Chapter 6
Psychopathy: Neurohype and Its Consequences

Jarkko Jalava and Stephanie Griffiths

Abstract Many argue that psychopaths suffer from a stable pattern of neurobiological dysfunctions that should be taken into account in sentencing and treatment decisions. These arguments are compelling only if the neuroimaging data are consistent. It is possible that such consistency is created by reviewers who ignore contradictory findings. To evaluate this, we examined how accurately forensic literature reported neuroimaging findings on psychopaths in a theoretically central structure – the amygdala. We found that forensic commentators consistently under-reported null-findings, creating a misleading impression of the data's consistency. We discuss this misrepresentation from the perspectives of spin and neurohype, and examine their causes and consequences.

Keywords Psychopathy · Psychopathy Checklist – Revised · Neuroimaging · Spin · Neurohype · Bias · Forensic · MRI

Psychopathy is often described as a brain-based disorder. In the scientific literature psychopathy is frequently called a "neurodevelopmental" or "neuropsychiatric" disorder (Anderson & Kiehl, 2014, p. 103; Gao et al., 2009, p. 813; Sethi et al., 2018, p. 88) or some variant thereof, and titles such as *the neurobiology of psychopathy* (Cummings, 2015; Gao et al., 2009; Glenn & Raine, 2008; Herba et al., 2007; Stratton et al., 2015) are common. The first contemporary neuroimaging studies on psychopathy were conducted in the early 2000s; by 2002, one leading researcher told an interviewer that psychopathy is "definitely a biologically based condition in the sense that the amygdala is functioning poorly" (Blair, quoted in Purdie, 2002). "They're wired differently than other people" explained another (Raine, quoted in University of Southern California, 2004). "The consistency of their brain abnormalities" a prominent psychopathy researcher noted, "never ceases to amaze me" (Kiehl, 2014, p. 262).

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L. Malatesti et al. (eds.), *Psychopathy*, History, Philosophy and Theory of the Life Sciences 27, https://doi.org/10.1007/978-3-030-82454-9_6

This apparent consensus naturally raised the question of what to do with psychopaths. Many argued for reduced moral or criminal responsibility (e.g., Blair, 2008; Knabb et al., 2009). Some maintained that psychopaths should not be held criminally responsible at all (e.g., Glenn et al., 2011). Others offered treatment suggestions, ranging from positive reinforcement (e.g., Reidy et al., 2015) to neurosurgery (De Ridder et al., 2009). Most agreed that psychopaths should not be punished in the same way as those without neurobiological deficits. A philosopher explained that "psychopathy is a mental illness with bio-genetic etiology …. At this point, this is something everyone in the debate about the philosophy of punishment should simply accept" (Nadelhoffer, 2015, pp. 174–175). In 2009, a team of researchers imaged American serial killer Brian Dugan's brain, and presented the results as mitigating evidence in his sentencing hearing. Dugan, the defense argued, suffered from "an emotional developmental disorder (i.e., psychopathy)" (Gaudet et al., 2014, p. 44), and the court found that the testimony passed the *Frye* test for admissibility.[1] Judges have admitted neurobiological evidence of psychopathy in other cases as well (Denno, 2015).

But the consistency of the neurobiological data on psychopaths seemed to depend on who one asked and how closely one scrutinized the data. Popular media articles, which usually included interviews with leading psychopathy researchers, tended to treat the neurobiology of psychopathy as a scientific fact (e.g., Bhattacharjee, 2018; Hagerty, 2017; Ridley, 2018). "We have a fairly good idea," an article in *The Atlantic* explained "what an adult psychopathic brain looks like" (Hagerty, 2017). Researchers cited in these stories often had neurobiological theories of their own, theories the researchers argued were supported by the data (e.g., Anderson & Kiehl, 2012, 2014; Gao et al., 2009; Glenn & Raine, 2014; Kiehl, 2006; Umbach et al., 2015).

