THE UNIVERSITY *of* EDINBURGH

Edinburgh Research Explorer

Behavior genetics and agent responsibility

Citation for published version:

Wundisch, J, Johnson, W & Bittner, R 2019, 'Behavior genetics and agent responsibility', *Zeitschrift für Ethik und Moralphilosophie*, vol. 2, no. 1, pp. 21-34. <https://doi.org/10.1007/s42048-019-00037-4>

Digital Object Identifier (DOI):

[10.1007/s42048-019-00037-4](https://doi.org/10.1007/s42048-019-00037-4)

Link:

[Link to publication record in Edinburgh Research Explorer](#)

Document Version:

Peer reviewed version

Published In:

Zeitschrift für Ethik und Moralphilosophie

Publisher Rights Statement:

This is a post-peer-review, pre-copyedit version of an article published in *Zeitschrift für Ethik und Moralphilosophie*. The final authenticated version is available online at:
<https://link.springer.com/article/10.1007/s42048-019-00037-4>

General rights

Copyright for the publications made accessible via the Edinburgh Research Explorer is retained by the author(s) and / or other copyright owners and it is a condition of accessing these publications that users recognise and abide by the legal requirements associated with these rights.

Take down policy

The University of Edinburgh has made every reasonable effort to ensure that Edinburgh Research Explorer content complies with UK legislation. If you believe that the public display of this file breaches copyright please contact openaccess@ed.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.

Behavior Genetics and Agent Responsibility

Joachim Wundisch

Wendy Johnson

Rudiger Bittner

Abstract

Recent evidence from psychological science and genetics suggests that genetic influences underlie all behavior as well as the most worrisome social inequalities. This may be considered to call into question traditional conceptions of agency and agent responsibility. They could be thought to be undermined if gene-environment transactions were sufficiently potent in influencing behaviors. Here we identify the theoretical parameters that require investigation and the conceptual challenges to agent responsibility that arise from research in behavior genetics. We (i) introduce the empirical basis of the discussion, (ii) identify the particular questions that arise from considering the connection between behavior genetics and agent responsibility in the context of the legal system, (iii) bring into focus the general challenges to agent responsibility, and (iv) outline a potential resolution.

Keywords: agent responsibility; agency; behavior genetics; gene-environment transaction; law; crime; culpability

1. Introduction

Traditionally, conceptions of the nature and extent of human agency and agent responsibility have focused on capacity for individual action, locating it to varying degrees within the individual and the broader social and historical structures in which individuals are embedded. Recent empirical evidence from psychological science and genetics, however, suggests that genetic influences and gene-environment interactions and correlations underlie behavior and the most worrisome social inequalities (in income, educational attainment, health, crime rates, etc.). Some have viewed this evidence as introducing another, biological, ‘force’ directing human action and limiting its expressive range. Because we are biological beings, empirical evidence about our biology is highly relevant in considering agentic capacity, as has long been recognized in the law with respect to capacity-limiting medical conditions. Thus, it may be reasonable to view the evidence of genetic involvement in behavior in this way if gene-environment transactions are sufficiently potent in influencing behaviors.

Whether traditional conceptions of agency and agent responsibility should be maintained is of twofold significance in the face of this evidence: First, it is pivotal to assessment of social interactions because agent responsibility is typically viewed as a condition of moral blame and legal culpability. Second, it may have concrete implications for public policy because assumptions of various degrees of agent responsibility underlie almost all social justice policies in e.g. the educational sector and the welfare state.¹ Therefore, observations from psychological science and behavior genetics may be socially and politically important – even if only because people take them to be. The most prominently discussed yet unresolved challenges here are the putative connections between behavior genetics and legal responses to crime.

In this article, we identify the theoretical parameters underlying traditional conceptions of agent responsibility that research in behavior genetics potentially challenges. We do not suggest that this research poses any novel threat to agency and responsibility. The questions behavior genetic information raises tie in with discussions going back to antiquity.² Our concern, however, is that, despite increasing evidence that presence of genetic influence on behavior and psychological characteristics is far from deterministic, such evidence continues to be considered – by some courts and members of the public – as

¹ Additionally or alternatively, consequentialist considerations may give reason to assign blame, praise, punishment, or reward. With regard to punishment, such considerations are typically discussed under the header of deterrence. Here we focus on questions of agent responsibility that are distinct from issues in deterrence theory.

² Bobzien (1998).

1 undermining judgements of responsibility. It is this conception that we wish to rebut. For example, an
2 Italian court reduced the sentence of a convicted murderer noting that some of his genetic variants have
3 been associated with violence, and the judge found this (association-only) evidence “particularly
4 compelling”.³ Further, survey research published in 2012 and based on responses from U.S. state trial
5 judges indicated that behavior genetic information could affect sentences more generally.⁴ Although the
6 efficacy of behavior genetic information as a legal tool has detractors (Denno 2013; Scurich and
7 Appelbaum 2017), empirical research suggests that behavior genetic as well as neuroscientific information
8 is frequently introduced in legal proceedings (Farahany 2016; McSwiggan, Elger, and Appelbaum 2016;
9 Kogel and Westgeest 2015). Accordingly, there is a clear need to develop recommendations on how the
10 criminal justice system *should* respond to behavior genetic evidence (González-Tapia and Obsuth 2015;
11 Farahany 2016).

12 The relevance of our inquiry is further emphasized by current visions of easily available, highly
13 controllable gene editing in the wake of advancing CRISPR-Cas9 technology.⁵ In cases where targeted
14 genetic manipulations have particular consequences, questions of whether genetic influence on behavior
15 may undermine agent responsibility could evolve into questions of whether agent responsibility is
16 transferred from the genetically manipulated individual to the person who is responsible for the decision
17 to carry out the genetic manipulation.

