuss et al., 2015; Cheung & Heine, 2015; Heine et al., 2017; see

also Turkheimer, 1998; Landen & Innala, 2002; Boysen & Vogel, 2007; Lebowitz, & Ahn, 2014) or ‘biological causes’ (see Morse, 2007; Dar-Nimrod & Heine, 2011a). Explanations invoking different aspects of biology may instead have disparate effects. Genetic explanations, for example, may affect punishment and responsibility differentially from other sorts of explanations. Previous research has not, however, specifically tested different types of ‘biological explanations’ against one another. In this manuscript, we explore whether there is a systematic reason for the inconsistent reductions of responsibility outside of simple sampling error across studies.

One factor that may moderate the mitigating impact of biological explanations is the degree to which such explanations pertain to the *true* versus *peripheral self*. *True self*-theory proposes that people believe individuals have a true, core, deep-down self as well as a more peripheral self (see Frankfurt, 1971; see also Sheldon et al., 1997). Furthermore, this *true self*, without information to the contrary, is assumed to be *good* (e.g., Newman, Bloom, & Knobe, 2014). This splitting of judgments of the self into core and peripheral components helps explain numerous phenomena in moral decision-making (e.g. Newman et al., 2015; see also de Freitas et al., 2017; de Freitas & Cikara, 2018; Newman & Knobe, 2018). For example, a likely reason to why people blame others more for their deliberate behaviors than for their impulsive ones (Pizarro et al., 2003) is that impulsive behaviors are viewed as reflecting the *peripheral self*, whereas deliberate behaviors reflects the *true self* (Newman et al., 2014; see also Watson, 1975).

We hypothesize that biological explanations may vary with respect to the degree to which they implicate the *true self*, which in turn may affect the degree to which they mitigate responsibility. Specifically, genetic factors may be viewed as the intrinsic source of one’s proclivities and may, therefore, be interpreted as corresponding to who someone *really is*, deep

down. In contrast, other biological explanations (e.g., brain damage) may be viewed as less inherent to the individual and thus more aligned with the peripheral self. As we will see, this is not just a degree of ‘kind’ of biological explanation but what the biological explanation connotes about who the actor is vs. *should have been*. If various explanations invoking biology differentially reflect the true or peripheral selves, then they may be expected to differ in the extent to which they mitigate responsibility. Accordingly, we hypothesize genetic explanations will reflect the *true self* and offer no mitigation while other biological explanations will reflect the *peripheral self* and mitigate.

Materials and Methods

General Approach

To test the differential tagging of the *true self* and resulting mitigation, participants read a vignette about someone who committed a crime. Participants were randomly assigned to learn about different biological reasons underlying the aggressor’s inability to stop himself from carrying out the crime. After reading about the underlying reasons for the crime, participants made judgments about guilt, recommended punishment, self-control, and reported the aggressor’s *true self*.

We build on the literature in two ways. First, by including a variety of different types of biological accounts in the same study, we are able to identify which accounts produce mitigation and which do not. Second, by investigating peoples’ perceptions of the relationship between behaviors and individuals’ true versus peripheral selves, we are able to explore whether assumptions about the true self underpin differences between those biological accounts that

provide mitigation and those that do not. Data, materials, pre-registration, and analysis plans can be found at <https://osf.io/m876f/>.

Study one: The Basic Effect

We first tested whether explanations that invoked different aspects of underlying biology provided varying levels of mitigation of responsibility for behavior by comparing explanations involving neurological damage against explanations involving genetics. One control group of participants was told about someone ‘born with’ the condition underlying the crime, but without any invocation of biology, and one control group of participants were given no explanation for the behavior was provided.

Methods

Participants were 376 adults drawn from Amazon Mechanical Turk with the aim of having 100 participants per condition. Participants were required to be in the United States, have an approval rate of 95% or higher, and have participated in less than 1,000 previous studies (HITs). Participants were paid \$.30 in exchange for their participation.

Participants were told to imagine a hypothetical scenario and were then randomly assigned to one of four conditions attributing the protagonist’s lack of self-control to either genes, traumatic brain injury (TBI), being born with low self-control, or no explanation (control). Those in the *genes* group read: “J--- is an American adult who was born with genes for below-average self-control.” Those in the TBI group read: “J--- is an American adult who was in a small car accident that led to minor brain damage. The only effect was the brain damage left J--- with below-average self-control.” If they were in the *born with* group, they read: “J--- is an American adult who was born with below-average self-control.” Those in the *no explanation*

group were just told: “J--- is an American adult.” All participants were then told: “One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.” We used the moniker J--- to prevent participants from intuiting more information about the individual based on details of their name.

Afterward, participants answered:

“Would you find J--- guilty of assault and battery? [yes/no]”

On a separate page, participants then recommended punishment for J---:

“Assume J--- is found guilty. This crime is punishable by up to five years in prison or on probation. Would you recommend J-- go to prison or be put on probation? [prison/probation].”

Based on participants’ responses, they reported: “You recommended [prison/probation], this assault and battery is punishable by between 0-60 months [in prison/on probation]. How many months would you recommend J--- [go to prison/be put on probation] for? [0-60 scale].”

To ensure that participants perceived the crime in the same way, on a separate page, we asked them:

“How much self-control did J-- exert before punching the man? [5-1 unnumbered scale: A great deal, A lot, A moderate amount, A little, None at all].”

