CRIMINOLOGY

HERITABILITY STUDIES IN THE POSTGENOMIC ERA: THE FATAL FLAW IS CONCEPTUAL*

CALLIE H. BURT¹ and RONALD L. SIMONS²

¹School of Criminology and Criminal Justice, Arizona State University ²Department of Sociology, University of Georgia

KEYWORDS: heritability, biosocial criminology, postgenomics, epigenetics, plasticity

In the emerging postgenomic paradigm, we are confronted with a biological world that is in many ways the opposite of that which has thus far enabled the methodologies of behavioral genetics. (Charney, 2012: 61)

In our recent article, drawing on advances in the life sciences and echoing the calls of prominent scholars, including renowned behavioral geneticists (e.g., Rutter, 1997; Turkheimer, 2011),¹ we called for an end to heritability studies in criminology and a recognition of the dubious nature of existing heritability estimates (Burt and Simons, 2014).² We argued that heritability studies are futile for two reasons: 1) Heritability studies suffer from serious methodological flaws with the overall effect of making estimates inaccurate and biased toward inflated heritability and deflated shared environmental influences, and *more importantly*, 2) the conceptual biological model on which heritability studies depend—that of identifiably separate effects of genes versus the environment on phenotype variance—is unsound. The aim of our original article was to educate readers about both the (often unacknowledged) methodological assumptions and the (outdated) biological model undergirding heritability studies, evidence that evinces that the heritability study should be superannuated. Our goal was not to foreclose but to *reinvigorate* biological research in criminology by pointing it away from a misguided gene-centric model and, in so doing, highlight recent evidence of developmental plasticity facilitated by the

* Additional supporting information can be found in the listing for this article in the Wiley Online Library at http://onlinelibrary.wiley.com/doi/10.1111/crim.2011.53.issue-1/issuetoc. The authors gratefully acknowledge Kara Hannula, John Laub, D. Wayne Osgood, Alex Piquero, and Sara Wakefield for valuable comments on earlier drafts of this rejoinder. The arguments presented herein are entirely those of the authors and do not reflect the views of those who provided feedback. A longer version of this article is available from the first author. Direct all correspondence to Callie H. Burt, School of Criminology and Criminal Justice, Arizona State University, 411 N. Central Ave, Ste. 600, Phoenix, AZ 85004 (e-mail: chburt@asu.edu).

1. Surprisingly, Wright et al. (2015) question our citing of Turkheimer as calling for an end to heritability studies; this rather captious line of attack, consistent with a "hitting it over the head style" defense, is belied by the facts. For clear quotes repudiating Wright et al.'s claim that Turkheimer is still advocating for the continued use of heritability studies, see Turkheimer (2011: 598; 2014: 532).

2. When it suits their purpose, Barnes et al. (2014) and Wright et al. (2015) incorrectly broaden our critique to behavioral genetics and/or biosocial research generally. We have clearly noted throughout that the heritability study method, including its fallacious technical and conceptual assumptions, is the focus of our critique, not behavioral genetics or biosocial work in general.

dynamic relationship between the biological (including genes) and the environmental (including the social).

Barnes et al. (2014) wrote a response to our article. In their response (and now rejoinder in this issue, Wright et al., 2015), they adopted a strategy common to behavioral geneticists, which Panofsky (2014: 141) called, "hitting them over the head style." This approach involves dodging criticisms by misrepresenting arguments and insinuating that critics are politically motivated and reject scientific truths as well as focusing on a few "tractable' empirical objections" while "ignoring the deeper theoretical objections" (Panofsky, 2014: 152). As Panofsky (2014: 144) noted: "'Hitting them over the head' was a strategy for building scientific capital that involved constructing one's intellectual interlocutors as mortal enemies and attacking them in spectacular, polemical fashion"; "the task was not to seek synthesis, integration, or sober rational persuasion but to engage in polemical scientific attack, declaring themselves as crusaders who would rout the antigenetics heresy gripping behavioral science" (Panofsky, 2014: 142). Consistent with Panofsky's characterization of earlier behavioral geneticists, Barnes et al. and now Wright et al. (2015) question our scientific objectivity, distort our arguments and their implications, and attempt to bury the reader under mounds of data and mathematical simulations, which are all grounded on the flawed biological model at the core of our critique. Unfortunately, we do not have the space to address all of Barnes et al.'s (2014) and Wright et al.'s (2015) distortions of our arguments or the facts. Moreover, we do not wish to question their credentials or motives. Instead, we focus on the science. Specifically, our aim in this brief rejoinder is to correct Barnes et al.'s portrayal of our case against heritability studies—namely, their misguided assertion that the crux of our case against heritability studies rests on the empirical validity of a few select methodological assumptions-and, in so doing, refocus attention on our central claim: Recent advances in the life sciences demonstrate that the foundational *biological* model undergirding heritability studies is invalid, and heritability studies attempt the impossible.