Reviews by authors without theoretical commitments painted a different, though not always cohesive, picture. Most reviews by non-theorists tended to find the data inconsistent (Brook et al., 2013; Griffiths & Jalava, 2017; Koenigs et al., 2011; Pujara & Koenigs, 2014; Santana, 2016; but also see also Del Casale et al., 2015). One meta-analysis, however, found consistent abnormalities in a number of brain regions [Poeppl et al., 2018; this finding contrasted with an earlier meta-analysis (Yang & Raine, 2009), which did not find abnormalities].

Regardless of theoretical inclinations, all writers who found the data consistent shared one thing in common: they included few or no null-findings. The meta-analysis above, for instance, reported on 753 findings from 155 studies, with no nulls (Poeppl et al., 2018). The most-cited review studies by the most-cited psychopathy researchers (Blair, 2003; Gao & Raine, 2010; Kiehl, 2006) included findings from a combined seventeen different sMRI, fMRI and SPECT studies. Together, they described no disconfirming null findings (the only nulls they reported were to distinguish between factor scores, successful and unsuccessful psychopaths, and

[1] The court did not allow actual fMRI images to be shown, however, but did allow general diagrams of the brain.

white and grey matter). The absence of null-findings from data-sets this large is statistically improbable: Even if a neural profile for psychopathy did exist, null findings should still occur by chance. For one, neither neuroimaging methodology nor personality measures, including psychopathy measures, are reliable enough to produce consistent and/or high correlations (see e.g., Vul et al., 2009). When we tabulated all findings in published neuroimaging studies between 1997 and 2017 using the most common definition of psychopathy (PCL-R, the same definition all but one study in the review above studies also used) we found that the most consistent findings were, in fact, nulls (Griffiths & Jalava, 2017). In other words, those writers who argued that the data showed consistent abnormalities had done something akin to evaluating baseball batters by counting the number of safe hits and where the ball had landed while, mostly, leaving out the times the batters had swung and missed.

If there is no neural profile of psychopathy, it is unclear whether psychopaths share anything in common aside from their diagnosis. This is because the evidence for theoretically relevant deficits in psychopaths, such as in emotion (Brook et al., 2013), moral judgment (Marshall et al., 2016), experience of fear (Hoppenbrouwers et al., 2016), and attention (Smith & Lilienfeld, 2015) is also relatively inconsistent, and in some cases possibly skewed by publication bias (Smith & Lilienfeld, 2015). These inconsistencies may be due to the way psychopathy is diagnosed: According to one calculation, the 30 item PCL-R (with 3 possible levels of severity for each item) allows for more than 15,000 different symptom combinations at or above the diagnostic cutoff score (Rogers, 1995).

If lack of clarity on psychopaths' neurobiology is a problem for basic science, it is an even larger problem for applied science. Incomplete or biased reviews of the data can lead readers to misconstrue the state of the science. Consequences are much worse if the data are used to determine how we should deal with actual people. It is for this reason that applied arguments require a higher burden of proof than those of pure science – consider for example the different levels of proof required for scientific claims in a single drug study and the acceptance of that drug for medical use.

How, then, does applied literature on psychopathy approach the data on psychopaths' neurobiology? Do philosophers, lawyers, and social scientists concerned with things such as criminal responsibility and punishment exercise caution traditionally expected of applied science or do they engage in what is commonly known as *neurohype* – the making of exaggerated claims about neuroscientific data? (see e.g., Lilienfeld et al., 2018). Exactly what kinds of conclusions do applied writers draw from neuroimaging data on psychopaths?

6.1 Reporting Accuracy

We reviewed applied arguments about how the criminal justice system should deal with psychopaths by searching PsycINFO, PubMed, and Google Scholar for published literature using the terms "psychopathy and responsibility" and

"psychopathy and punishment." We also examined sources cited in these publications for anything our first search missed. Overall, we found 65 publications since 2000, the year the first MRI study on psychopaths was published (Raine et al., 2000). Of these, 85% argued for one kind of application or another, the most common of which concerned legal responsibility (reduced criminal responsibility, no criminal responsibility or, more generally, that the empirical evidence should somehow affect responsibility; we did not include publications that argued about moral responsibility only). Other proposed applications involved punishment, treatment, crime prediction, and preventive detention. The arguments showed varying levels of prescriptiveness; some called for immediate application while others only hinted at the possibility of application and offered caveats. Overall, 80% of the publications (52 out of 65) cited neuroimaging evidence for their conclusions (see Table 6.1). The vast majority cited individual studies, a few cited reviews by psychopathy theorists only, and a single paper cited a critical review study by non-theorists (Table 6.1).