18 These are sufficient reasons to continue to address the question to what extent amending judgements
19 of responsibility based on evidence from behavior genetics is warranted. Behavior genetic evidence does
20 not raise questions fundamentally different from those arising in the context of purely social explanations
21 of crime. Still, superficial understanding of genetics may lead people to be more inclined to excuse a person
22 carrying “bad genes”. While no credible studies point towards substantive main effects of any supposedly
23 criminal genes, behavior genetic evidence in the aggregate does suggest that the genome as a whole – in
24 transaction with particular environments – contributes to criminality. It is one goal of our paper to
25 strengthen the argument that we should not treat behavior genetic evidence as exculpatory. For that
26 purpose, we seek to substantiate three claims. First, in Section 2, we discuss the empirical basis for genetic
27 involvement in human behavior to argue that existing and likely future evidence is far too weak to guide
28 assessments of legal or moral guilt. This weakness of the empirical evidence in particular is often neglected

³ Feresin (2009). This sentence reduction was specified independently of a different sentence reduction that was made due to the defendant’s psychiatric illness.

⁴ See Aspinwall et al. (2012) but also Denno (2013). For a discussion, see section 3 below.

⁵ Doudna and Sternberg (2017).

1 in the discussion of legal or moral responsibility. Second, Section 3 identifies the particular questions that
2 arise from considering the connection between behavior genetics and agent responsibility in the context
3 of the legal system to argue that *even if* behavior genetic information were substantially stronger, legal
4 systems should not necessarily rely upon it. Section 3 also identifies the separate question of the correct
5 role of behavior genetic evidence in capital sentencing. Third, Section 4 brings into focus the general
6 challenges that behavior genetic evidence poses to agent responsibility and outlines a potential resolution.
7 Section 5 concludes.

8 9 2. Empirical basis

10 Accumulated evidence of pervasive and substantial genetic influence on patterns of behavior has shaken
11 many people's confidence in longstanding and broadly-held assumptions that systematic patterns of
12 behavior are learned response processes, and that individuals' failure to establish socially acceptable
13 patterns results primarily from deprived and/or abusive or otherwise inappropriate developmental
14 environments. It has shaken such confidence because 'genetic influence' is commonly understood to mean
15 that each unit of material present at conception in any organism's genome unleashes a set of biochemical
16 transactions in the one manner it evolved to do, with inevitable and specific impacts on that organism's
17 life that lie beyond the organism's control. If this is a valid interpretation of the evidence, it may be difficult
18 to hold people responsible for their actions. The interpretation also fosters the scientific goal of identifying
19 specific genetic variants involved in socially relevant behavior patterns, to which considerable and
20 increasing effort and research funding have been dedicated in the past 40 years or so. This has resulted in
21 frequent public media reports of genetic linkage 'discoveries', along with discussion of their prospects for
22 the longer-term goal of altering functions of genes whose effects we do not like and implanting or
23 enhancing effects of ones whose effects we do. This has been enhanced considerably in the last few years
24 by the very recent development of powerful new gene editing capabilities.

25 Despite a few major and well-publicized breakthroughs for conditions such as Huntington's
26 Disease (1993) and macular degeneration (2005), however, the overall result of this effort has been to
27 recognize that literally thousands of genes are involved even in seemingly straightforward physical traits
28 such as height (e.g., Wood, Esko, Yang et al., 2014), as well as in most commonly occurring medical (e.g.,
29 for Type 2 diabetes, Prasad & Groop, 2015) and psychopathological conditions (e.g., for schizophrenia,
30 Harrison, 2015), and more like tens of thousands in most behavioral patterns and personal characteristics
31 expressed to varying degrees by all humans (e.g., for intelligence, Plomin & Deary, 2015, and neuroticism,

Luciano et al., 2018). Chabris et al. (2015) recently noted the pervasiveness of this extreme polygenicity of behavioral traits and declared it the ‘Fourth Law of Behavioral Genetics’.

Moreover, this research has revealed that the same individual genetic variants are involved in many different traits and behavioral patterns, indicating that genes do not tend to associate uniquely with specific traits. Breeding and gene-deletion experiments in model organisms also indicate that the genome is extremely ‘deep’, in the sense that, as some genes are bred out of a population or removed from individual organisms, others often ‘step in’ to maintain phenotypes under study (e.g., Hill, 2005). And studies in developmental biology, again primarily in model organisms, make clear that the degree to and manner in which genes are expressed is heavily dependent on environmental circumstances. These researchers are increasingly describing genes as acting more in response to environmental demands than to ‘determine’ observable characteristics (e.g., Deacon, 2014; Jablonka and Lamb, 2014), though evidence for ‘Lamarckian’ gene action goes back to the turn of the last century (e.g., Stockard, 1921; Wilder, 1908).

Though the relevance to humans is always a question in such experiments on other species, the idea that the human genome functions in a manner simpler than those of fruit flies, corn plants, or mice is untenable. Taken together, this research indicates that – though genes may constrain general ranges of some potentials in humans – they do not exert similar constraints on how people acquire or shed socially defined virtues or vices, and even less do they dictate expressions of particular behaviors on specific occasions. In particular, recent studies in developmental genetics and epigenetics indicate a need to consider the possibility that transactions between environmental conditions and genetic expression undermine the commonly held premise that the genome is a primary cause of phenotype – that presence of a genetic variant with a particular function can *prima facie* be considered a causal force (e.g., Jablonka & Lamb, 2014). That is, frequently the environment draws out new applications for gene expression.

3. Behavior genetics and the law

Behavior genetic research has long motivated not only philosophical arguments⁶ pertaining to agent responsibility, but also legal arguments in the courts.⁷ Internationally, and in the U.S. legal system in particular, genetic predispositions have been used either in attempts to undermine judgements of criminal responsibility or to argue for sentence mitigation.⁸ According to Farahany and Bernet (2006: 72) “human

⁶ See e.g. Wasserman and Wachbroit (2001), Wasserman (2004), and Wasserman (2006).

⁷ See Farahany and Coleman (2006: Part II), Farahany and Bernet (2006), Bernet et. al. (2007), Denno (2006), Denno (2011) and also Coffey (1993), Johnson (1998), Stone (2003), and Feresin (2009).

⁸ Farahany and Bernet (2006) and Farahany and Coleman (2006: Part II). See also Denno (2009) and Baum (2013). See also the literature reviewed in Scurich and Appelbaum (2016: 141-3).