CORRECTION: THE CRUX OF OUR ARGUMENT IS CONCEPTUAL

Barnes et al. (2014) wrongly oversimplified the core of our case against heritability studies as being that violation of several methodological limitations, especially the equal environments assumption (EEA), upwardly bias heritability estimates. This depiction of our argument is seriously misguided but has the advantage of allowing them to claim we merely rehash arguments from the 1970s that have been refuted.³ They are, however, mistaken. Although the EEA is but one of several technical limitations we discussed and the

^{3.} Barnes et al. (2014) briefly attempted to respond to our conceptual critique with a 30-year-old rectangle example that was presented as a rebuttal during the "genomic era" but is not longer apropos given advances in the biosciences. We now know that genetic and environmental influences on variance (area of rectangle) are not analogous to the length (G) and width (E) of a rectangle, unless these are super special rectangles, such that, for example, the width can influence the contribution of the length to the area by turning off, decreasing, or increasing the length's effect. Because Barnes et al. (2014) and Wright et al. (2015) seem to confusedly align our postgenomic arguments with ones from the 1970s, it seems that they think rebuttals from the 1970s speak to our postgenomic critiques. They do not.

debate over the technical soundness of heritability studies remains hotly contested (e.g., Turkheimer, 2014), most importantly, *the crux of our argument against heritability studies is conceptual*. Genes and environments *do not* have identifiably separate influences on variation in complex phenotypes (e.g., crime), and their effects *cannot* be separated. As we noted, it is *not* merely that independent genetic and environments are correlated (as in *r*GE); the problem is much more fundamental. Advances in molecular genomics evince that genes and environments are involved in an interpenetrating and interdependent dynamic relationship that renders the attempt to demarcate separate influences—the goal of heritability studies—illogical at both the individual and population levels.

The burgeoning research in epigenetics, which we discussed in our original article (Burt and Simons, 2014: 248–50), provides perhaps the most visible example of these conceptual breakthroughs. Epigenetic research demonstrates that the traditional focus on the effects of inherited DNA sequences (and protein-coding genes) as causally prior and impervious to the effects of environmental influences is misguided because gene expression (whether a gene is turned on and to what degree) can be influenced by environmental factors in ways that influence behavioral development. Moreover, the same gene can produce multiple (protein) products in response to environmental demands (Kim et al., 2014). Altogether, such transformational evidence reveals that (epi)genomes allow organisms to respond flexibly to environmental signals, facilitating adaptive developmental plasticity the capacity of organisms to modify physiological, morphological, or behavioral phenotypes in response to environmental conditions (Charney, 2012; Griffiths and Stotz, 2013). "One way of beginning to think about epigenetics is to realize that the genome, as much as the organism, is a process rather than a static thing" (Dupré, 2012: 3).

As we elaborated in our article, the foundational biological model underlying heritability methods of a distinct, particulate gene that directs cellular activity and performs one job (produces one protein product) independent of environmental input has been debunked in the past decade (Meloni, 2014b). Genes are now understood to be cellular resources rather than codes in development (Keller, 2010). In response to the falsification of this dichotomous, gene-centric model, a new developmental model that recognizes the permeability and malleability of the genetic in response to the environmental is taking shape. This postgenomic model conceptualizes and studies genes as part of a broader cellular environment that is responsive to social input, with epigenetic factors as important mechanisms whereby life experiences become biologically embedded (Meloni, 2014b). This new understanding of genetic activity is incompatible with the goal of heritability studies. Epigenetic and other postgenomic findings reveal that any attempt to separate "genetic" from "environmental" effects on complex social phenotypes, much less determine their degree of influence, is a futile endeavor.