Given that most articles focused on single-study findings, we evaluated the accuracy of data reporting in the applied literature. To do this, we compared the narrative description of neuroimaging data in the 52 applied articles to the empirical findings in the published studies. We focused on the amygdalae, one of the most widely studied and theoretically important brain regions in psychopathy (e.g. Blair, 2010). The amygdaloid bodies contain many specialized nuclei that are important in processes such as emotions (especially fear) and motivated behavior. These structures have obvious theoretical relevance to psychopathy as psychopathy includes symptoms such as callousness, shallow affect, and antisocial behavior. We focused on the most common diagnostic measure, the PCL-R (Hare, 2003), widely considered the 'gold standard' in the field (e.g., Glenn & Raine, 2008).

Of the 52 publications in our sample that argued for application based on neuroimaging evidence, most authors cited more than one primary neuroimaging study to support their arguments (see Table 6.2). None of the arguments rested exclusively on neuroimaging of the amygdala – other neurobiological data were cited as well. We only included publications that cited original research (i.e., not review studies) on adult psychopaths, and publications that clearly stated the direction of the findings (+ = increased structure or function, – = decreased structure or function, or 0 = null finding, bilaterally or in either right or left amygdala). This allowed us to evaluate whether the narrative description of the neuroimaging findings of the amygdalae were congruent with the original research findings themselves.

The original findings are summarized in Table 6.3 (modified from Griffiths & Jalava, 2017; also includes PCL-SV, Hart et al., 1995 screening version of the PCL-R). Roughly half of the original neuroimaging studies, both structural and functional, found no differences between psychopaths and non-psychopaths. The remaining studies showed either reduced structure and function or increased structure and function in psychopaths. Therefore, if the applied literature accurately described neuroimaging in psychopathy, null findings should feature prominently. However, across all of the specific neuroimaging effects described in the applied literature, only three were null results (3% of the total 108 effects described in Table 6.2). Since one of these effects was in reality *not* a null finding, the applied

Table 6.1 Summary of applied literature on psychopathy and neuroimaging

Authors	Primary conclusion[a]	Neuroimaging Y/N
N articles included = 65	*N* articles arguing for application = 55 *(85%)*	*N* citing neuroimaging = 52 *(80%)*
Anderson and Kiehl (2013)	CR	Y
Anderson and Kiehl (2014)	TR	Y
Baccarini and Malatesti (2017)	BIO	Y
Baskin-Sommers and Newman (2012)	TR	Y
Berryessa (2016)	CR	Y
Blair (2008)	CR	Y
Brink and Nelkin (2013)	CR	Y
Canavero (2014)	BIO	Y
Chialant et al. (2016)	TR	Y
Ciocchetti (2003)	CR	N
Corrado (2013)	PUN	N
DeLisi et al. (2009)	TR	Y
De Ridder et al. (2009)	BIO	Y
Dressing et al. (2008)	PUN	Y
Duff (2010)	CR	N
Fabian (2010)	CR	Y
Fine and Kennett (2004)	CR	Y[b]
Finlay (2011)	CR	N
Fischette (2004)	CR	N
Focquaert et al. (2013)	CR	Y
Focquaert et al. (2015)	CR	Y
Fox et al. (2013)	CR	Y
Freedman and Verdun-Jones (2010)	CR	Y
Fumagalli and Priori (2012)	CR	Y
Glannon (2008)	CR	Y
Glenn and Raine (2009a)	PUN	Y
Glenn and Raine (2009b)	TR, PREV	Y
Glenn and Raine (2014)	CR	Y
Glenn et al. (2011)	CR	Y
Glenn et al. (2013)	CR7	Y
Haji (2003)	PUN	N
Haji (2017)	CR	N
Hauser (2016) Jurjako and Malatesti (2018a)	CR NLA	Y Y
Jurjako and Malatesti (2018b)	NLA	Y
Knabb et al. (2009)	CR	Y