Finally, participants evaluated:

“How much do you agree with the following: Deep down, the true self of J--- is not a violent person [1-7 unnumbered, fully labeled scale strongly disagree to strongly agree]”

To see if providing *any* biological explanation altered the extent to which participants believed that humans are automata, which would, therefore, be mitigating blame, participants

were then administered the dualism scale (Nadelhoffer et al., 2014). This scale asks participants about their beliefs that humans are responsible agents. Questions from this scale were answered on a numbered 7-point Likert scale anchored with 1=Strongly Disagree, and 7=Strongly Agree, and the questions were presented in random order:

- 1) The fact that we have souls that are distinct from our material bodies is what makes humans unique.
- 2) Human action can only be understood in terms of our souls and minds and not just in terms of our brains.
- 3) Each person has a non-physical essence that makes that person unique.
- 4) The human mind cannot simply be reduced to the brain [Reverse coded].
- 5) The human mind is more than just a complicated biological machine.

We added a number of covariates to test both potential moderators and build a model of punishment based on the previous literature, using measures previously shown to relate to punishment. These covariates include the New General Self-Efficacy Scale (Chen et al., 2001), a measure of belief in Free Will (Protzko, Ouimette, & Schooler, 2015), and the belief in Immanent/Ultimate Justice scale (Maes, 1998). The measures consistently did not covary with our DVs significantly, and so we do not discuss them further. All studies were programmed in Qualtrics. Question wordings and pre-registration can be found at <https://osf.io/r4z6h/>.

Results and Discussion

For our primary DV, we used a probit regression on whether participants would find the attacker guilty. Probit was chosen a priori as the probit model's assumption of normal errors, we reason, more closely follows the mental process of a non-binary process of attributing guilty being presented as a binary output (unlike the logit model, whose logit-assumed error structure may more closely match true binarity). We found that participants were less likely to pronounce

the man guilty if his self-control failure was caused by TBI ($M = 75.8\%$) than if it was caused by genes ($M = 92.5\%$, $b_{\text{probit}}(372) = -.738$, $p = .005$, $95\% \text{ CI} = -.27 \text{ to } -1.206$) or if they were simply “born with” low self-control ($M = 91.2\%$, $b_{\text{probit}}(372) = -.654$, $p = .002$, $95\% \text{ CI} = -.197 \text{ to } -1.111$; See Figure 1). The TBI mitigation was also seen compared to a no-explanation control ($M = 96.907$, $b_{\text{probit}}(372) = -1.168$, $p < .001$, $95\% \text{ CI} = -.602 \text{ to } -1.734$). These differences are large enough to withstand even Bonferroni correction.

Figure 1: Estimated probability with 95% CI of finding the man guilty of assault and battery as a result of self-control failure.

In addition, there were no differences between the ‘genes’ and ‘born with’ conditions compared to the control (all $ps > .1$). The only significant difference in punishment was TBI reducing guilty verdicts. To put these numbers in context, all else being equal, such a pattern of verdicts in a jury

pool would change a unanimous guilty verdict in the control condition to a hung jury with 1-2 jurors advocating not guilty.

Additional punishment.

We also asked, assuming he *was* found guilty, whether the attacker should be sent to prison or be put on probation. If the biological cause was TBI, participants were less likely to recommend prison ($M = 13.7\%$) than if the cause was genes ($M = 41.9\%$, $b_{\text{probit}}(372) = -.858$, $p < .001$, 95% CI = $-.449$ to -1.268) or being ‘born with’ low self-control ($M = 40.7\%$, $b_{\text{probit}}(372) = -.891$, $p < .001$, 95% CI = $-.484$ to -1.298) or if no explanation was given ($M = 47.4\%$, $b_{\text{probit}}(372) = -1.03$, $p < .001$, 95% CI = $-.627$ to -1.433 ; see Figure 2). Finally, there was no difference between the control and the genes and ‘born with’ conditions ($ps > .35$). Thus, we have a secondary measure of harshness of punishment: if the biological cause was genes or being born with low self-control, there was no mitigation. If instead the behavior resulted from self-control failure due to TBI, the proposed punishment was more lenient.

Figure 2: Estimated probability of recommending the attacker go to prison or probation; error bars represent 95% CI

As a tertiary measure of harshness of punishment, we tested whether there were differences in how many months participants would subject the attacker to either prison or probation. A one-way ANOVA indicated no evidence for mitigation ($F(3, 372) = 1.297, p > .27$).

Level of self-control, the ‘biological automaton model,’ and true-self explanations.

We next tested different explanations for why TBI would be mitigating but not other biological causes. Participants were asked: *How much self-control did J--- exert before punching the man*. There was no effect of biological explanations on perceived self-control (all β s $< .07$, all $ps > .21$), indicating that participants understood the action and ability to resist and do otherwise to be equal across the conditions.

Finally, we tested whether participants believed the different biological causes reflected differences in who the *true self* of the attacker was. Participants were asked how much they

agreed with the statement *Deep down, the true self of J--- is not a violent person*. We argue that the more participants agree with this statement, the more it reflects the idea that J---'s violent behavior was not attributable to his *true self*. We found that people agreed more with this statement in the TBI condition ($M = 4.431$) than in the control condition ($M = 3.237$, $\beta = .349$, $p < .001$, 95% CI = .243 to .454; see Figure 3). Furthermore, there were no differences between the control and the genes or 'born with' conditions (all β s $< .09$, p s $> .17$). Crucially, people believed the *true self* of the attacker was more violent if the cause of their self-control failure and subsequent attack was caused by genes ($M = 3.527$) than by TBI ($\beta = 1.069$, $p < .001$; see Figure 3).

Figure 3: Error bars represent 95% CI. The scale was on a 1-7 unnumbered Likert scale.

Thus, we propose the reason genetic explanations are not mitigating is that people intuit the behavior to represent the *true self* of the actor. In contrast, explanations like TBI indicate the

agent's *true self* is not violent, and the behavior is attributed to the *peripheral self*. The actor really, deep-down, did not want to attack, and the mitigation comes from attributing some responsibility onto this *peripheral self*.