RESPONSE TO BARNES ET AL.'S METHODOLOGICAL DEFENSE OF TWIN STUDIES

Despite our clear statement that the "most crucial" flaw with heritability studies is the unsound conceptual model (Burt and Simons, 2014: 245–6), Barnes et al. focused their response on demonstrating the validity of the "mathematical framework" of the classic twin study, especially the EEA. (Conspicuously, they did not defend the other heritability models that we criticized.) We find it telling that Barnes et al. largely ignored our critique of other questionable methodological assumptions or practices in twin studies of heritability, which have the general effect of biasing estimates toward inflated heritability, including assumptions of genetic additivity, no/minimal $G \times E$ effects, and the use of kinship pair samples.⁴

In their response, Barnes et al. (2014) ostensibly acknowledged that the EEA is invalid but contended that EEA violations negligibly affect heritability estimates. They attempted to marshal evidence to support their position by identifying a comprehensive list of studies that have examined the effects of EEA violations, in contrast to our focus on EEA violations for shared environmental factors related to *criminal* phenotypes.⁵ Instructively, despite what Barnes et al. alleged, the evidence we cited from such sources as Cronk et al. (2002), which tested for EEA violations, was correctly cited as showing EEA violations. In short, we contended (see our original article, Burt and Simons, 2014) that research clearly demonstrates that the EEA is untenable for criminal phenotypes; that EEA violations, along with violations of other technical assumptions, bias heritability upward to some unknown degree; and that some absurdly high heritability estimates are due in part to such technical flaws in the model. Barnes et al. disagreed. Fortunately, the evidence is available for readers to view to come to their own conclusions. At a minimum, this evidence should be sufficient for readers to recognize that Barnes et al.'s (2014) claim that these methodological debates have long been "settled" and the validity of these assumptions "proven" is patently false.

Barnes et al. (2014) faulted our critique for not including a "mathematical demonstration" of the consequences of violations of the flaws of the model, and they attempted to sway the reader with simulations and appeals to the incontrovertibility of algebra. Importantly, however, the core of our critique is not statistical or algebraic. Barnes et al.'s simulations rest on a conceptual model that is simply not viable. No amount of mathematical simulations, no matter how sophisticated, can align the heritability study model with the biological fact of an interpenetrating and interdependent relationship between genes and environments.

While we could get embroiled in a protracted debate over the methodological flaws of heritability studies, we do not have the space (or desire) to do so. Ultimately, in our view, given that the foundational biological model of heritability studies has been shown to be false, the technical limitations of the twin study are rather immaterial, and further wrangling over the methodological assumptions and statistical minutiae of heritability studies

^{4.} We would like to correct the record that we did not call the results of heritability studies "preposterous" as Barnes et al. (2014) claimed. Instead, we called the assumption that the environments of opposite-sex cousins are equally similar to monozygotic twins "preposterous" (Burt and Simons, 2014: 236), and we continue to believe that is an apposite descriptor. Notably, they did not defend the use of kinship samples.

^{5.} Wright et al. (2015) stated: "Notably, we found that they failed to cite any of the more than 60 studies that tested for violations of the EEA—a body of work that is inconsistent with their claims." This is false. We included studies that tested for EEA violations (e.g., Cronk et al., 2002; Horwitz et al., 2003), and they identified these studies in their table (Barnes et al., 2014: Appendix D table) and discussed them in the text.

is a waste of scientific energy and attention.^{6,7} *Regardless* of the validity of the EEA or other technical assumptions, the fact remains that the conceptual model is unsound and the goal of heritability studies is biologically nonsensical given what we now know about the way genes work.