(continued)

Table 6.1 (continued)

Authors	Primary conclusion[a]	Neuroimaging Y/N
Koenigs and Newman (2013)	CR	Y
Levy (2007)	CR	Y
Levy (2011)	PUN, PREV	Y
Levy (2014)	CR	N
Ling and Raine (2018)	CR	Y
Litton (2008)	UN	N
Litton (2013)	UN	N
Maibom (2008)	NLA	N
Marazziti et al. (2013)	CR	Y
Mei-Tal (2002)	CR	Y
Mendez (2009)	CR	Y
Morse (2008)	CR	N
Nadelhoffer (2015)	CR	Y
Nadelhoffer et al. (2012)	PRED	Y
Nair and Weinstock (2008)	CR	Y
O'Neill (2004)	PUN	Y
Ortega-Escobar et al. (2017)	CR	Y
Patrick et al. (2012)	NLA	Y
Poldrack et al. (2018)	PRED, PUN, etc.	Y
Reidy et al. (2015)	PREV	Y
Sartori et al. (2011)	TR	Y
Schopp and Slain (2000)	NLA	N
Shaw (2016)	CR	Y
Sifferd and Hirstein (2013)	CR	Y
Skeem et al. (2011)	NLA	Y
Umbach et al. (2015)	CR	Y
Vierra (2016)	CR	Y
Vitacco et al. (2013)	NLA	Y
Weber et al. (2008)	CR	Y

[a]CR = at least some bearing on criminal responsibility (no criminal responsibility, reduced responsibility, etc.); *BIO* neurobiological interventions, *TR* treatment/assessment, *PUN* punishment and/or sentencing, *PREV* prevention (including preventive detention), *PRED* prediction/neuroprediction, *NLA* data do not support legal application, *UN* undecided

[b]Cited data from individuals with prefrontal injuries, but not psychopaths

literature identified two 'true' nulls (and of these, one was only to distinguish successful from non-successful psychopaths). The applied literature mostly cited findings of one type: reduced structure or function (88% and 86% of all neuroimaging effects described, respectively). In other words, the applied studies' summaries of the neuroimaging data were consistently inaccurate (see Table 6.3).[2] The applied

[2] Null findings have been present since 2001. Their absence from the applied literature, therefore, is not a function of their unavailability for early publications.

Table 6.2 Neuroimaging effects cited in applied literature: Amygdala

Authors	Studies cited sMRI fMRI – Italicized	Effect	Suggested application
Anderson and Kiehl (2013)	Kiehl et al. (2001) Birbaumer et al. (2005) Veit et al. (2002) Harenski et al. (2010) Dolan and Fullam (2009) Müller et al. (2003)	– – – – –, + +	Legal responsibility
Anderson and Kiehl (2014)	Ermer et al. (2012) Yang et al. (2010) Kiehl et al. (2001) Birbaumer et al. (2005) Veit et al. (2002) Harenski et al. (2010) Dolan and Fullam (2009)	– – – – – – –	Treatment
Baskin-Sommers and Newman (2012)	Birbaumer et al. (2005) Glenn et al. (2009) Kiehl et al. (2001) Müller et al. (2003)	– – – +	Treatment
Blair (2008)	Kiehl et al. (2001) Birbaumer et al. (2005)	– –	Reduced responsibility
Chialant et al. (2016)	Kiehl et al. (2001)	–	Punishment, prevention, treatment
De Ridder et al., (2009)	Kiehl et al. (2001)	–	Neurosurgery
Fabian (2010)	Kiehl et al. (2001)	–	Mitigation, moral culpability, punishment
Focquaert et al. (2013)	Yang et al. (2009) Kiehl et al. (2001) Birbaumer et al. (2005)	– – –	Punishment
Focquaert et al. (2015)	Yang et al. (2009) Kiehl et al. (2001) Glenn et al. (2009)	– – –	Criminal responsibility, treatment
Fumagalli and Priori (2012)	Yang et al. (2006): Conference abstract Müller et al. (2003) Schneider et al. (2000) Kiehl et al. (2001) Veit et al. (2002) Birbaumer et al. (2005) Glenn et al. (2009) Harenski et al. (2010)	– + + – – – – –	Not specified: “Legal and clinical implications”
Glenn and Raine (2009)	Yang et al. (2006) Conference abstract Kiehl et al. (2001) Birbaumer et al. (2005) Veit et al. (2002) Glenn et al. (2009)	– – – – – 	Pharmacological treatment, transcranial magnetic stimulation, crime prediction, punishment