1 behavioral genetics research [...] in the U.S. criminal justice system [...] will only become more prevalent”.⁹
2 As mentioned in the introduction, prominently published survey research has suggested that behavior-
3 genetic evidence could indeed have important effects on verdicts. Specifically, Aspinwall et al. (2012)
4 published results of a survey of U.S. state trial judges in *Science*, concluding that judges’ expressed
5 sentencing intentions in a hypothetical case were statistically significantly (albeit marginally) lower when
6 expert testimony had included information concerning associations among genetic variants, emotional
7 and moral development, and violent behavior.¹⁰

8 Denno (2013) noted, however, rather limited evidence that judges or juries take such information
9 into consideration in actual cases. She pointed out several factors limiting the ecological validity and
10 generalizability of Aspinwall et al.’s (2012) study, and reviewed her own survey of actual U. S. cases in
11 which behavior genetic information had been put forward, concluding that it had come into play only as
12 one of many other factors supporting a phenotypic diagnosis that was sometimes considered mitigating
13 and sometimes not. Her approach also has limitations, though, including inability to standardize judicial
14 conditions across actual cases, inability to control case conditions and legal presentations to enable causal
15 inferences, selectivity in availability of records of court decisions, and inability to assess potential impact
16 of newly emerging science (Brown et al. 2016). Moreover, media reports of those and other cases in which
17 behavior genetic defenses were used, have often focused on the genetic information, under headings such
18 as “Can Your Genes Make You Kill?” or “Can Your Genes Make You Murder?” (Hagerty 2010; Parshley
19 2016). This might propagate a public impression that genes can be powerful in determining action, thus
20 undermining or at least limiting personal responsibility for it; or the impression may be that at any rate
21 judges or juries think so, whether or not they actually do. As Denno pointed out, the Aspinwall et al. study
22 is important if only because it was carried out by a team of appropriately credentialed professors of
23 psychology, law, and philosophy and published in a prominent scientific journal, thus making those
24 impressions appear highly credible. Of course, the opinions of judges and laypeople do not offer
25 substantive guidelines for how behavior genetics *should* relate to agent responsibility or the law. To

⁹ Scurich and Appelbaum (2017: 772) contended that the “introduction of genetic evidence of a predisposition to violent or impulsive behaviour is on the rise in criminal trials” but argued that “its use in the legal process is likely to diminish” because, “a panoply of data suggests that such evidence is ineffective at reducing judgements of culpability and punishment”. Morse (2011: 208) lamented that “[i]t is seldom explained why a genetically driven predisposition justifies a sentence reduction”. That said, he noted that “[t]he use of G x E for making decisions about criminal responsibility and sentencing will surely increase”.

¹⁰ But also see Scurich and Appelbaum (2017); Scurich and Appelbaum (2016); Appelbaum, Scurich, and Raad (2015); as well as Appelbaum and Scurich (2014: 96); and Denno (2013). For evidence of the continued relevance of behavior genetic information in the law see González-Tapia and Obsuth (2015); Kogel and Westgeest (2015); Farahany (2016); as well as McSwiggan, Elger, and Appelbaum (2016).

1 develop such guidelines, consideration of empirical evidence regarding just how genes are involved in
2 behavior must be primary.

3 As regards the legal system, four related questions demand investigation. First, behavior genetic
4 information may be viewed as a challenge to the application of existing laws. In particular, the defense
5 may argue that the defendant is not responsible for the crime in question because her genes caused her
6 to act in a particular way. In “From Chance to Choice,” Buchanan et al. (2000) were among the first to
7 present sustained philosophical analyses of issues of punitive justice arising from genetic information.
8 While they only touched briefly on the subject of responsibility in the course of dismissing outright an
9 interpretation of genetic determinism (24-6), they argued that, if genes were to cause behavior, this would
10 indeed be exculpatory because “tiny chemical factories embedded within them” would be responsible for
11 individuals’ actions rather than the individuals themselves. Such a strictly deterministic interpretation of
12 genetic influence cannot be sustained, but one may well argue that genetic predispositions to act in
13 particular ways could give grounds to claims of reduced responsibility. As noted above, this has already
14 taken place: claims of genetic predispositions to violence have been used to undermine claims of
15 responsibility. Assessing the merits of such an argument requires understanding the nature of the links
16 between genes and individual behaviors observed in empirical studies. However, not only does the
17 evidence we have not underwrite such direct connections, it also makes clear that there are many
18 alternative pathways through which even highly robust associations can rise, and the vast majority of
19 observed associations are both vanishingly small in magnitude and inconsistently observable.

20 Conversely, it may be asked how existing legal systems such as that in the U.S., which is typical of
21 many other countries’ in seeing at least ‘ordinary adults’ to be responsible for their actions, should respond
22 if such directly deterministic connections were to be established. This requires not just addressing
23 philosophical questions of whether, when, and to what degree humans have free will, but also the legal
24 thinking that went into the law’s conditions of punishment. For example, Morse (2011b: 379) argued that
25 U.S. criminal law is based on a compatibilist theory of responsibility which posits that determined behavior
26 and free will can co-exist. If this were true, even if behavior genetic research could establish that
27 sometimes human actions are directly caused by genes, this would not suffice to undermine ascriptions of
28 responsibility in U.S. criminal law.

29 In opposition to this compatibilist interpretation of U.S. criminal law, Kaye (2004) defended the
30 “causal theory of the excuses”. He (2004: 1117) interpreted the causal theory to hold that criminal law
31 considers that (i) *sometimes* actions are “caused by forces beyond the actor’s control”, and (ii) “actors
32 cannot be blamed for conduct caused by forces beyond their control”. In explicating (i), and to avoid having

1 the causal theory of the excuses simply excuse *all* actions, he argued that the law relies on some version
2 of *partial* determinism, on the claim, that is, that not all but only some human actions are causally
3 determined. However, partial determinism has long been a highly contentious claim, since it is hard to see
4 a reason to exempt a particular set of human events, *some* human actions, from deterministic causal
5 explanation. A compatibilist interpretation of criminal law would thus appear to be preferable since it does
6 not need to justify such an exemption. In any case, very different interpretations of the underlying basis
7 of the same legal system could also be made and genetic evidence may imply rather different conclusions
8 depending on both which basis, if any single one, *actually* underlies the legal system and whatever
9 particular judges may think about what underlies it.

