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Hans Eysenck's contribution to our understanding of personality and psychological disorders: A personal view

Gordon Claridge

University of Oxford, Department of Experimental Psychology, South Parks Road, Oxford OX1 4UD, United Kingdom

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ABSTRACT

In this autobiographically themed paper an account is first given of an early testing, in psychiatric patients, of the causal part of Eysenck's introversion–extraversion (I–E) theory. On a range of laboratory measures dysthymic and hysterico-psychopaths were found to differ, exactly as predicted; especially on a key index of depressant drug response, the sedation threshold. However, closer scrutiny revealed that observed effects in the data were due not solely to I–E, but to an interaction between I–E and neuroticism (N), true in both normal and clinical populations. Eysenck's recognition of the importance of N in personality differences is discussed, together with his shift from a Pavlovian to a more Western-style model of the nervous system. It is noted that the significance of this new formulation was overtaken by the revision to the theory developed by Gray and his followers. Considering the latter prompted a discussion about whether theories in this genre are really theories of *temperament*, and not *personality* in the full sense. Eysenck's later revision of his psychoticism dimension is then evaluated and found to be fatally flawed due to its failure to incorporate key defining features of psychosis. The overall conclusion reached is that, despite serious deficiencies in the details of his theorising, Eysenck made important contributions to the field reviewed: a) emphasising a dimensional view of psychological disorders; b) opening the discussion, at a time of much opposition, about unitary psychosis; c) promoting a biological approach to the study and explanation of personality (or temperament!).

In his lifetime Hans Eysenck was such a controversial figure that any comment on his work is bound to be touched by individual bias; even the serious academic research discussed here, let alone his ventures into socially divisive topics and fringe areas of science. His ideas about personality were contentious and evoked strong opinions, even from those of us who knew and worked with him. It is appropriate to let an autobiographical theme run through this paper, in order to explain how Eysenck influenced my own thinking and my eventual conclusions about his contribution to personality theory and abnormal psychology. I have already covered part of the topic in several previous publications (Claridge, 1981, 1983, 2009) and fully in an earlier *Festschrift* to Eysenck (1997). Given the lighter touch of the present piece, consulting, especially, the last of those papers will help the reader fill in details of evidence and arguments about the various themes to be introduced (Claridge, 1997).

I first encountered Eysenck in the early 1950s when, as an undergraduate in the Psychology Department at University College London, I took his lecture course on personality. Even to my naive student eye it was obvious that the Department was not at the best place in its previous and subsequent prestigious history. I was aware that the famous – eventually to become infamous – Cyril Burt had just retired and the place had the taste of a collection of leftovers, spiced up by large dollops of Hullian learning theory, which seemed pointlessly irrelevant to the human psychology I had gone to university to study. I was probably on the point of dropping out. For good or ill Eysenck saved me from that fate.

Eysenck's lectures were a revelation. With the lucidity and confidence that we all came to recognise as the hallmark of his public presentational style, he laid out an approach to individual differences that seemed awesomely complete: a statistically derived account of the descriptive features (dimensions) of personality; an explanation of their underlying biology, accessible through laboratory investigations (In my enthusiasm I obviously failed to notice his heavy reliance there on learning theory concepts and methodology!); and a connection to psychological disorders, envisaged as merely extreme positions on personality dimensions. In that last part of his theory he was foreshadowing his eventual vehement dismissal of the medical model (Eysenck, 1960), a stance that evoked much furore among the psychiatric profession but the arguments for which were already obvious to disciples such as myself — reflected in my scepticism about the content of a parallel series of lectures I was attending on mental 'diseases', given by a clinical psychiatrist.

Despite considerable shifts in my research interests over the years, the power of this 'Eysenck epiphany' has lasted a lifetime and traces of it still pervade my thinking; especially about the dimensionality of personality and spectrum approach to disorder, and a belief that biology and genetics must play some part in shaping these variations. (Nothing

E-mail address: gordon.claridge@psy.ox.ac.uk.

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new there to some contemporary observers but easy to forget that Eysenck pioneered both ideas in the modern era). I have assimilated alternative perspectives, but that has always required a curious kind of *effort* not demanded by the Eysenck dogma. I sometimes liken the phenomenon to the experience I have noticed in lapsed Roman Catholics among my friends and colleagues: try as they might they can never fully shake off the last vestiges of their faith.