To test this pathway, we built an indirect model testing the effect of the type of biological explanation on guilty verdicts both directly and indirectly through our *true self* measure. This was modeled with three independent dummy variables for the TBI, genes, and born groups (control was the reference variable), ratings of the *true self* as the indirect variable, and guilty verdicts as the DV.

Results from this exploratory analysis confirmed the findings above.¹ There was no significant indirect effect through the *true self* on guilty verdicts if the self-control failure was due to the biological explanation of genes ($b_{\text{indirect.genes}} = -.146, p > .259$) or if the person was 'born with' low self-control ($b_{\text{indirect.born}} = -.065, p > .59$). As expected, however, there was a significant indirect effect of having traumatic brain injury leading people to think the *true self* of the attacker was less violent, leading to less guilty verdicts ($b_{\text{indirect.tbi}} = -.602, p = .005, 95\% \text{ CI} = -1.094 \text{ to } -.254$). Thus, the process model reaffirms the findings above, biological explanations invoking genes or being 'born with' low self-control do not mitigate punishment because they do not alter beliefs in the nature of the *true self* of the actor. TBI, as a biological explanation for self-control failure and subsequent violence, however, leads people to believe the *true self* of the attacker is less violent. Thus the attacker is not held accountable.

¹ The process model approach was suggested by Julian De Freitas in his role as a reviewer. Thus, we list them as exploratory as they were not part of our pre-registered approach. We thank him for this suggestion. All analyses were run with 10,000 bootstrapped standard errors.

Study two: Replication and Testing Deficiency ²

In study two, we attempted to replicate the primary finding of study one, with a larger and more representative sample than Amazon Mechanical Turk. We tested whether genetic explanations for crime differed from similarly-biological TBI-based explanations for crime in the degree to which they are viewed as pertaining to the *true self*, and whether this difference mediates the degree to which the explanations are viewed as mitigating responsibility. We additionally tested the possibility that a further reason for mitigation is that in cases of TBI, people view the actor as categorically different from other actors, in the sense that there is something uniquely “wrong” with them.

Methods

Participants.

We sought a substantially larger sample size to ensure the robustness of our results. Using an online panel and participant recruitment company (CriticalMix), we recruited 1,500 American adults, drawn from the panel’s roster in a stratified way with unequal probabilities of selection, so respondents resembled the nation's adult population (according to the most recently available Current Population Survey, conducted by the U.S. Census Bureau) in terms of gender, age, education, ethnicity (Hispanic vs. not), race (allowing each respondent to select more than one race), region, and income.

Procedure.

² This study was chronologically run after study three, but we include it here as study two for narrative purposes.

All participants were first told to “Please consider the following hypothetical scenario.” Then, they were randomly assigned to one of two conditions, either genes or TBI. As dependent variables, we again tested guilt, the harshness of punishment [prison or probation], fault, blame, judgments of self-control, and *true self*-ratings. On a final page, participants answered, “Based on J-- having [genes/minor brain damage] causing below-average self-control, to what extent would you say that there is something ‘wrong’ with them?” [1-5 unnumbered Likert scale: None at all, A little, A moderate amount, A lot, A great deal]. We included this final question to test the hypothesis that people believe that there is something ‘wrong’ with someone with TBI, and thus they are held to different standards of guilt/fault/blame. All other procedures matched study one. All methods, data decisions, and analysis plans were pre-registered at <https://osf.io/h2pwm/> prior to data collection.

Results and Discussion

We tested whether participants thought the attacker exerted different levels of self-control before attacking and whether participants felt there was something ‘wrong’ with the attacker. The difference in perceived self-control between TBI and genes conditions was small ($\beta = .06$) and did not withstand multiple-testing correction; with people thinking the person with TBI showing *more* self-control than in the ‘genes’ condition. In addition, there was no difference in participants perceiving something was ‘wrong’ with the actor as a function of having either genes or mild TBI causing self-control failure ($\beta = .008$). Thus, neither of these two factors drive our results.

The rest of our dependent variables were subjected to an indirect process model of the difference in the two biological explanations. The indirect effects for all variables were the extent to which participants believed the *true self* of the attacker was not a violent person. We

found evidence for the expected indirect effect: Participants believed that an attacker with a traumatic brain injury had a *true self* that was less inherently violent than one with a genetic predisposition, and to the extent that they believed that the *true self* was less violent, they were less likely to judge them as guilty ($b_{\text{indirect.guilty}} = -.42, p < .001, 95\% \text{ CI} = -.563 \text{ to } -.299$). This indirect effect through the *true self* was also significant for reducing prison recommendations over probation ($b_{\text{indirect.prison}} = -.346, p < .001, 95\% \text{ CI} = -.451 \text{ to } -.253$), believing the attacker is less at fault ($b_{\text{indirect.fault}} = -.146, p < .001, 95\% \text{ CI} = -.191 \text{ to } -.106$), and believing the victim should blame the attacker less ($b_{\text{indirect.guilty}} = -.136, p < .001, 95\% \text{ CI} = -.179 \text{ to } -.098$). In addition, there are significant residual direct effects on all four dependent variables (see Figure 4).

Figure 4: Process model of the effects of learning a biological explanation for a violent attack is caused by either TBI or genes on judgments of the attacker's *true self* and subsequent mitigation on guilty verdicts, recommending prison over probation, blame and fault. All coefficients are significant at $p < .006$. 95% CI are calculated using 10,000 bootstrapped resamples. Visual paths in the diagram are thickened to match the magnitude of the standardized effect.

Thus, in a large-scale study, we were able to confirm that not all reasons for self-control failure are equally mitigating. Even though our participants believed that the attacker exerted the

same amount of self-control across both conditions, different biological causes lead to different levels of mitigation to the extent that they involved the *true self* of the attacker.