10 Second, behavior genetic research may not only challenge how the law is applied, but also the law
11 itself. As Morse (2011b: 379) noted, behavior genetic information can be interpreted as an external
12 challenge to the law. In particular, behavior genetic information could support the age-old hypothesis that
13 everything – including but not limited to human actions – is determined by antecedent conditions and the
14 laws of nature. Such determinism would only be in part genetic¹¹, but it could appear to give grounds to
15 doubt the law's current ascriptions of responsibility. To counter that general challenge requires a similarly
16 general defense of compatibilism. As to what extent public opinion would be swayed by observations
17 regarding genetic influences on behavioral characteristics, there is evidence to suggest that even general
18 public acceptance of determinism would not necessarily lead to rejection of agency and responsibility.¹²

¹¹ See Greene and Cohen (2004: 1781) and Kaebnick (2006: 223-224).

¹² See e.g. Nahmias, Morris, Nadelhoffer and Turner (2006), Knobe (2014); Nahmias, Shepard, and Reuter (2014) as well as Scurich and Appelbaum (2016: 155). Knobe (2014: section 2) reported the surprising yet well replicated result that study participants who are asked to pass judgement on agents who are stipulated to be fully causally determined tend to assign them full moral responsibility. However, as Nichols and Knobe (2007) have observed, this result seems to persist only in cases where subjects pass judgment on concrete agents and cases. The abstract question of whether persons are fully morally responsible in a causally determined universe elicits the opposite and incompatibilist response. Knobe (2014: section 2) reported that no conclusive explanation of the divergence between abstract and concrete judgements has been presented. The intuitive explanation that concrete cases elicit emotional responses which bias judgements, while abstract cases elicit no such responses has been observed to be insufficient to explain the discrepancy (Knobe 2014). Knobe (2014: note 2) hypothesized that people simply do not rely on deterministic understandings of human action if the case presented is sufficiently concrete. Standard non-deterministic interpretations of human decision-making is hypothesized to override descriptions of determinism in these cases.

According to Nahmias and Murray (2014) the incompatibilist judgements of study participants (holding agents not to be morally responsible in a deterministic universe) can be explained by a mistaken interpretation of determinism. The inferred mistake lies in interpreting “determinism to imply that agents’ mental states are bypassed by the causal chains that lead to their behavior” (p. 434). Once study participants realize that no such bypassing occurs and that human decision-making remains operative and tied to mental states even in a deterministic universe “significantly higher scores for agents’ moral responsibility, free will, and blameworthiness in the abstract scenarios” result (p. 452). These differences in scholars’ interpretations of study participants’ attributions aside, acceptance of determinism does not necessarily lead them to reject agency and responsibility.

1 Thirdly and separately, it may be asked whether the law should consider behavior genetic
2 evidence as grounds for milder sentencing. The importance of sentencing is especially clear in capital cases
3 where defense is focused on avoiding the death penalty. Given that defense teams have great leeway in
4 introducing mitigating evidence and that the evidence does not need to diminish responsibility or
5 culpability, it is possible that behavior genetic information could mitigate punishment without affecting
6 assessment of responsibility. As O'Brien (2008: 725) noted, "it has long been recognized that a competent
7 mitigation investigation has to include family history going back at least three generations, and must
8 document genetic history, patterns, and effects of familial medical conditions", and he (2008: 715)
9 observed that "[t]he [Supreme] Court recently emphasized that the concept of mitigation extends far
10 beyond factors related to the defendant's culpability in the underlying offense, striking down any
11 requirement to establish a causal nexus between a mitigating factor and the crime." Thus, even if behavior
12 genetic evidence is insufficient to undermine agent responsibility, it could nevertheless be used to support
13 e.g. the most common kinds of claims to mitigation¹³, which are based on the defendant's greater difficulty
14 in abiding by the law. In fact, though, the questions of whether agent responsibility is undermined and of
15 whether mitigation is appropriate are only dissimilar on the surface. In the end, both must be answered
16 with reference to the relevant conception of personal autonomy. If personal autonomy is undermined so
17 is agent responsibility. But only if personal autonomy is undermined can we support claims to mitigation
18 based on a 'defendant's greater difficulty in abiding by the law'. If we endorse a conception of personal
19 autonomy that relies on the ability to act otherwise, behavior genetic information does not pose a threat
20 because behavior genetic determinism is untenable. If, alternatively, we rely upon some compatibilist
21 interpretation of personal autonomy, behavior genetic information could only support mitigation if
22 autonomy in that sense (e.g. as the subjective experience of independent decision-making) were to be
23 undermined by it as well. Again, behavior genetic research does not bear this out and, therefore, cannot
24 support claims for mitigation except in cases where it functions as one factor supporting a clinical diagnosis
25 of diminished responsibility (e.g. in a case of Down's Syndrome).

26 A fourth important question pertains to the potential power of any method of genomic prediction
27 of violent behavior.¹⁴ Depending on the sensitivity and specificity of such a method and perceived
28 importance of public safety over individual liberty, genomic prediction could aggravate sentencing
29 decisions in ways that counteract any potential mitigation of individual responsibility on individual case

¹³ Appelbaum (2014: 946-7).

¹⁴ On the subject of prediction see e.g. Wasserman (2004), Walsh (2014), and Buckholtz and Meyer-Lindenberg (2014).