(continued)

Table 6.2 (continued)

Authors	Studies cited sMRI fMRI – Italicized	Effect	Suggested application
Glenn and Raine (2014)	Yang et al. (2009) Kiehl et al. (2001) Birbaumer et al. (2005) Glenn et al. (2009) Müller et al. (2003)	– – – – +	Treatment, punishment, responsibility
Glenn et al. (2011)	Yang et al. (2009) Birbaumer et al. (2005) Kiehl et al. (2001) Glenn et al. (2009)	– – – –	No criminal responsibility
Hauser (2016)	Yang et al. (2009) Yang et al. (2010) Kiehl et al. (2001)	– – –	Not specified: Responsibility, punishment etc.
Knabb et al. (2009)	Kiehl et al. (2001)	–	Criminal responsibility, crime prediction
Koenigs and Newman (2013)	Yang et al. (2009) Kiehl et al. (2001) Birbaumer et al. (2005) Müller et al. (2003) Glenn et al. (2009) Deeley et al. (2006)	– – – + – 0	Not specified: Culpability, crime prediction, treatment
Ling and Raine (2018)	Yang et al. (2009) Ermer et al. (2012) Glenn et al. (2009) Birbaumer et al. (2005) Kiehl et al. (2001) Schultz et al. (2016)	– – – – – +	Crime prediction, treatment, punishment
Marazziti et al. (2013)	Veit et al. (2002)	–	Not specified: possibly relevant to responsibility
Mendez (2009)	Veit et al. (2002)	–	Legal culpability, treatment
Nadelhoffer et al. (2012)	Veit et al. (2002) Kiehl et al. (2001) Kiehl et al. (2004)	– – –	Violence prediction
Nadelhoffer (2015)	Veit et al. (2002) Kiehl et al. (2001) Kiehl et al. (2004)	– – –	Punishment
Ortega-Escobar et al. (2017)	Boccardi et al. (2011) Yang et al. (2009) Kiehl et al. (2001) Glenn et al. (2009) Birbaumer et al. (2005) Decety et al. (2014)	+ – – – – 0[a]	Not specified: criminal responsibility

(continued)

Table 6.2 (continued)

Authors	Studies cited sMRI fMRI – Italicized	Effect	Suggested application
Poldrack et al. (2018)	Ermer et al. (2012) Yang et al. (2010) Veit et al. (2002) Birbaumer et al. (2005) Harenski et al. (2010) Glenn et al. (2009) Decety et al. (2013) Motzkin et al. (2011)	– – – – – – – –	Crime prediction, sentencing, treatment, prevention
Reidy et al. (2015)	Yang et al. (2009) Birbaumer et al. (2005)	– –	Prevention, intervention
Sartori et al. (2011)	Birbaumer et al. (2005) Glenn et al. (2009) Müller et al. (2003)	– – +	Mens Rea
Sifferd and Hirstein (2013)	Birbaumer et al. (2005)	–	Criminal responsibility, treatment
Umbach et al. (2015)	Yang et al. (2009) Boccardi et al. (2011) Ermer et al. (2012) Yang et al. (2010): Successful psychopaths Unsuccessful psychopaths Birbaumer et al. (2005) Glenn et al. (2009) Kiehl et al. (2001) Müller et al. (2003)	– + – 0 – – – – +	Punishment, crime prediction, intervention
Weber et al. (2008)	Raine et al. (2000) Tiihonen et al. (2000) Birbaumer et al. (2005)	– – –	Prevention, treatment, responsibility

