From Invective to a Biomedical Disorder

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The 1972 psychopathy paper represented the first convincing evidence of other than psychosocial factors contributing towards psychopathy. Earlier assumptions of societal or rearing factors as being causative were never studied using genetic control as part of the methodology. Apart from the application of the adoption paradigm as described by S.S. Kety et al., the concept of psychopathy which I used, was also important. Whereas the speculative historical concepts were inappropriate to contemporary research, the European as well as the American definitions of psychopathy or sociopathy were static to such a degree that they hardly could be expected to elicit etiological mechanisms. Therefore I chose a "dynamic" definition originally proposed by Franz Alexander in 1924. The core of this definition was low frustration tolerance resulting in mainly alloplastic abstractions. This, again corresponded well with the widespread notion that psychopaths were unable to learn from their own earlier averse experiences.

The main result of the 1972 paper was the documentation of a genetic liability towards psychopathy as I defined it for the purpose of my study. This means that psychopathy—at least in part—is transmitted through a biological mechanism, which again justifies a search for biological traits as possible markers of psychopathy. It was, however, impossible for me to pursue this line of thought due to other research obligations. Fortunately other scientists affiliated with my institute independently entered into a line of research resulting in findings which confirmed the existence of physiological concomitants of psychopathy. Their research is described in a book edited by S.A. Mednick and K.O. Christiansen.

First, it was found in studies of children at high risk for schizophrenia that those of them who were delinquent had a slower psychophysiological recovery rate during learning experiments than had the nondelinquent children of severely schizophrenic mothers. This finding could tentatively be interpreted as being a physiological correlate of relatively poor avoidance learning, as shown in psychopaths in earlier studies by other researchers. In studies at our institute, results indicated that criminal sons of noncriminal fathers showed slow autonomic recovery. The positive correlation between criminal behavior and slow electrodermal recovery has been found by other groups studying psychopathic and criminal populations.

That psychopathic behavior, in the form of poor ability to learn from bad experience, may be rooted in an inherited abnormality of the autonomic nervous system, may indicate that primary prevention of psychopathy/sociopathy will be a very difficult task.

There is no general consensus on the concept of psychopathy. The American DSM III-R definition is to some extent contaminated by circular reasoning, i.e., if a subject behaves in an antisocial way, he has an antisocial personality disorder. This is not a very fruitful type of definition when it comes to the creation of powerful hypotheses for research in psychopathy. The present invitation to comment on my more than 20 year old psychopathy research came to me almost simultaneously with an invitation from the most prominent researcher in the field of psychopathy, Robert D. Hare, to participate in a prospective treatment evaluation project of criminal offenders of which many are psychopaths. I am grateful to Current Contents for this occasion to revive and revise my life with psychopathy.


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