GCTA STUDIES AND SOCIAL PHENOTYPES: LET'S CALL THE WHOLE THING OFF

As Barnes et al. (2014) discussed, over the past few years scientists have developed a new methodology for identifying gene variants in human populations called genome-wide complex trait analysis (GCTA).⁸ We dispute Barnes et al.'s contention that GCTA studies provide convergent support for the validity of standard heritability studies. Perhaps because the findings are more consistent with their argument, they focused on a GCTA study of height—a physical phenotype. Importantly, to our knowledge, every GCTA study of complex social phenotypes that are relevant to antisocial behavior has identified small-to-nonexistent heritability estimates, even when finding substantial twin-study estimates within the same sample (e.g., Trzaskowski, Dale, and Plomin, 2013; Trzaskowski et al., 2013; Viding et al., 2013). Thus, contrary to the contention of Barnes et al., GCTA studies provide further evidence that something is amiss in the high standard heritability estimates of criminal phenotypes.

Importantly, however, like standard heritability studies, GCTA studies suffer from significant methodological problems that augment the likelihood of flawed estimates due to spurious genetic associations, such as population stratification (Charney, 2013). More importantly, GCTA studies, like other heritability models, rest on the outdated, false gene versus environment dichotomy. To repeat, the lack of scientific merit in heritability studies is not based on technical limitations that can be improved over time or can be addressed with new cutting-edge techniques. Restating Lewontin's (1974: 411) 40-year-old statement that remains quite apt: "[we] suggest we stop the endless search for better methods of estimating useless quantities. There are plenty of real problems."

POLITICAL SENSITIVITIES AND VIGILANT SCIENCE

Barnes et al. (2014) began their response in standard "hitting them over the head style" by situating our critique among politically motivated (possibly "anti-science") scholarship that is "ideologically opposed to biology," alleging that we wish to "censor" findings that suggest that biology has anything to do with behavior. This is false. We are not opposed

^{6.} While we think the technical/statistical limitations are rather immaterial given the method's unsound conceptual foundation, we realize that some may disagree. As such, we revisited the evidence we cited, and we stand by our interpretations of the evidence as demonstrating significant EEA violations for criminal phenotypes. Although we discourage readers from getting bogged down in these technical issues, our more detailed response to Barnes et al.'s (2014) EEA defense can be found in the online supporting information.

^{7.} Additional supporting information can be found in the listing for this article in the Wiley Online Library at http://onlinelibrary.wiley.com/doi/10.1111/crim.2011.53.issue-1/issuetoc.

^{8.} See the online supporting information for more information on these GCTA studies and methodological limitations of the GCTA method.

to research that recognizes a biological influence on behavior. Indeed, we consider ourselves biopsychosocial scientists, and we engage in research that incorporates biological influences (susceptibility and pathways) on social behaviors (e.g., Burt, Sweeten, and Simons, 2014; Simons et al., 2011). This is not a "war" between biosocial criminologists and environmental determinists. This is a scientific discussion about the validity of a specific method, which includes both technical/statistical and conceptual assumptions, that we, among others, believe evidence clearly demonstrates is "fatally flawed" and lacking utility.⁹ Drawing on cutting edge *biological* research, not ideological objections, we argue that heritability studies do not advance scientific knowledge of complex social behaviors.¹⁰ As Charney (2008: 340–1) averred: "To point out the flaws in a supposedly scientific methodology (twin studies), to point out that it fails to meet the rigorous criteria of scientific knowledge is hardly to reject the scientific method but to uphold it."

Although ours was not a politically motivated critique, we are cognizant of the political sensitivities surrounding this line of research. The possibility that unpleasant facts may result from a line of inquiry does not mean that it should be ignored or findings censored. Political or social sensitivities should not disrupt scientific progress. However, as Pinker noted (2011: 614): "The fact that a hypothesis is politically uncomfortable does not mean that it is false, but it does mean that we should consider the evidence very carefully before concluding that it is true." History is replete with examples of enormous human abuses justified in part by (later determined) flawed scientific research linking biology to crime or other deviant behaviors (Rafter, 2008). There is nothing unscientific about acknowledging this potential danger and proceeding carefully (Orr, 2014). At times, however, many biosocial criminologists seem to take the opposite approach while intimating that they alone are scientific enough to face politically sensitive scientific questions. For example, in a recent chapter by Wright and Morgan (2014) arguing for the biological basis of race and challenging the "egalitarian fiction" (specifically implying that Blacks, as a biological [racial] category, have higher rates of offending in part due to genetic differences; p. 58), they stated:

[B]iosocial criminologists are a unique breed—no pun intended. As a group they are contrarian to the core. By this we mean that they question every theory, every postulate, and every finding. ... Simply put, biosocial criminologists are skeptical of entire bodies of research in part because they believe much of it to be misspecified, or worse yet, infused with disciplinary or political bias. (p. 55)

^{9.} Barnes et al. (2014) claimed that we fail to appreciate the benefits of heritability studies for modern behavioral genetics research. We counter that we fail to appreciate the benefits because *they do not exist*. While space constraints did not allow us to respond in the text, we do have a response. Interested readers can see our brief response in the online supporting information.

^{10.} In their rejoinder, Wright et al. (2015) mischaracterize our arguments as saying that "nothing positive has come out of behavioral genetic research" and point to a few examples in which biosocial work "led to ... more humane treatment of individual differences." First, we at no point state that "nothing" positive has come out of behavioral genetic research (for more on this, see the Santa Claus analogy in the online supporting information). We criticize heritability studies and their lack of utility *at the present time*. Second, we are clear in that we are *not* criticizing biosocial work generally, and these examples *are not at all related to heritability studies*. Linking these examples to the utility of heritability studies is totally misleading.

Organized skepticism *is* a core value in science. Scientists *should* question results until they are convinced that they have rooted out error and bias, and in our view, heritability study scholars have not applied this organized skepticism to their own models. This may be an acceptable way of conducting science in a different world, one without a history of eugenics and pervasive racism, sexism, heterosexism, and other 'isms. To be sure, our attention to the methodological and conceptual flaws in heritability studies may seem relatively uncommon, hence, the conclusion by Barnes et al. (2014) that our critique was politically motivated. We believe, however, that in this politically sensitive realm such organized skepticism is certainly justified.

CONCLUSION

"[O]ur knowledge grows only through the correction of our mistakes" (Popper, 1965: xvi).

Our goal in this rejoinder was to reiterate and refocus attention on the arguments we made in our original article (Burt and Simons, 2014). Among many, we contend that heritability studies have serious methodological flaws that render their estimates highly ambiguous and biased toward inflated heritability, but more importantly, heritability studies rest on an unsound conceptual (biological) model that assumes identifiably separate "genetic" and "environmental" effects on phenotypic variance. These glaring fatal flaws together led us to call for an end to heritability studies in criminology and recognition of the problematic nature of existing heritability estimates.

Barnes et al. (2014) responded to our article by focusing on the methodological critique of twin studies ("tractable' empirical objections"), especially the EEA, and attempted to defend the heritability study method in the postgenomic era while ignoring our fundamental conceptual objections. To be sure, we are disappointed that Barnes et al. did not engage with our conceptual critique, but not surprised. Given that recent advances in molecular genomics debunk central biological assumptions that undergird heritability studies, the only way to justify heritability studies is to ignore or dismiss this evidence (as "fanciful," for example). Heritability studies rely on a conception of genetic influences uninformed by basic principles of genetics and by advances in molecular genomics in recent decades. Although such a deterministic model of genetic function may be ideally suited for heritability studies, it is chimerical (Charney and English, 2013).

As we have noted, Barnes et al.'s (2014) implication that we are politically motivated critics who are ideologically opposed to biological influences is specious. We are not. We are, however, strongly dissatisfied with the *way* biology is currently being invoked in biosocial criminology (as exemplified in heritability studies). Much biosocial criminology is increasingly out of touch with the recent advances in the biosciences that recognize the permeability of the biological, including the epigenome, to social conditions and life experiences. For all the lip service paid to interactionism, biosocial criminology is still largely situated in an outdated model of the biological (as a fixed, preexisting resource) and genetic (as a context-independent, fundamental cause of development), which makes it highly problematic to import this work into mainstream criminology in any serious way (Meloni, 2014a). In short, biosocial criminology is hopelessly stalled by the use of outdated concepts and models to confront a much more complicated, interactive reality to which they clearly do not apply.