1 sentencing. For example, sentencing policies could be harsher for people with genetic predispositions to
2 antisocial behavior on the grounds that rehabilitation would be more difficult and recidivism more likely.
3 The law might even be used to attempt to reduce crime rates if it were possible to use genomic scores to
4 predict violent behavior with sufficient accuracy to restrict the liberties of those whose scores were above
5 some level, regardless of behavior to date. So far, prospects for justifying such strategies do not look
6 promising. Buckholtz and Meyer-Lindenberg (2014: 141-142) noted two major limitations of genetically-
7 based prediction. First, “genetic risk for psychiatric disorders and behavioral dispositions is pleiotropic,
8 meaning that heritable factors confer liability to broad dimensions of symptomatically related disorders”,
9 not to mention behavioral tendencies that are not considered disordered. Thus, for example, any putative
10 genetic indicator for antisocial behavior could just as well function as an indicator for “anxiety
11 psychopathology” or any of several other psychopathologies involving quite different specific antisocial
12 behaviors or even behaviors considered heroic such as protecting or rescuing someone by fighting off an
13 attacker or pulling people from burning buildings. Second, as Buckholtz and Meyer-Lindenberg (2014: 142)
14 put it, “most major forms of psychopathology and complex traits are characterized by polygenic
15 inheritance [...], meaning that many small-effect risk alleles [genetic variants] produce a continuous
16 distribution of genetic liability”. Thus, genomic prediction would – if at all – need to function based on
17 polygenic profiles containing many, even thousands of genetic variants, not individual variants or small
18 handfuls of them. It is increasingly clear that even when these profiles can in aggregate predict noticeable
19 proportions of population-level variance, there are so many individual variant combinations of these
20 variants that no single variant combination, as would be carried by any specific individual, could predict
21 anything.

22 Furthermore, and as noted above, the value of potential crime reduction achieved by sentence
23 aggravation or preventive detention on the basis of genomic prediction must be weighed against the value
24 of personal liberty. Therefore, such interventions in the justice system would be highly questionable.
25 Whether or not people have free will, they value living without restrictions imposed on them, and our
26 states are committed to protecting a core range of individuals’ unrestricted activity unless it be forfeited
27 by the individuals themselves. This liberal tenet has already suffered in what is called the ‘war on
28 terrorism’. Still, extended incarceration and preventive detention simply on the grounds of genetic profile
29 would be a hard blow to most states’ basic liberal self-understanding.

30 31 4. Behavior genetics as a challenge to traditional conceptions of agency

1 From the special question of how evidence from behavior genetics should affect legal practice, we turn to
2 the general challenge that such evidence may be supposed to raise for traditional conceptions of agency
3 and agent responsibility. The challenge is this. Traditional conceptions see agents as being “at the helm”:
4 determining what to do in view of the situation obtaining and then doing just that, thereby becoming
5 responsible for having done it. To be sure, people sometimes do things they did not consciously decide to
6 do, perhaps acting from mere habit, perhaps having lost control, in a fit of rage for instance, but no less
7 still acting intentionally, i.e., to further an intentionally held goal. Such cases notwithstanding, the central
8 idea in traditional conceptions of agent responsibility is that of agents as masters of what they do. What
9 we learn from behavior genetics, however, may appear to undermine that idea. The point is not that
10 particular actions, decisions or character traits could be shown to result from a person’s genetic
11 endowment – we argued earlier that there is no warrant for derivations as specific as that, and none is
12 likely forthcoming. The point is rather that the agents themselves thus may seem to dissolve into a
13 multitude of genetic and environmental influences that would leave no room for their being in control of
14 what they do.

15 Yet this will only follow if a more demanding understanding of being in control is assumed than is
16 commonly used in everyday conversation. To be the one who does what one came to see as a good or
17 indeed the best thing to do under the circumstances does not require independence from influences over
18 which one is not master. Here we take issue with a line of argument pressed in particular by Robert Kane
19 who insisted that to understand what people do in terms of free will requires seeing them as ultimate
20 sources of what they do.¹⁵ The requirement has been attacked as unsatisfiable by a number of writers,¹⁶
21 but even before that its motivation is difficult to understand. Why should full agency require agents to be
22 last origins of actions? Why should an action be any less mine for my giving rise to it itself depending on
23 prior factors? The source metaphor is revealing here: something is no less a source, in the ordinary sense,
24 for being fed by the water in the ground. We certainly sometimes do what we decided to do, and in that
25 sense we are in control, whatever goes into the process of making our decisions, which similarly always
26 involves factors internal and external beyond our control. We do not get, but then neither do we need for
27 ordinary agency, self-determination all the way down. Hence, the mere fact that agents became what they
28 are thanks to, among other factors they do not control, ‘stuff’ in their genomes does not undercut their
29 agency. Nor does it undercut their responsibility. Their genomes do not compete with the agents’
30 determining what they do; they do not lead them away from the path which left to themselves they would

¹⁵ Kane (1996: 35). See also Kane (2011).

¹⁶ See, for instance, Wolf (1990: 7-15).

1 have chosen. Their genomes constitute, in part, what the agents are, so there is no appealing to what the
2 agents themselves would have chosen in contrast to what so conditioned they did choose: there are no
3 agents more themselves than these genetically conditioned ones. Thus, there is no reason to see the
4 agents' genetic make-up as replacing the agents themselves "at the helm", and so diminishing their
5 responsibility.

6 This view is compatibilist – in that it entails that free will is compatible with determinism – but it differs
7 from other versions of compatibilism in two respects. First, it accepts Frankfurt's (1969) argument against
8 the principle of alternate possibilities, which states that one is responsible for what one did only if one
9 could have acted otherwise. Second, it does *not* share the widespread concern, originating in Frankfurt's
10 (1971) work, to show the compatibility of freedom and determinism for a demanding sense of freedom,
11 with freedom being "a matter of the unimpaired operation of normally functioning human agency"
12 (McKenna 2011: 177). Admittedly, our stance with respect to both points is controversial. As regards the
13 first, quite a number of writers tried to rebut Frankfurt's attack on the principle of alternate possibilities
14 (see for instance Vihvelin 2013, chap. 4 with further references), and no agreement is in sight here. As
15 regards the second, it has indeed become the prevailing view that the freedom whose compatibility with
16 determinism is at issue needs to be understood in terms of some sort of healthy human functioning,
17 opinions diverging on how to spell out this notion of health, either structurally (e.g. Watson 1975) or by
18 reference to the agent's responsiveness to reasons (e.g. Fischer and Ravizza 1998, Nelkin 2011). We have
19 laid the basis for an argument that a modest understanding of freedom in terms of Hobbesian
20 unhinderedness (Hobbes 1651) suffices, and that in particular it underwrites treating agents' genetic
21 endowment on the lines indicated above. To be sure, this argument still needs to be laid out, and to that
22 extent, what we have presented here remains hypothetical.