[a]Incorrectly reported

Table 6.3 Distribution of effects in neuroimaging studies vs applied literature

Type of effects				
sMRI	Original studies	*(% of total)*	Applied literature	*(% of total)*
Null (none)	13	*46%*	1	*4%*
Smaller size	9	*32%*	22	*88%*
Larger size	6	*21%*	2	*8%*
Total	28	*100%*	25	*100%*
fMRI				
Null (none)	5	*46%*	2	*2%*
Hypoactive	4	*36%*	71	*86%*
Hyperactive	2	*18%*	10	*12%*
Total	11	*100%*	83	*100%*

Note. N findings in applied literature exceed original studies N because applied studies cite original studies more than once

and empirical literatures, at least with respect to the amygdalae and the PCL-R, describe different universes of data: one with a clear neurobiological signal that amounts to a good case for action, the other constituting mostly noise.

We found only two publications that unequivocally argued against legal application of the neurobiological evidence (Patrick et al., 2012; Skeem et al., 2011). These publications also gave the most accurate review of the data. It is also important to note that, while they did not cite amygdala data specifically, two of the most recent applied papers (Haji, 2017; Jurjako & Malatesti, 2018a, b) were generally more careful about empirical data than their predecessors. If this holds, it may signal a shift toward improved accuracy in data reporting.

6.2 Spin

What might account for the citing imbalance in the applied literature? One possibility is that null findings in particular are difficult to find in the original research, as they are often de-emphasized in order to increase the likelihood of publication. This problem, along with other problematic reporting practices, is often termed *spin* and is widely discussed in the medical literature (e.g., Dwan et al., 2013; Franco et al., 2014; Lazarus et al., 2015). Spin may have played a role here as well, as null findings are indeed difficult to find in original studies on psychopaths. Consider for example a study by de Oliveira-Souza et al. (2008) that found reduced grey matter volume in several areas of psychopaths' brains, but not in the amygdalae. The only way to locate the null finding was to compare the study's a priori regions of interest, which included the amygdalae, to tables showing areas with statistically significant differences, which did not include the amygdalae. Unsurprisingly, only the positive findings from that study appear in the applied literature.

Spin in the empirical research may not fully explain problems in applied research, however. Some applied writers are also experts in the research field and should be able to detect even poorly reported null findings. Furthermore, some are demonstrably aware of studies with null findings (i.e. they cite them in other publications) but fail to cite them in the applied literature.[3] Findings may even reverse direction depending on the type of publication. Two researchers ran a meta-analysis and found that while antisocial individuals showed abnormal pre-frontal cortex function and structure, psychopathy scores did not moderate this effect (Yang & Raine, 2009). One of the authors subsequently argued that psychopaths should not be held criminally responsible in part because the meta-analysis had found "significant reductions in both structure and function in emotion-related areas [prefrontal cortex] in psychopathic individuals" (Glenn et al., 2011, p. 303).

[3] For example, Raine does not mention a null finding in de Oliveira-Souza et al. (2008) in several applied publication, but notes it in an empirical publication (Pardini et al., 2014).

Cases like these suggest that the applied literature on psychopathy may have an additional bias. As a rough test of this proposition, we examined citations of studies in which the direction of findings is clearly stated. We examined two studies published almost simultaneously by two research groups with overlapping personnel and samples, and the same imaging method (structural MRI), but opposite findings. One study was titled *Increased Volume of the Striatum in Psychopathic Individuals* (Glenn et al., 2010a, b), and the other *No Volumetric Differences in the Anterior Cingulate of Psychopathic Individuals* (Glenn et al., 2010a, b). The latter was the only null finding in the field so clearly stated. The positive finding was cited 55 times on PsycINFO, 131 times in Google Scholar, and 40 times in PubMed. In the applied literature it was cited five times (Anderson & Kiehl, 2012; Glenn & Raine, 2014; Ling & Raine, 2018; Poldrack et al., 2018; Reidy et al., 2015). Overall, the citations for the null finding numbered 5 in PsychINFO, 34 in Google Scholar, and 4 in PubMed. Applied literature did not cite it at all. To test whether this was because reviewers were more interested in the striatum than the cingulate cortex, we also examined a study that found reduced cingulate cortex volume in psychopaths (Boccardi et al., 2011). That study was cited 15 times in PsycINFO and 122 times in Google Scholar (PubMed did not list it). Applied literature cited it 4 times (Anderson & Kiehl, 2012, 2014; Hauser, 2016; Ortega-Escobar et al., 2017). In other words, citing appears to be more a function of positive findings than their neuroanatomical location.