Independent Replications

To test the robustness of our results, and a part of a prospective replication study of newly discovered findings (Protzko et al., *In Preparation*) study two was replicated four additional times, once by us and by three separate labs, each using a separate, independent sample of participants matching the demographic distribution of the United States. The primary dependent variable used was whether participants would find the man guilty due to a crime committed as part of self-control failure caused by either genes or TBI. This primary finding was supported by three separate labs and once again by ourselves. The results from the replications are presented in Table 1.

Table 1: Effect sizes from four separate replications of study three. All p -values < .001.

Replication	b_{probit}	Link
Current study	-.61	https://osf.io/h2pwm/
Replication 1	-.44	https://osf.io/v3thd/
Replication 2	-.44	https://osf.io/v2qjs/
Replication 3	-.46	https://osf.io/fbnkg/
Replication 4	-.41	https://osf.io/5xqya/

Study three: The Role of Timing, Specificity, and Rarity

In Study three, we sought to narrow down the precise difference between the genetic and TBI explanations that lead to varying degrees of mitigation. One possibility entails the timing of the biological explanation. In both the genetic and ‘being born with’ explanations for low self-control, the biological event occurs at birth or conception. TBI, while unspecified so far, may be inferred to have happened in adulthood. The timing could be important because having

something occur at birth and present through one's whole life could make it a more central trait (CITE?). In study three, we, therefore, varied the timing of the TBI to occur either in the womb or in adulthood.

Another possible source of the difference between explanations has to do with their specificity. Specific information could be easier to separate from the general *true self*, such that people may think of genes in a general sense. In contrast, something specific could be easier to dissociated from the *true self* (CITE some of the construal literature?). In studies one and two in the TBI condition, we indicated the injury occurred during a car accident, whereas no additional details were given in the 'born with' or 'genes for' conditions. To address the possible role of specificity, we varied whether participants were given specific information or more general information about genes and TBI.

A third potential source of the disparity between the genetic and TBI explanations is the rarity of the biological cause. People may believe suffering TBI is rarer than being born with or having genes for low self-control. Thus, we manipulated whether a genetic abnormality was described as rare. All data decisions, analysis plan, data, and materials were pre-registered before data collection began and can be found at <https://osf.io/unse8/>.

Methods

We planned to collect 700 participants drawn from Amazon Mechanical Turk to have 100 participants per condition. Participants were required to be in the United States, have an approval rate of 95% or higher, and have participated in ≤ 1000 previous studies (HITs). We also used an embedded script to prevent participants who took part in study one from taking part in this study. Our final sample size was 695; they were paid \$.45 for their participation.

Participants were told to imagine a hypothetical scenario and were then randomly assigned to one of seven conditions: unspecific genes, genes specific, genes rare, unspecific TBI, TBI womb, TBI adult-specific, and a control condition.

They read:

Genes: unspecific group:

“J--- is an American adult who was born with genes for permanent below-average self-control. One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

Genes: specific group:

“J--- is an American adult who was born with a rare genetic mutation on part of a gene called rs16838844. The only effect of this mutation left J-- with permanent below-average self-control. One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

Gene: rare group:

“J--- is an American adult who was born with a rare genetic mutation. The only effect of this mutation left J-- with permanent below-average self-control. One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

TBI: unspecific group:

“J--- is an American adult who suffered minor brain damage as an adult. The only effect was the brain damage left J--- with permanent below-average self-control. One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

TBI: womb group:

“J--- is an American adult who's mother was in a car accident while she was pregnant with J---. This left him being born with minor brain damage. The only effect was the brain damage left J--- with permanent below-average self-control. One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

TBI: specific group:

“J--- is a American adult who was in a car accident as an adult that led to minor brain damage. The only effect was the brain damage left J--- with permanent below-average self-control. One day, he gets into a heated argument with a stranger. Unable to

resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

Control group:

“J--- is an American adult. One day, he gets into a heated argument with a stranger. Unable to resist, he punches the man in the face so hard, the man goes permanently blind in one eye.”

Afterward, participants were asked the same guilty and prison/probation questions as in earlier studies. To elicit judgments of moral responsibility, we also asked about the different levels of fault and blame for the attacker (following Murray et al., 2018). Participants were asked: “How much at fault is J--- for the man going blind in one eye?” [1-5 unnumbered scale, 1=None at all, 2=A little, 3=A moderate amount, 4=A lot, 5=A great deal] and “How much should the victim blame J--- for making him go blind in one eye?” [1-5 unnumbered scale, 1=None at all, 2=A little, 3=A moderate amount, 4=A lot, 5=A great deal] in random order. On a separate page, we again asked the questions about self-control and *true self* as in studies one and two.

Results and Discussion

For our primary DV, we again used a probit regression on whether participants would find the attacker guilty. We replicated the basic finding of study one, namely, there was no mitigation of being found guilty between the control condition ($M = 99.092\%$) and having non-specific genes for low self-control ($M = 93.94\%$, $b_{\text{probit}}(688) = -.784$, $p > .06$). All other biological causes showed significant mitigation of guilty verdicts (all b 's < 1.13 , all $ps < .007$; see Figure 8).

Figure 8: Estimated probability with 95% CI of finding the man guilty of assault and battery as a result of self-control failure. Bars that are the same shade are not statistically different from one another.

To address our alternate explanations, we looked at the specificity and age of onset. For specificity, there was no difference between specific TBI as an adult ($M = 79.208\%$), and unspecific TBI as an adult ($M = 82\%$, $p > .61$), suggesting specificity is not a source of mitigation in these studies. Similarly, age did not alter the findings, with there being no difference in guilty verdicts from TBI as an adult from TBI in the womb ($M = 85.149\%$, $p > .26$). Contrarily, the rare gene and specific gene both provided mitigation of guilty verdicts. This suggests there is something about the specificity of a genetic explanation that provides mitigation, whereas simply saying someone has genes for low self-control does not. It may be because presenting the genes as a mutation or rarity further indicates that the *true self* of the person was supposed to be nonviolent, and the rare abnormality altered the *peripheral self*. This

would be borne out if the rare gene and mutation conditions had effects on how people interpret the actor's *true self*.