24 5. Conclusion

25 Behavior genetic evidence is currently perceived by some courts and some members of the public as a
26 reason to change our judgements of responsibility, and we may expect this perception to continue as new
27 developments in genetics and gene technology emerge. The underlying inference is that genetic influences
28 on behavior undermine agent responsibility by causing individual actions. In its most pronounced form,
29 evidence of genetic influence on behavior is often viewed as entailing some version of determinism. To
30 substantiate the claim that behavior genetic influences should not be assigned this weight we have argued
31 that (i) in the aggregate our genomes influence behavioral tendencies only in the general sense, with
32 context determining the consequences and significance of any particular expression of those tendencies

1 and (ii) their influence on these tendencies is very indirect, with no single variant exerting more than
2 miniscule influence even at that level.

3 Nevertheless, we have identified four important challenges arising from superficial and more
4 deterministic interpretations of behavior genetic evidence, which any legal system must still address
5 because they arise with some frequency. These challenges lie in possibilities that people think that (i)
6 application of existing laws should change based on revised ascriptions of responsibility, (ii) consequent
7 rejection of agency and agent responsibility necessitates a revision of existing laws, (iii) behavior genetic
8 evidence should count as mitigating even if it cannot undermine agent responsibility, and (iv) genomic
9 scores should be used to predict and prevent violent behavior. We conclude that behavior genetic
10 information does not warrant major changes to the law, its application, or crime prevention strategies.

11 However, independently of its application to the law, behavior genetic information may be viewed
12 as calling into question philosophical conceptions of agency and agent responsibility. We have pointed out
13 that even directly causal genetic influences on behavior should not be viewed as having such effects.

Bibliography

- Appelbaum, P. S. (2005):** Behavioral genetics and the punishment of crime. In: *Psychiatric services (Washington, D.C.)*, 56 (1), pp. 25–27.
- Appelbaum, P. S. (2014):** The double helix takes the witness stand: behavioral and neuropsychiatric genetics in court. In: *Neuron*, 82 (5), pp. 946–949.
- Appelbaum, P. S. & Scurich, N. (2014):** Impact of behavioral genetic evidence on the adjudication of criminal behavior. In: *The journal of the American Academy of Psychiatry and the Law*, 42 (1), pp. 91–100.
- Appelbaum, P. S., Scurich, N. & Raad, R. (2015):** Effects of Behavioral Genetic Evidence on Perceptions of Criminal Responsibility and Appropriate Punishment. In: *Psychology, public policy, and law: an official law review of the University of Arizona College of Law and the University of Miami School of Law*, 21 (2), pp. 134–144.
- Aspinwall, L. G., Brown, T. R. & Tabery, J. (2012b):** *Supplementary Materials for: The Double-Edged Sword: Does Biomechanism Increase or Decrease Judges' Sentencing of Psychopaths?*
<http://science.sciencemag.org/content/sci/suppl/2012/08/15/337.6096.846.DC1/1219569.Aspinwall.SM.pdf>.
- Aspinwall, L. G., Brown, T. R. & Tabery, J. (2012a):** The double-edged sword: does biomechanism increase or decrease judges' sentencing of psychopaths? In: *Science (New York, N.Y.)*, 337 (6096), pp. 846–849.
- Baron, M. (2001):** Crime, Genes, and Responsibility. In: Wasserman, D. T. & Wachbroit, R. S. (eds.): *Genetics and criminal behavior*. Cambridge: Cambridge University Press.
- Baum, M. L. (2013):** The Monoamine Oxidase A (MAOA) Genetic Predisposition to Impulsive Violence: Is It Relevant to Criminal Trials? In: *Neuroethics*, 6 (2), pp. 287–306.
- Bernet, W., Vnencak-Jones, C. L., Farahany, N. & Montgomery, S. A. (2007):** Bad nature, bad nurture, and testimony regarding MAOA and SLC6A4 genotyping at murder trials. In: *Journal of forensic sciences*, 52 (6), pp. 1362–1371.
- Blume, J., Johnson, S. & Sundby, S. (2008):** Competent Capital Representation: The Necessity of Knowing and Heeding What Jurors Tell Us About Mitigation. In: *Hofstra Law Review*, 36 (3).
- Bobzien, S. (1998):** *Determinism and freedom in stoic philosophy*. Oxford: Clarendon Press.
- Brown, T. R., Tabery, J. & Aspinwall, L. (2016):** Understanding Validity in Empirical Legal Research: The Case for Methodological Pluralism in Assessing the Impact of Science in Court. In: *Hastings LJ*, 67, pp. 1067–1086.
- Buchanan, A., Brock, D. W. & Wikler, D. (2000):** *From chance to choice: Genetics and justice*, 1. publ. Cambridge u.a.: Cambridge Univ. Press.
- Buckholtz, J. W. & Meyer-Lindenberg, A. (2014):** MAOA and the Bioprediction of Antisocial Behavior: Science Fact and Science Fiction. In: Singh, I., Sinnott-Armstrong, W. & Savulescu, J. (eds.): *Bioprediction, biomarkers, and bad behavior: Scientific, legal, and ethical challenges*. Oxford: Oxford Univ. Press.
- Catley, P. & Claydon, L. (2015):** The use of neuroscientific evidence in the courtroom by those accused of criminal offenses in England and Wales. In: *Journal of law and the biosciences*, 2 (3), pp. 510–549.
- Chabris, C. F., Lee, J. J., Cesarini, D., Benjamin, D. J. & Laibson, D. I. (2015):** The Fourth Law of Behavior Genetics. In: *Current directions in psychological science*, 24 (4), pp. 304–312.
- Coffey, M. (1993):** The Genetic Defense: Excuse or Explanation? In: *William & Mary Law Review*, 35 (1), p. 353.
- Denno, D. W. (2006):** Revisiting the Legal Link Between Genetics and Crime. In: *Law and Contemporary Problems*, 69 (1), pp. 209–258.
- Denno, D. W. (2009):** Behavioral Genetics Evidence in Criminal Cases: 1994–2007. In: Farahany, N. A. (ed.): *The impact of behavioral sciences on criminal law*. New York, NY: Oxford Univ. Press, pp. 317–354.