This matters for criminology. We are at an important point where the intersection between biology and the social is ripe for research and theorizing (e.g., Landecker and Panofsky, 2013; Meloni, 2014b). Clearly, ours is not a call for criminologists to neglect biological or environment influences because their combined effects are "irreducibly complex"¹¹; this is a call for biosocial criminology *to engage with current bioscientific knowledge*.¹² Rejecting heritability studies and the false nature–nurture dichotomy and gene-centric model on which they are grounded is a necessary step forward that will pave the way for a reconceptualization of the link between the biological and the social in shaping criminal propensities in ways that are consistent with postgenomic knowledge.

As scientists we value the scientific method and believe it is important to hold scientific research—especially high-profile research that has the potential to significantly influence social understanding and public policy—to the highest standards. Heritability studies have gained a lot of attention in criminology in recent years based in part on the notion that they are supported by rigorous, cutting-edge science. There is value in showing how this work does not measure up to that billing. Although we find fault in heritability studies and the gene–environment dichotomy, we hope that those who read our critique take it in the spirit of constructive criticism in which it was offered. It is our hope that our critique advances criminology.

REFERENCES

- Barnes, J. C., John Paul Wright, Brian B. Boutwell, Joseph A. Schwartz, Eric J. Connolly, Joseph L. Nedelec, and Kevin M. Beaver. 2014. Demonstrating the validity of twin research in criminology. *Criminology* 52:588–626.
- Burt, Callie H., and Ronald L. Simons. 2014. Pulling back the curtain on heritability studies: Biosocial criminology in the postgenomic era. *Criminology* 52:223–62.
- Burt, Callie H., Gary Sweeten, and Ronald L. Simons. 2014. Self-control through emerging adulthood: Instability, multidimensionality, and criminological significance. *Criminology* 52:450–87.
- Charney, Evan. 2008. Genes and ideologies. Perspectives on Politics 6:299-319.
- Charney, Evan. 2012. Behavior genetics and postgenomics. *Behavioral and Brain Sciences* 35:331–410.
- Charney, Evan. 2013. Still chasing ghosts: A new genetic methodology will not find the "missing heritability." *Independent Science News*. http://www.independentsci encenews.org/health/still-chasing-ghosts-a-new-genetic-methodology-will-not-findthe-missing-heritability/.
- Charney, Evan, and William English. 2013. Genopolitics and the science of genetics. *American Political Science Review* 107:382–95.
- Cronk, Nikole J., Wendy S. Slutske, Pamela A. F. Madden, Kathleen K. Bucholz, Wendy Reich, and Andrew C. Heath. 2002. Emotional and behavioral problems among female twins: An evaluation of the equal environments assumption. *Journal of the American Academy of Child & Adolescent Psychiatry* 41:829–37.

^{11. &}quot;Irreducible complexity" is the caricature of our argument that Wright et al. (2015) employ.

^{12.} See our original article (Burt and Simons, 2014) for our suggestions for how biopsychosocial criminology might proceed in a way informed by postgenomic knowledge.