Additional punishment.

Aside from whether participants would find the man guilty, we also asked, assuming he *was* found guilty, whether the attacker should be sent to prison or be put on probation. First, there was no difference between the unspecific genes and the control condition ($p > .18$), replicating again having genes for a condition does not provide mitigation for behavior. All other explanations led to participants being less likely to recommend prison in favor of probation (a less harsh punishment, all $ps < .012$; see Figure 9). This means we again found the rare gene and the mutation conditions also provided mitigation compared to the control or 'genes for' condition, following from the results of primary mitigation in the form of less guilty verdicts.

Figure 9: Estimated probability of recommending the attacker go to prison or probation; error bars represent 95% CI.

Level of self-control, fault, blame, and true-self explanations.

Next, we tested whether participants thought the attacker exerted different levels of self-control before attacking. There were no differences among the conditions (all β s $> -.08$ and $< .064$, all $ps > .15$), indicating participants understood the action and ability to do otherwise as equal across the conditions.

Again, instead of asking about moral responsibility, we asked about fault and blame. Participants were asked *How much at fault is J--- for the man going blind in one eye* and *How much should the victim blame J--- for making him go blind in one eye?* We found both ratings of fault and blame were mitigated in all conditions relative to the control (all β s $< .2$, all $ps < .001$; see Figure 10). The unspecific genes condition was lower than the control condition ($\beta = -.206$, 95% CI = $-.321$ to $-.091$) but also higher than all other biological explanations (all β s $< -.199$, all $ps < .001$). This is the first time the unspecific genes condition has been found to be mitigating in any way. In addition, there were no differences among the other explanations from one another (all $ps > .42$). Thus, with the exception of significantly less fault in the genes condition than the control, these results indicate biological explanations cause a reduction in ascriptions of fault.

Figure 10: Error bars represent 95% CI. The scale was an unnumbered 5-point scale.

For how much the victim should blame the attacker, our results mirror those seen in the guilty verdicts and harshness of the punishment. Specifically, the unspecific genes condition was not statistically different from the control ($\beta = -.049, p > .72$), whereas all other biological explanations meant the victim should blame the attacker less for making them go blind permanently in one eye (all β s $< -.51$, all p s $< .001$; see Figure 11). In addition, there was no difference among the different biological explanations (all p s $> .07$), except the unspecific genes condition was higher in blame than all other biological conditions (compared to all other non-control groups, all p s $< .002$, see Figure 11).

Figure 11: Error bars represent 95% CI. The scale was an unnumbered 5-point scale.

Finally, we replicated whether participants believed the different biological causes reflected differences in the *true self* of the attacker. There was no difference in the unspecific genes condition versus control ($p > .75$). We also found all other biological causes provided increased beliefs the attacker was not really violent relative to the control and the unspecific genes conditions (all β s $> .89$, all p s $< .001$; see Figure 12). This helps place the mitigating effects of the ‘rare gene’ and ‘specific mutation’ findings into context. Participants are viewing the mutations as not a part of the *true self* of the actor (whom they are interpreting as less of a violent person). Such explanations instead indicate the *peripheral self* is partly to blame for the violent behavior.

Figure 12: Error bars represent 95% CI. The scale was a 1-7 unnumbered Likert scale.

Thus, the reason some biological explanations provide mitigation is that people sense the *true self* of the actor is non-violent; neurological explanations or explanations involving rare mutations tag the *peripheral self*, which is held less responsible. The actor deep down did not want to attack; mitigation comes from attributing some responsibility elsewhere. This is consistent with the notion of the good *true self*-bias (e.g., Newman, Bloom, & Knobe, 2014). However, the control and unspecific genes conditions both showed beliefs that the attacker's *true self* was more violent (see Figure 12).

An exploratory process model reaffirmed this pattern of results. We constructed the process model using the same procedures as in study two. This time, however, there are six indicator variables for the seven different conditions, with direct effects on guilty verdicts, prison recommendations, fault and blame, and indirect effects through the *true self*-beliefs. Indirect

effects (the focus of the hypotheses) are in Table 2, what is noteworthy is all indirect effects are statistically significant at $p < .001$ except for the Genes: unspecific group showing no indirect mitigation (all $ps > .75$).

Table 2: Indirect effects of different biological explanations on mitigation for assault and battery through beliefs that the *true self* of the attacker is a non-violent person. 95% CI calculated using 10,000 bootstrapped standard errors. All effects are relative to the control condition where no biological explanation was given. All indirect mitigations were significant at $p < .006$ except for having genes for low self-control conditions that showed no mitigation.

Condition		b_{indirect}	95% CI
<hr/> Genes: unspecific			
	Guilty	-.034	-.27 to .182
	Prison	-.032	-.251 to .171
	Fault	-.013	-.102 to .07
	Blame	-.013	-.098 to .067
<hr/> TBI: specific			
	Guilty	-.774	-1.204 to -.448
	Prison	-.733	-1.048 to -.464
	Fault	-.304	-.426 to -.205
	Blame	-.293	-.413 to -.193
<hr/> TBI: unspecific			
	Guilty	-.804	-1.234 to -.465
	Prison	-.762	-1.098 to -.474
	Fault	-.316	-.444 to -.215
	Blame	-.304	-.438 to -.199
<hr/> TBI: womb			
	Guilty	-.669	-1.062 to -.377
	Prison	-.633	-.948 to -.38
	Fault	-.263	-.383 to -.171
	Blame	-.253	-.377 to -.158
<hr/> Genes: rare			
	Guilty	-.615	-.99 to -.341
	Prison	-.582	-.879 to -.343
	Fault	-.242	-.36 to -.148
	Blame	-.232	-.349 to -.14
<hr/> Genes: specific			
	Guilty	-.559	-.935 to -.29
	Prison	-.53	-.827 to -.293
	Fault	-.22	-.337 to -.128
	Blame	-.212	-.332 to -.119

A finding of further note is characterizing a genetic predisposition as either a ‘rare gene’ or a ‘mutation’ mitigated via less guilt, a more lenient sentence, less fault, and less blame. This

contrasts with other investigations where criminal actions ascribed to genetic abnormalities inconsistently mitigated such factors (e.g., Appelbaum & Scurich, 2015a; Tabb et al., 2018). We may have found this effect where others had not for both methodological and theoretical reasons.