- 1 **Denno, D. W. (2011):** Courts' Increasing Consideration of Behavioral Genetics Evidence in Criminal Cases: Results of a
2 Longitudinal Study. In: *Michigan State Law Review*, 2011, pp. 967–1047.
- 3 **Denno, D. W. (2013):** What real-world criminal cases tell us about genetics evidence. In: *Hastings LJ*, 64, pp. 1591–
4 1618.
- 5 **Doudna, J. A. & Sternberg, S. H. (2017):** *A crack in creation: Gene editing and the unthinkable power to control*
6 *evolution*: Houghton Mifflin Harcourt.
- 7 **Farahany, N. & Bernet, W. (2006):** Behavioural Genetics in Criminal Cases: Past, Present and Future. In: *Genomics,*
8 *Society & Policy*, 2 (1), pp. 72–79.
- 9 **Farahany, N. & Coleman, J. (2006):** Genetics and Responsibility: To Know the Criminal From the Crime. In: *Law and*
10 *Contemporary Problems*, 69 (1/2), pp. 115–164.
- 11 **Farahany, N. A. (Ed.) (2009):** *The impact of behavioral sciences on criminal law*. New York, NY: Oxford Univ. Press.
- 12 **Farahany, N. A. (2015):** Neuroscience and behavioral genetics in US criminal law: An empirical analysis. In: *Journal of*
13 *law and the biosciences*, 2 (3), pp. 485–509.
- 14 **Feltz, A. & Cova, F. (2014):** Moral responsibility and free will: A meta-analysis. In: *Consciousness and cognition*, 30,
15 pp. 234–246.
- 16 **Feresin, E. (2009):** Lighter sentence for murderer with 'bad genes'. In: *Nature*.
- 17 **Fischer, J. M. & Ravizza, M. (1998):** *Responsibility and control: A theory of moral responsibility*: Cambridge Univ.
18 Press.
- 19 **Frankfurt, H. G. (1969):** Alternate Possibilities and Moral Responsibility. In: *The Journal of Philosophy*, 66 (23),
20 pp. 829–839.
- 21 **Frankfurt, H. G. (1971):** Freedom of the Will and the Concept of a Person. In: *The Journal of Philosophy*, 68 (1), pp. 5–
22 20.
- 23 **Fuss, J. (2016):** Legal responses to neuroscience. In: *Journal of psychiatry & neuroscience : JPN*, 41 (6), pp. 363–365.
- 24 **Gordon, N. & Greene, E. (2018):** Nature, nurture, and capital punishment: How evidence of a genetic-environment
25 interaction, future dangerousness, and deliberation affect sentencing decisions. In: *Behavioral sciences & the law*,
26 36 (1), pp. 65–83.
- 27 **Greene, J. & Cohen, J. (2004):** For the law, neuroscience changes nothing and everything. In: *Philosophical*
28 *transactions of the Royal Society of London. Series B, Biological sciences*, 359 (1451), pp. 1775–1785.
- 29 **Hagerty, B. B. (NPR, Ed.) (2010):** *Can Your Genes Make You Murder?*
30 <https://www.npr.org/templates/story/story.php?storyId=128043329&t=1550138520953> (accessed 14.02.2019).
- 31 **Harrison, P. J. (2015):** Recent genetic findings in schizophrenia and their therapeutic relevance. In: *Journal of*
32 *psychopharmacology (Oxford, England)*, 29 (2), pp. 85–96.
- 33 **Hill, W. G. (2005):** Genetics. A century of corn selection. In: *Science (New York, N.Y.)*, 307 (5710), pp. 683–684.
- 34 **Hobbes, T. (1651/1998):** *Leviathan*. Oxford: Oxford University Press.
- 35 **Hursthouse, R. (1991):** Arational Actions. In: *The Journal of Philosophy*, 88 (2), pp. 57–68.
- 36 **Jablonka, E. & Marion J. Lamb (2014):** *Evolution in Four Dimensions: Genetic, Epigenetic, Behavioral, and Symbolic*
37 *Variation in the History of Life*. Cambridge: The MIT Press.
- 38 **Johnson, M. (1998):** Genetic Technology and Its Impact on Culpability for Criminal Actions. In: *Cleveland State Law*
39 *Review*, 46 (3), p. 443.
- 40 **Kaebnick, G. E. (2006):** Behavioral Genetics and Moral Responsibility. In: Parens, E., Audrey R. Chapman & Nancy
41 Press (eds.): *Wrestling with behavioral genetics: Science, ethics, and public conversation*. Baltimore Md.: Johns
42 Hopkins University Press, pp. 220–234.