6.3 Neurohype

In short, it appears that the applied literature is subject to two types of spin, at least when it comes to the amygdala: general reporting bias toward positive findings in the empirical studies, plus spin unique to applied publications. Data exaggeration of this type does not seem to be limited to neurobiology, however. For example, a study by Blair (1995) found differences in 10 psychopaths' and 10 non-psychopaths' performance in a test of moral reasoning. Blair took the data to mean that the psychopaths were incapable of this type of reasoning, a conclusion several philosophers used as supporting evidence for why psychopaths should enjoy reduced moral responsibility. Noting a critical methodological flaw in the study, a research group set out to replicate the findings, twice (minus the flaw and with a much larger sample sizes) and found no effect (Aharoni et al., 2012, 2014). However, philosophers mostly continued to cite the original study and maintained their original conclusion (see Jalava & Griffiths, 2017a, b).

But if all of the above is correct, the question is why the hype, and why in the direction of an apparent dysfunction? One possibility is that the philosophers and legal theorists rely on the scientific literature in good faith, and so miss the nulls along with almost everyone else. This, however, does not explain why some of these writers go to great and apparently independent lengths to describe the empirical studies that support their arguments, but miss others that do not. Nor does it explain why social scientists – some of whom are leading psychopathy researchers – miss them as well.

Another possibility for the spin is systemic: In the applied field, just like in the empirical field, strong data and strong arguments are more likely to find publication. Data, when strong, are rich source material for theories and recommendations, while there are only so many ways of saying the data are weak and not ready for application. Examining the data critically, in other words, comes with an opportunity cost.

Finally, the neurobiology of psychopathy is an appealing discourse because it appears to offer a straightforward explanation for a set of questions that are in reality very difficult to answer. The ultimate questions such as what causes crime, how personality is related to crime, and how neurobiology is related to personality, are complex and, most likely, require expertise in multiple scientific fields to answer. Most people would demand additional information before answering a question like: "Bob, a psychopath, shot Bill. What should we do with Bob?" However, by shifting the discourse from the specific (Bob) to the general (psychopaths) and adding a premise (psychopaths have abnormal brains) applied writers can propose a straightforward answer without having to tackle the hard questions. Kahneman termed this "substitution bias" (Kahneman, 2011). One way of dealing with complex questions, Kahneman (2011) argued, is to substitute easier ones for them. An easier question in this context is what neurobiological events correlate with psychopathy? This is often followed by another, linguistic, substitution: the term "correlates" is replaced with terms like "underlies", "is implicated in" or "plays a role in", to imply causal-like knowledge without the burden of proof that comes with an actual causal claim. Claims so construed pass as statements that something – in this case the behavior of psychopaths – is in some unspecified way *understood* (for a general treatment of this issue in the neurosciences, see Krakauer et al., 2017). The result often sounds something like this:

> Elucidation of the neural correlates of psychopathy may lead to improved management and treatment of the condition. Although some methodological issues remain, the neuroimaging literature is generally converging on a set of brain regions and circuits that are consistently implicated in the condition: the orbitofrontal cortex, amygdala, and the anterior and posterior cingulate and adjacent (para)limbic structures. We discuss these findings in the context of extant theories of psychopathy and highlight the potential legal and policy implications of this body of work (Anderson & Kiehl, 2012, p. 52).

Note the transition from "correlates" to brain regions "implicated" to an understanding of psychopathy sufficient for putting it to "legal and policy" use.

Substitutions like these change the rules not only for how we speak about psychopaths but also for who can talk about them. A philosopher, say, can find out what correlates with psychopathy and substitute that correlation for an understanding of why any given psychopath did what he or she did. The philosopher can then concentrate on the more manageable question of "implicated" brain regions and their relevance to such things as criminal responsibility. The resulting just-so account creates a concrete and intuitive dysfunction around which applied arguments can be built, a process similar using the game of Monopoly to explain why wealth concentrates in the real world.