The methodological reason is we were directly testing this prediction within a controlled test (conditions against one another), whereas other tests have been embedded into 3-way (e.g., 4x3x2) ANOVAs. Such higher-order designs are dramatically underpowered and thus may not have been able to detect any mitigation (see Gelman, 2018). The theoretical reason has to do with the severity of the crimes. In our studies, the crime to be mitigated was assault leading to permanent damage. In other investigations, the crimes were shooting a police officer in the head or killing someone by stabbing them 17 times. While in the law, mitigating factors are often independent of the severity of the crime, in normal human reasoning, people are more likely to judge someone as more morally responsible for a severe outcome than for a benign outcome (Walster, 1966; Robbennolt, 2000). If someone forgets to put the parking brake on when parking on a hill and their car rolls down the hill, the forgetful driver is held more morally responsible for the accident if the car hospitalizes a bystander than if it bumps into a tree and only gets a tiny dent. Thus, severe outcomes such as shooting a police officer until an entire clip has been emptied may not be amenable to biological cause mitigation, whereas less severe violent crimes may be. Future research should address these possibilities by randomly varying the severity of the behavior to be mitigated in the context of different explanations.

A weakness of study three is the lack of full symmetric conditions. While we manipulated the timing of the TBI as occurring either in adulthood or in the womb (both of which mitigated punishment and responsibility), no such crossing of genetic effects between conception and adulthood was administered. We also did not administer a condition where

genetic effects only occur in adulthood (analogous to TBI occurring in adulthood instead of in utero). We address this issue in study four.

Study Four: Conceptual Replication with New Vignette

In Study four, we build on the previous three studies. In Study three, we found that while having genes for low self-control does not provide any mitigation, specific genes or rare genetic mutations do. We proposed that this difference may have arisen because the rare gene and ‘mutation’ accounts led to a splitting of the perpetrator from their (non-violent) *true self*. As it not clear whether it is the rarity or the ‘mutation’ that causes the mitigating, we sought to unconfound those two. Accordingly, we test whether referring to the genetic effect as a ‘variation’ instead of ‘mutation’ may be enough to remove the mitigating effect, as ‘mutation’ connotes change while ‘variation’ does not. Additionally, by randomly assigning genetic explanations to include the term ‘rare,’ we can test whether the rarity of a change alters mitigation. Finally, we test this in a pre-registered mediation process model (unlike the models of Studies one, two, and, three, where the mediations were exploratory), in which the effects of different explanations on mitigation are mediated through judgments of the *true self*, and do so using a new scenario (not just the specific assault and battery used in studies one through three), allowing for a test of the broader generalizability of our effect.

Methods

Participants.

Participants were drawn by CriticalMix, with the additional criterion that no participant that CriticalMix invited to participate in study two could be invited to participate in study four, using the same stratified random sample of panelists with unequal probabilities of selection. A

power analysis of the ‘Genes’ group versus the ‘Genes: rare’ group from study three suggested that the Odds ratio of learning the cause of low self-control being a ‘rare gene’ causes a reduction in guilty verdicts by $OR = .378$ over learning the cause was just ‘genes for’ low self-control. To replicate this effect with 80% power would require 129 participants per condition. Thus, as we are increasing the number of conditions beyond those two, and attempting to extend the finding into new scenarios whose effect size is unknown, we sought to maximize power by collecting 1,500 participants, around 214 per condition.

Method.

Participants were told to imagine a hypothetical scenario, and were then randomly assigned to one of seven conditions: ‘Genes for,’ ‘Genes: mutation,’ or ‘Genes: variant’; each of these conditions with the term ‘rare’ inserted just before the explanation, or a control group.

Control group

“C--- is an American adult. One day, he is walking down the street and sees a wealthy looking woman walking towards him. Unable to resist, he trips her as she walks past and runs off with her purse.”

This crime was chosen because its seriousness matches that of the assault case in studies 1-3 being a low-level felony with the possibility of mandatory jail time.

Genes for group [with rare condition]

“C--- is an American adult who was born with [rare] genes for permanent below-average self-control. One day, he is walking down the street and sees a wealthy looking woman walking towards him. Unable to resist, he trips her as she walks past and runs off with her purse.”

Genes: mutation group [with rare condition]

“C--- is an American adult who was born with a [rare] genetic mutation on part of a gene called rs16838844. The only effect of this mutation is that it left C-- with permanent below-average self-control. One day, he is walking down the street and sees a wealthy looking woman walking towards him. Unable to resist, he trips her as she walks past and runs off with her purse.”

Genes: variant group [with rare condition]

“C--- is an American adult who was born with a [rare] genetic variant of part of a gene called rs16838844. The only effect of this genetic variant is that it left C-- with permanent below-average self-control. One day, he is walking down the street and sees a wealthy looking woman walking towards him. Unable to resist, he trips her as she walks past and runs off with her purse.”