- 1 **Kane, R. (1996):** *The significance of free will*. New York: Oxford Univ. Press.
- 2 **Kane, R. (2011):** Rethinking free will: new perspectives on an ancient problem. In: Kane, R. (ed.): *The Oxford handbook*
3 *of free will*, 2. ed. ed. Oxford: Oxford Univ. Press, pp. 381–404.
- 4 **Kane, R. (Ed.) (2011):** *The Oxford handbook of free will* (2. ed.). Oxford: Oxford Univ. Press.
- 5 **Kaye, A. (2004):** Resurrecting the Causal Theory of the Excuses. In: *Neb. L. Rev.*, 83, pp. 1116–1177.
- 6 **Kim, J., Boytos, A., Seong, Y. & Park, K. (2015):** The Influence of Biomedical Information and Childhood History on
7 Sentencing. In: *Behavioral sciences & the law*, 33 (6), pp. 815–826.
- 8 **Knobe, J. (2014):** Free Will and the Scientific Vision. In: Machery, E. & Elizabeth O'Neill (eds.): *Current controversies in*
9 *experimental philosophy*. NewYork, NY: Routledge.
- 10 **Luciano, M., Hagenaaars, S. P., Davies, G., Hill, W. D., Clarke, T.-K. & Shirali, M., et al. (2018):** Association analysis
11 in over 329,000 individuals identifies 116 independent variants influencing neuroticism. In: *Nature genetics*, 50 (1),
12 pp. 6–11.
- 13 **Machery, E. & Elizabeth O'Neill (Eds.) (2014):** *Current controversies in experimental philosophy*. NewYork, NY:
14 Routledge.
- 15 **McKenna, M. (2011):** Contemporary compatibilism: mesh theories and reasons-responsive theories. In: Kane, R. (ed.):
16 *The Oxford handbook of free will*, 2. ed. ed. Oxford: Oxford Univ. Press, pp. 175–198.
- 17 **McSwiggan, S., Elger, B. & Appelbaum, P. S. (2017):** The forensic use of behavioral genetics in criminal
18 proceedings: Case of the MAOA-L genotype. In: *International journal of law and psychiatry*, 50, pp. 17–23.
- 19 **Moore, M. S.:** Causation and the Excuses. In: *California Law Review*, 73, p. 1091.
- 20 **Morse, S. J. (2011b):** Genetics and criminal responsibility. In: *Trends in cognitive sciences*, 15 (9), pp. 378–380.
- 21 **Nahmias, E., Shepard, J. & Reuter, S. (2014):** It's OK if 'my brain made me do it': people's intuitions about free will
22 and neuroscientific prediction. In: *Cognition*, 133 (2), pp. 502–516.
- 23 **Nelkin, D. K. (2011):** *Making sense of freedom and responsibility*. Oxford: Oxford Univ. Press.
- 24 **Nichols, S. & Knobe, J. (2007):** Moral Responsibility and Determinism: The Cognitive Science of Folk Intuitions. In:
25 *Nous*, 41 (4), pp. 663–685.
- 26 **O'Brien, S. (2008):** When Life Depends on It: Supplementary Guidelines for the Mitigation Function of Defense Teams
27 in Death Penalty Cases. In: *Hofstra Law Review*, 36 (3), pp. 693–762.
- 28 **Parens, E., Audrey R. Chapman & Nancy Press (Eds.) (2006):** *Wrestling with behavioral genetics: Science, ethics, and*
29 *public conversation*. Baltimore Md.: Johns Hopkins University Press.
- 30 **Parshley, L. (Popular Science, Ed.) (2016):** *Can Your Genes Make You Kill?: Science's search for the roots of violence*.
31 <https://www.popsci.com/can-your-genes-make-you-kill> (accessed 14.02.2019).
- 32 **Plomin, R. & Deary, I. J. (2015):** Genetics and intelligence differences: five special findings. In: *Molecular psychiatry*,
33 20 (1), pp. 98–108.
- 34 **Prasad, R. B. & Groop, L. (2015):** Genetics of type 2 diabetes-pitfalls and possibilities. In: *Genes*, 6 (1), pp. 87–123.
- 35 **Rommel, R. J., Glenn, A. L. & Cox, J. (2018):** Biological Evidence Regarding Psychopathy Does Not Affect Mock Jury
36 Sentencing. In: *Journal of personality disorders*, pp. 1–21.
- 37 **Scurich, N. & Appelbaum, P. (2016):** The Blunt-Edged Sword: Genetic Explanations of Misbehavior Neither Mitigate
38 Nor Aggravate Punishment. In: *Journal of law and the biosciences*, 3 (1), pp. 140–157.
- 39 **Scurich, N. & Appelbaum, P. S. (2017):** Behavioral Genetics in Criminal Court. In: *Nature human behaviour*, 1,
40 pp. 772–774.
- 41 **Singh, I., Sinnott-Armstrong, W. & Savulescu, J. (Eds.) (2014):** *Bioprediction, biomarkers, and bad behavior:*
42 *Scientific, legal, and ethical challenges*. Oxford: Oxford Univ. Press.

- 1 **Stockard, C. R. (1921):** Developmental rate and structural expression: An experimental study of twins, 'double
2 monsters' and single deformities, and the interaction among embryonic organs during their origin and
3 development. In: *American Journal of Anatomy*, 28 (2), pp. 115–277.
- 4 **Stone, R. D. (2003):** The Cloudy Crystal Ball: Genetics, Child Abuse, and the Perils of Predicting Behavior. In: *Vanderbilt*
5 *Law Review*, 56 (5), p. 1557.
- 6 **Vihvelin, K. (2013):** *Causes, laws, and free will: Why determinism doesn't matter*. Oxford u.a.: Univ. Press.
- 7 **Walsh, C. K. (2014):** Bioprediction in Youth Justice. In: Singh, I., Sinnott-Armstrong, W. & Savulescu, J. (eds.):
8 *Bioprediction, biomarkers, and bad behavior: Scientific, legal, and ethical challenges*. Oxford: Oxford Univ. Press.
- 9 **Wasserman, D. (2004):** Is there value in identifying individual genetic predispositions to violence? In: *The Journal of*
10 *law, medicine & ethics: a journal of the American Society of Law, Medicine & Ethics*, 32 (1), pp. 24–33.
- 11 **Wasserman, D. (2006):** Genetic Predispositions to Violent and Antisocial Behavior: Responsibility, Character, and
12 Identity. In: Parens, E., Audrey R. Chapman & Nancy Press (eds.): *Wrestling with behavioral genetics: Science, ethics,*
13 *and public conversation*. Baltimore Md.: Johns Hopkins University Press, pp. 303–327.
- 14 **Wasserman, D. T. & Wachbroit, R. S. (Eds.) (2001):** *Genetics and criminal behavior*. Cambridge: Cambridge
15 University Press.
- 16 **Watson, G. (1975):** Free Agency. In: *The Journal of Philosophy*, 72 (8), pp. 205–220.
- 17 **Wilder, H. H. (1908):** The morphology of cosmobia; speculations concerning the significance of certain types of
18 monsters. In: *American Journal of Anatomy*, 8 (1), pp. 355–440.
- 19 **Wolf, S. (1990):** *Freedom within reason*. New York: Oxford Univ. Press.
- 20 **Wood, A. R., Esko, T., Yang, J., Vedantam, S., Pers, T. H. & Gustafsson, S., et al. (2014):** Defining the role of
21 common variation in the genomic and biological architecture of adult human height. In: *Nature genetics*, 46 (11),
22 pp. 1173–1186.