- Dupré, John. 2012. Processes of Life: Essays in the Philosophy of Biology. Oxford, U.K.: Oxford University Press.
- Griffiths, Paul, and Karola Stotz. 2013. *Genetics and Philosophy: An Introduction*. Cambridge, U.K.: Cambridge University Press.
- Horwitz, Allan V., Tami M. Videon, Mark F. Schmitz, and Diane Davis. 2003. Rethinking twins and environments: Possible social sources for assumed genetic influences in twin research. *Journal of Health and Social Behavior* 44:111–129.
- Keller, Evelyn Fox. 2010. *The Mirage of a Space between Nature and Nurture*. Durham, NC: Duke University Press.
- Kim, Min-Sik, Sneha M. Pinto, Derese Getnet, Raja Sekhar Nirujogi, Srikanth S. Manda, et al. 2014. A draft map of the human proteome. *Nature* 509: 575–581.
- Landecker, Hannah, and Aaron Panofksy. 2013. From social structure to gene regulation and back: A critical introduction to environmental epigenetics and society. *Annual Review of Sociology* 39:333–57.
- Lewontin, Richard C. 1974. The analysis of variance and the analysis of causes. *American Journal of Human Genetics* 26:400–11.
- Meloni, Maurizio. 2014a. Biology without biologism: Social theory in a postgenomic age. *Sociology* 48:731–46.
- Meloni, Maurizio. 2014b. The social brain meets the reactive genome: Neuroscience, epigenetics, and the new social biology. *Frontiers in Human Neuroscience* 8:1–11.
- Orr, H. Allen. 2014. Book review: Stretch genes. *The New York Review of Books* 61:10. http://www.nybooks.com/articles/archives/2014/jun/05/stretch-genes/?insrc=toc.
- Panofsky, Aaron. 2014. *Misbehaving Science: Controversy and the Development of Behavior Genetics*. Chicago, IL: University of Chicago Press.
- Pinker, Steven. 2011. *The Better Angels of Our Nature: Why Violence Has Declined*. New York: Penguin Books.
- Popper, Karl. 1965. *Conjectures and Refutations: The Growth of Scientific Knowledge*, 2nd ed. London: Routledge.
- Rafter, Nicole. 2008. *The Criminal Brain: Understanding Biological Theories of Crime*. New York: New York University Press.
- Rutter, Michael L. 1997. Nature-nurture integration: The example of antisocial behavior. *American Psychologist* 52:390–8.
- Simons, Ronald L., Man Kit Lei, Steven R. H. Beach, Gene H. Brody, Robert A. Philibert, and Frederick X. Gibbons. 2011. Social environment, genes, and aggression: Evidence supporting the differential susceptibility perspective. *American Sociological Review* 76:883–912.
- Trzaskowski, Maciej, Philip S. Dale, and Robert Plomin. 2013. No genetic influence for childhood behavior problems from DNA analysis. *Journal of the American Academy of Child and Adolescent Psychiatry* 52:1048–56.
- Trzaskowski, Maciej, Thalia C. Eley, Oliver S. P. Davis, Sophia J. Doherty, Ken B. Hanscombe, Emma L. Meaburn, Claire M. A. Haworth, Thomas Price, and Robert Plomin. 2013. First genome-wide association study on anxiety-related behaviors in childhood. *PLoS ONE* 8:1–7.
- Turkheimer, Eric. 2011. Commentary: Variation and causation in the environment and genome. *International Journal of Epidemiology* 40:598–601.
- Turkheimer, Eric. 2014. A phenotype null hypothesis for the genetics of personality. *Annual Review of Psychology* 65:515–40.

- Viding, Essi, Thomas S. Price, Sara R. Jaffee, Maciej Trzaskowski, Oliver S. P. Davis, Emma L. Meaburn, Claire M. A. Haworth, and Robert Plomin. 2013. Genetics of callous-unemotional behavior in children. *PLoS ONE* 8:1–9.
- Wright, John Paul, J. C. Barnes, Brian B. Boutwell, Joseph A. Schwartz, Eric J. Connolly, Joseph L. Nedelec, and Kevin M. Beaver. 2015. Mathematical proof is not minutiae and irreducible complexity is not a theory: A final response to Burt and Simons and a call to criminologists. *Criminology* 53:113–20.
- Wright, John Paul, and Mark Alden Morgan. 2014. Human biodiversity and the egalitarian fiction. In *The Nurture Versus Biosocial Debate in Criminology*, eds. Kevin M. Beaver, J.C. Barnes, and Brian B. Boutwell. Thousand Oaks, CA: Sage.

SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article at the publisher's web site:

S.1. Detailed Response to Barnes et al.'s Methodological Defense of Twin Studies

S.2. More Detail: GCTAs and Social Phenotypes: Let's Call The Whole Thing Off **S.3.** Heritability Studies Lack Utility