All participants read (on the same page): “Would you find C---- guilty of robbery” [no/yes];

Then, all on separate pages: “Assume C---- was found guilty. This crime is punishable by up to 5 years in prison or on probation. Would you recommend C--- go to prison or be put on probation?” [prison/probation]. “How much self-control did C---- exert before snatching the purse?” [1-7 a great deal to very little scale]; “Deep down, the true self of C---- is a dangerous person” [1-7 scale strongly agree to strongly disagree]. On the next page: “On a scale of 0-100, with 0 meaning not likely at all, 50 meaning a 50% chance like getting heads on a coin flip, and 100 meaning 100% likely, how likely do you think C---- would be to commit a crime like this again if they were put on probation?” Participants then answered: “How much should the victim blame C--- for stealing her purse?” [1-5 unnumbered scale, 1=None at all, 2=A little, 3=A moderate amount, 4=A lot, 5=A great deal].

Study three showed that the mitigating effect of TBI on resulting low self-control and criminality was not driven by people thinking there was ‘something wrong’ with the actor. We attempt to test this idea again using items meant to tap into aspects of individuals that inform how much we hold different people accountable for the same behavior (Bastian et al., 2011). Participants rated: ‘To what extent do you think C--- is culturally refined?’ and ‘To what extent do you think C--- is rational?’ (Bastian et al., 2011). Ratings were made on a 0-100% scale with increments at 5 points, and labels at 0 = Completely not [culturally refined/rational], 50 = Completely unsure; and 100 = Completely [culturally refined/rational]. Both questions were on the same page, presented in random order, and were averaged for the analysis (following Bastian

et al., 2011). All materials, methods, exclusion criteria, and analysis code was pre-registered prior to data collection at <https://osf.io/g7zet/>.

Results

Rarity

First, being told any of these events or conditions was ‘rare’ did not alter the results; there was no main effect of rarity ($p > .12$), nor did rarity interact with any of the specific conditions (all $ps > .78$). Consistent with our pre-registration plan, we continue all analyses collapsing across this rarity factor.

Pre-registered Mediation Model

Figure 5: Pre-registered mediation model showing the effect of different biological explanations invoking genes (e.g., Genes for low self-control, a genetic variant causing low self-control, a genetic mutation causing low self-control, the reference group is a no-explanation control group) on mitigation for committing the robbery. Paths are thickened to correspond to their standardized effect size.

We hypothesized that mitigation for criminal activity would occur only when learning a biological explanation entails differences about the actor’s *true self*. Changing belief about the

true self will subsequently alter judgments of guilty verdicts, harshness of punishment, and how much blame should be accorded. The results of the overall model can be seen in *Figure 1*.

We found support for this hypothesis. Specifically, only having a ‘genetic mutation’ led to the belief that the person’s *true self* was not violent ($\beta = -.105$, 95% CI = $-.175$ to $-.033$); there was no effect of having a ‘genetic variant’ or ‘genes for’ low self-control on *true self*-beliefs. This effect of a ‘genetic mutation’ on having a better *true self* mediated the mitigation of guilty verdicts for robbery ($\beta = -.019$, $p = .015$, 95% CI = $-.037$ to $-.006$); in short because people believed the person with the genetic mutation was, deep down, not a dangerous person. There was no indirect effect for either having ‘genes for’ low-self-control ($p > .96$) or a ‘genetic variant’ ($p > .21$).

For harshness of punishment (i.e., less prison sentencing opposed to probation), there was also an indirect effect of having a ‘genetic mutation’ through the belief that the *true self* was not a dangerous person ($\beta = -.047$, $p = .005$, 95% CI = $-.08$ to $-.015$). There was no such indirect effect for having ‘genes for’ low self-control ($p > .96$) or a ‘genetic variant’ ($p > .19$).

Furthermore, blame was also reduced when one had a ‘genetic mutation,’ mediated through judgments that the true self is not dangerous ($\beta = -.032$, $p = .005$, 95% CI = $-.055$ to $-.01$), but not have ‘genes for’ low self-control ($p > .96$) or a genetic variant ($p > .19$).

Additional Analyses

We further sought to understand the phenomena by testing whether the different explanations invoking genetics altered how ‘human’ people viewed the robber as being as well as the likelihood of performing the act again if put on probation.

We found no differences between the different types of genetic explanations on how ‘human’ participants saw the robber (all $ps > .66$). For recidivism (robbing again if put on

probation), there was likewise no difference between the different explanations invoking genetics (all $ps > .06$).

Thus, overall, we found support for the hypothesis that only biological explanations representing a ‘change’ from who the person ‘should have been’ mitigate illegal behavior. Genetic explanations indicating a change in the person (i.e., they would be this way if not for a mutation) play on people’s beliefs of the good *true self*, believing the accused is still good deep down and should be less harshly punished, while genetic explanations connoting no change (e.g., born with genes for... has a genetic variant for...) imply the criminal’s *true self* is to blame.

General Discussion

Across four studies, we find that only the biological explanations that invoke a *change* in a criminal agent mitigate guilt and punishment. People appear to interpret changes like genetic mutations or brain damage as altering who the person is on the surface while leaving their *true self* untouched. As people tend to believe that one’s *true self* is usually good, they feel that the agent is less to blame for their criminal behavior. When, on the other hand, the biological explanation does not imply *change*, as in being born with a condition or having genes for the same level of self-control, it is seen as constitutive of the *true self* of the person: That person really is a criminal deep down.

Genetic explanations, as a general reason, do not mitigate guilt or punishment or blame precisely because genes are viewed as constitutive of who we are (see also Newman & Knobe, 2018). Thus, someone with genes for X is considered to have a *true self* that is reflecting X. Instead, specifics are required indicating the genes are some sort of abnormality or break from who the person really is supposed to have been (a mutation). Committing a crime because one has genes for poor self-control means one’s *true self* is the one committing the crime. This can

explain why genetic explanations lead laypeople and judges to fear a murderer more than if no genetic cause is given (e.g., Appelbaum & Scurich, 2014; Costa et al., 2017); the murderer is feared because deep down, their *true self* is a murderer. Posing the genetic cause as stemming from a mutation, however, does provide mitigation precisely because it is indicating the *true self* is one way, and the mutation has caused a change. This can be seen in studies three and four when genetic mutations indicated that the *true self* of the attacker was indeed less dangerous.