6.4 Consequences of Neurohype

Neurohype has several potential consequences. One is reputational. Imagine the amygdala as a story and the applied writers as a news organization. In this case, the organization regularly gets the story wrong. No possible reason for this – bias, desire for publishable stories, uncritical trust in their sources – looks good for the organization, and it is reasonable to ask whether it can be trusted to get other stories right.

The applied field – though with exceptions – also reinforces a backward logic to understanding the relationship between brain and behavior. The important legal and clinical questions concern dysfunctions in psychopaths' cognition, emotion, and/or behavior. These dysfunctions, should they exist, may have neurobiological causes, but dysfunctions are not the same thing as their causes. By drawing attention to neurobiological abnormalities in the absence of other abnormalities, neurohype obscures the reason why psychopathy could be relevant to responsibility in the first place. Since psychopaths' cognition, emotion, and behavior, as discussed above, does not appear consistently different from non-psychopaths, applied writers are left to arrange the data in creative ways. For instance, one study found reduced amygdala activity in psychopaths while processing moral dilemmas (Glenn et al., 2009). Several applied writers cite the finding as evidence that psychopaths have problems in moral processing, and discuss other studies that show a link between the amygdala and moral emotions. However, the writers neglect to mention that psychopaths in the original study were indistinguishable from non-psychopaths in their actual responses to the moral dilemmas (the majority of fMRI studies in fact show no behavioral differences; Griffiths & Jalava, 2017).

This is also relevant to arguments about treatment, especially neurosurgery. Some writers suggest that treatment should be informed by psychopaths' amygdala deficits. This raises the question of what a treatment for a condition with no consistent and significant cognitive, emotional or behavioral dysfunction – unless one counts psychopathy itself as a dysfunction – and no known cause would hope to accomplish: a person with increased or reduced amygdala activity or someone with activity identical to non-psychopaths? Since the empirical literature shows psychopaths to fit all three profiles, neurobiologically-informed treatment should approach randomness.

Most importantly, however, the point of applied literature is to change policy and practice. If these are determined not by how psychopaths' brains actually are but how writers choose and report data about them, law and policy would arguably be more biased with neuroscience than without it.

6.5 Conclusion and Recommendations

Philosophers, social scientists, and lawyers have long argued for significant change in how we deal with psychopaths. This change, they argue, is justified on empirical – typically neurobiological – grounds. The arguments are on the whole subtle

and substantive, but the data they rely on, at least with regards to the amygdala, are much weaker than the writers propose. The literature, in other words, shows neurohype. Applied arguments require a higher burden of proof than arguments in basic science. Therefore, if our findings generalize to brain structures beyond the amygdala, there is little reason to consider neurobiology in determining how we deal with psychopaths in the criminal justice system. This argument, though, comes with a caveat: If the data change, this conclusion should change as well.

There are a few simple ways to counteract neurohype. First, authors proposing application could frame their arguments as conditional on data, in the form of, say, "if the data are replicable and specific, then…". This would pre-empt the possibility of an entire body of work becoming obsolete if the data do not turn out as expected. Second, authors and editors might consider their arguments not as academic exercises but as something analogous to a physician prescribing medication. Here, the patients' health functions as checks and balances against carelessness or overstatement. Raising the stakes for the applied literature might likewise increase accuracy in data reporting.

More broadly, applied writers should pay closer attention not only to published data but also to the act of publishing that data. In the medical and social sciences it is becoming increasingly clear that published data are often skewed toward positive findings. Some researchers have begun to take steps to address this problem by, for example, estimating the number of unpublished null-findings needed to alter overall findings in meta-analyses. This sort of care – or even a basic recognition that spin and neurohype exist – is largely absent in applied psychopathy literature.

Finally, applied writers should pay closer attention to incentives. If a social scientist with a theory of their own reports little or no counter-evidence to it, it is likely that their evidence is too good to be true. Even if a neurobiological phenomenon did in reality correlate with a psychopathy diagnosis, null findings should still occur by chance. A field with no or very few null findings, therefore, looks, and is, unrealistic.

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