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It Isn't as Simple as It Seems: Understanding and Treating Psychopathy

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Gillett and Huang (2013) claim that “research on the relationship between upbringing and the genesis of psychopathy and ASPD [antisocial personality disorder] . . . incorporates a local context and the artifacts within it as part of the psychopath’s cognitive machinery and invites us to examine the brain plus adaptive context (B + C) as the unit of cognitive adaptation” (3). However, we must distinguish ASPD, as defined by the DSM–IV (American Psychological Association 2000), and psychopathy, as defined by Hare’s checklist (Hare 1991). (Psychopathy is not a DSM-recognized disorder.) The symptomology and symptoms can overlap, but one is not a subset of the other (see Hare 1996). In particular, ASPD is defined primarily in terms of behaviors, but psychopathy is defined by personality traits as well. As a result, those with ASPD are identified by the sorts of trouble they get into, while it is entirely possible that a psychopath could go unnoticed and undetected for the entirety of his or her life. Most patients diagnosed with ASPD do not meet the criteria for psychopathy, while most psychopaths who come into contact with the criminal justice system do meet the criteria for ASPD. However, those who do not also do not typically meet ASPD criteria. The most important differentiator between psychopathy and ASPD is that psychopaths appear to be incapable of feeling empathy and processing many types of emotional stimuli, but those with ASPD exhibit neither deficit. (DSM–V, due out in May 2013, was supposed to fix the relative sloppiness of the ASPD diagnosis and unite these two syndromes under one umbrella. At last update, however, all proposed revisions to the classifications of personality disorders were voted down.)

If we just focus on psychopathy, the developmental picture becomes considerably murky. While Blair (e.g., 2006) does argue for a developmental approach to understanding psychopathy, he is also very clear in his analysis that social and environmental factors do not shape psychopathy’s unfolding: “As yet, the environmental influences on the emergence of psychopathy remain unclear. The genetics studies. . . . [suggest] relatively little influence” (421). He further notes that “while positive parenting techniques are associated with reduced antisocial behavior levels in healthy children, they have no impact on the level of antisocial behavior expressed by children who present with the emotional dysfunction associated with psychopathy” (423). While it is true that environmental factors will determine which particular behaviors psychopaths (and the rest of us) will choose at particular moments in time, this is an uninteresting influence of “nurture,” as the environment does not seem to do much to affect the existence or basic symptomatology of the syndrome.

Reviews and meta-analysis of the literature on the biomarkers for psychopathy indicate that while there appears to be some correlation between violence, aggression, and amygdala and prefrontal abnormalities, there are, as of yet, no dysfunctions associated particularly and exclusively with psychopathy (Wahlund and Kristiansson 2009; Yang and Raine 2009). Reasons for this lacuna include poor differentiation between psychopathy and ASPD in the studies, the possibility that psychopathy is not a homogenous disorder, comorbidity of multiple mental disorders in patients, the challenge of identifying the brain structures underlying empathy, and overlapping circuits in the brain that underlie social interactions, as well as the usual challenges of small subject numbers, poor controls, ignoring potential regions of interest, and little-to-no standardization of protocols or equipment across labs.

Moreover, it appears that psychopathy does not moderate the structural and functional changes in the brain associated with antisocial behavior, violence, and aggression (Yang and Raine 2009, 86). In other words, we have virtually no idea what differentiates the brains of psychopaths from the brains of others. This fact belies the authors’ desire to use data connecting a history of childhood abuse and consequent attenuated right temporal activity to the etiology of psychopathy (Gillett and Huang 2013). While I have no doubt that severe abuse as a child can lead to reduced right hemisphere volume that correlates with increased violence and aggression, we can also find the reduced volumes for reasons other than abuse, and, more to the point, the right hemisphere abnormalities and the concomitant tendency toward aggressive violence are not hallmarks for psychopathy. Some psychopaths are not violent, and most display instrumental, as well as aggressive, violence. (That is, psychopaths are cold and calculating in their use of violence, though they can also be impulsive.)

At this point, there is also little evidence concerning any environmental factors that influence psychopathy. Not
only do parenting styles seem to have no effect on psychopathy, but as mentioned earlier, the percentage of the population that tests high on the psychopathy scale is consistent across socioeconomic backgrounds, degree of education, race, gender, ethnicity, and nationality. (That we do find such consistency is one reason why the genetic hypothesis is so attractive.) In short, I flatly deny the authors’ claim that the “psychopathic tendencies reflect cycles of disadvantage and marginalization, in addition to whatever neural dysfunctions and genetic predispositions contribute to them” (4). There is simply no evidence to support this contention.

Finally, we have little evidence connecting any therapeutic intervention with decreased recidivism in psychopathic offenders (Salekin, Worley, and Grimes 2010), and indeed about half of the retrospective studies on male psychopathic offenders indicate that treatment is correlated with increased violence and recidivism. (It is interesting to note that these therapies are effective in decreasing re-offending with prisoners diagnosed with ASPD.) Of course, there are many problems with these studies as well, including small sample sizes, skewed samples due to prison populations, no treatments available specifically designed for psychopaths, poor controls, no clear or consistent definition for psychopathy, and difficulties in running prospective studies on therapeutic interventions. Most important, any such study, even if designed well and with large numbers of participants, would only be touching a special segment of the psychopathic population—those convicted of crimes. We have essentially no data regarding treatment possibilities for the “successful” psychopath. Of course, we cannot conclude that any therapy will always fail to overcome or manage psychopathy. We just know that thus far, we have not been successful. And this fact should not be surprising, given that we really do not understand the causes of psychopathy, its developmental etiology, or how to affect its developmental course.

I should note, as an aside, that I have found no reason to believe that all the therapies and interventions referenced in the preceding deny the intrinsically social nature of our psyches, as Gillett and Huang hypothesize. Space limitations prevent me from recounting the different therapeutic approaches in detail. Suffice it to say: Modern talk therapy and cognitive–behavioral modification therapies, both of which have been tried unsuccessfully on incarcerated psychopaths, are fundamentally based on the notions that humans exist in a community and that therapeutic success is measured in no small part by successful interactions with others.

Gillett and Huang want to conclude that the “genesis [of psychopathy], on a model of restoration or recompense for injuries suffered, implies a societal obligation to remEDIATE or repair the damaged human potential evident in psychopathic criminality” (4). In other words, because psychopathy is at least in part a social disorder, society has some obligation to fix what is has broken. Leaving aside whether psychopathy does in fact need some sort of social push to develop in an individual, one could still claim that, due to the high social cost of not remediating psychopaths, society has some obligation to fix what is broken. I am not sure that anyone would disagree with this sentiment. But the problem is, no one has any idea how to do this.

In conclusion: Gillett and Huang assume a strong division between the search for the neural underpinnings for psychopathy and potential socio-environmental causes. But one does not preclude the other. Moreover, while we do not know much about the neural correlates for psychopathy, neither do we know much about any potential socio-environmental causes. In fact, there is little consistent evidence for either biomarkers or social indices of psychopathy. That is one reason psychopathy is such a challenging disorder. More importantly, though, regardless of where psychopathy falls on the nature–nurture scale, the fact is that we have no good rehabilitation therapy for the disorder. Therefore, insinuating that the reason why psychopathic offenders in our penal system are not being treated for their problems is that society has accepted that there is a genetic or neural foundation for psychopathy is misguided. We want to treat all disorders, even those that have strong genetic or neural components, like autism or schizophrenia. But we should only treat when we actually know how to help patients get better.

REFERENCES