When the explanation involves *changes* to individuals at the neurologic level, the resulting behavior was always mitigated. This occurred because such behavior could be attributed to the *peripheral self* of the actor. Accordingly, all forms of neurological explanations led to less unanimous guilty verdicts, less severe punishments, less fault, less blame, and people believing the true selves of the actors were less violent.

Individuals also did not believe there was something more *wrong* with the aggressor between the TBI and genes conditions in study two. In all conditions, participants believed the aggressor had the same capacities for self-control and behavior. Thus, differences in the degree of responsibility, fault, and blame could not be attributed to differences in self-control capacity for violent action (see Nelkin, 2016; Murray, 2017; for example).

The simplest explanation for heterogeneous mitigation of assorted biological explanations across various studies, therefore, stems from how much those explanations reveal about the agent's *true self*. Behavior resulting from neurological damage, or genetic mutations, or brain trauma is not taken to be indicative of an agent's *true self*. The source of the inconsistencies in the literature has likely arisen from lumping all genetic and neural reasons under the same heading of 'biological explanations.' As we show here, different explanations,

instead, create different interpretations of the underlying core of the individual, altering our ascriptions of responsibility.

Limitations

One potential limitation of this study, which may be a limitation of most experiments on criminal and responsibility mitigation, is the brief nature of the experiment. Giving people a couple of sentences to read about a fictitious person and then asking about the agent seems to ask people to make judgments that go well beyond the information they have been given. To answer these questions outside the bounds of an experiment, one would need much more information about the person, especially details of the person's history. Making judgments about self-control, intentions, motives, and whom the agent is, deep down, based on very little information, is inherently a hypothetical exercise, unlike real life. This paper, however, is still important as it may lead to a better understanding of the role and use or misuse of biological explanations in the minds of the general population who make up juries. It is as yet unclear that the results will be the same in a courtroom where much more information may be available to the individuals being asked.

It is important to remember that while the punishment was reduced in the neurological or mutation conditions, it was not absent. In study one, for example, guilty verdicts were reduced, but only from 93% to 76%. While this is both statistically and practically large, it does not indicate the criminal is absolved of responsibility and liability. This indicates that participants believe that people should still be punished despite their behavior not being entirely indicative of who they really are. In the studies presented in this manuscript, people who had genes for self-control failure leading to criminal behavior were interpreted as having a *true self* that was more violent or dangerous than people who had other sorts of underlying biological underpinnings of

their self-control failure. Even in the case of a presumably less morally wrong crime (study four), 84.1% of participants still found the agent guilty in the most mitigating condition. Thus, the term 'mitigation' should not be conflated with 'exoneration' based on the evidence here.

Future Directions

A number of future directions lie open. People propose harsher punishments for those with a genetic predisposition towards bad behavior than other types of explanations. Future research can explore the extent to which genetic versus other biological explanations for a behavior alter the extent people believe the actor had free will. Reducing people's belief in free will has been suggested to lead to lower punishment for others (Shariff et al., 2014). While we did not find any effect of genetic explanations on perceptions about the ability to do otherwise (sometimes considered free will; e.g., Hobbes, 1654/1999), people may be interpreting genetic predispositions as indicating a lack of free will. Free will may thus be another mediator of the mitigation effect, in addition to ascriptions away from the *true* and to the *peripheral self*.

While we saw differences in the extent explanations mitigated criminal behavior, different explanations could also alter how people *praise* behavior. There exist a large number of asymmetries in how people treat factors surrounding good versus bad behavior. When people are overwhelmed with emotion and act spontaneously, for example, they receive less blame if their actions are morally bad but not less praise if their actions are morally good (Pizarro et al., 2003). A reason for this is the good behavior is ascribed to the *true self* while the bad behavior is ascribed to the *peripheral self* (Newman et al., 2014). Therefore, we may expect ascribing good traits to genes (like physique or singing aptitude) to receive different levels of praise than if ascribed to neurology or some sort of biological change. Someone who is a great athlete due to genetics may be praised differently than someone who is an equally good athlete through hard

work, for example. Future research in this area can help highlight further disconnects between different types of explanations listed as ‘biological.’

Our genetic profile is with us at conception, and in the earliest sense is what differentiates us from others. More research is particularly needed to understand why genes so specifically indicate what our *true self* is. While the true-self theory is about people believing in true vs. peripheral selves, no such actual distinction may exist in nature; genes, brain injury, rare mutations, on the other hand, do. There is no difference, biologically speaking, between a person with a genetic mutation for X and someone with a rare gene for X. How they are treated by people narratively, however, is different; we see different understandings of people’s conceptions of their *true self*. Therefore, the process by which people map some but not all biology onto the *true self* can help elucidate when biology mitigates behavior. It may be people can view genes to be both primary and immutable, less a specific mutation, which maps them onto the *true self*. Genes may have come to replace the psychological essence that used to be considered as the core of who someone is (e.g., Dar-Nimrod & Heine, 2011b). Interestingly, the *true self* maps onto anything biological at all, instead of being purely metaphysical like a soul. Only future research can tease these things apart.

Conclusion

Overall, not all explanations invoking biology are equal. People generally believe that genetic predispositions reflect someone’s *true self*. Thus, acting because of genetic predispositions offers little mitigation because the agent is acting from whom they really are. Explanations involving neurology, or brain injury, or genetic mutations, on the other hand, intone *change from the true self* and provide mitigation precisely because people believe the behavior does not reflect who the individual truly is, deep down.

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