Eysenck's influence on my thinking – and eventually my perceptions of the flaws in his work – were reinforced when he took me on as a research assistant. My remit, was to test out in psychiatric patients the 'causal' part of his theory, *viz.* using laboratory measures to explore correlates of the psychological disorders that formed the 'criterion groups' allegedly defining the end points of introversion–extraversion (I–E): anxiety based neuroses (dysthymics, as Eysenck called them) for introversion; and, for extraversion, various forms of hysterical disorder, as well as psychopathy. Neuroticism (N), being an orthogonal dimension and therefore assumed to be similarly high in both clinical groups, was not at the time considered relevant; predictions from the theory were solely about differences between the extreme manifestation of I–E.

A further word about dysthymia–hysteria, the clinical manifestation, according to Eysenck (1957), of extreme introversion and extraversion. The introverted end was unproblematic in being defined by a recognisable and relatively homogenous group of neurotic disorders. This was less true at the extraverted end, as eventually proved to be the case in subsequent thinking about those conditions, both within and outwith Eysenckian theory. Psychopathy was later hived off to define the third, psychoticism (P), dimension (Eysenck & Eysenck, 1976). Meanwhile 'hysteria', because of its dubious sexist connotations, dropped out of psychiatry altogether. The ragbag of disorders previously denoted by the term 'hysteria' remained in the psychiatric classification, to be spread diagnostically across other abnormal reactions to stress: somatoform and dissociative. In the research discussed below, using the old nomenclature – that also included a category of hysterical personality disorder – and bundling in psychopathy as well, made the 'extraverted disorders' group quite mixed, diagnostically: they were often labelled 'hysterico-psychopaths' for the purpose of data analysis and reporting. To anticipate slightly, it actually turned out that there were very few differences between the various subgroups on important experimental measures, suggesting that the heterogeneity across that half of the sample was not as marked as might have been feared.

Despite the fact that Eysenck had promoted his theory as an explanation of *both* normal introversion-extraversion *and* clinical dysthymia–hysteria, a comparison had never been properly made of the two criterion groups. Work attempting to connect the personality and clinical domains had been confined, albeit with a vigorous debate, to questionnaire studies (McGuire, Mowbray, & Vallance, 1963; Sigal, Star, & Franks, 1958; Slater, 1960; Storms & Sigal, 1958) Results there were ambiguous and Eysenck judged – to my good fortune – that a comprehensive laboratory based investigation of the questions raised was necessary.

The setting for the research was unusual and deserves mention. My job was based not in the Institute of Psychiatry but, by special arrangement with the army, at the Royal Victoria Military Hospital, Netley near Southampton. There, freed from the constraints of actually working in the IoP Psychology Department and away from Eysenck's eagle eye, I established an outpost where I was free to do what I liked — and did: pursuing lines of research not immediately connected with testing his theory of neurosis. That 'unofficial' research involved studying an extra group of psychotic patients, unconnected at the time to the main hypotheses about neurosis and personality disorder. The work is not relevant to the present discussion, though it did introduce me to the topic of psychosis and eventually, among other things, shaped my thoughts about Eysenck's psychoticism dimension, returned to later. The whole programme of research at Netley – as it was known for short – was published in my book *Personality and Arousal* (Claridge, 1967).

Netley, now demolished, was a unique hospital, ideal for the research contemplated. As well as a ready supply of easily matched healthy controls, it offered patients covering all of the diagnoses necessary for the research. Referring back to my earlier remarks, it is worth noting the particular availability of patients falling into the category of hysteria, especially those with the now defunct diagnosis of conversion hysteria. Reportedly infrequently seen at that time in civilian *psychiatric* practice, such individuals were very common in the military setting. Patients covered the whole range of expression of that once commonly diagnosed syndrome of pseudo-neurological disorder; *viz. psychogenic* blindness and deafness, anaesthesia and partial paralysis of limbs, and fugue (amnesic) states. Importantly – especially for that particular patient subgroup – all of the participants were young fit men, having been screened on entry to the army for obvious physical complications. Furthermore, the way the military services processed their psychiatric casualties meant that patients when tested were generally free of, or on minimal, medication.

The battery of behavioural procedures administered covered a wide variety of laboratory measures then regularly employed in Eysenck's department to examine individual differences. They included tests of perception (*e.g.*, Archimedes spiral after effect), motor performance (serial reaction time and Stroop interference), and auditory vigilance. To which we added a group of psychophysiological measures: EEG indices and tests of drug response. Among the latter the most salient – and figuring large in later research explicating certain features of Eysenck's theory – was the sedation threshold. This was a procedure used to determine individuals' tolerance of sedative drugs by injecting them intravenously with a barbiturate (commonly amylobarbitone sodium), continuing the infusion until they reached a defined end-point of loss of consciousness. It was introduced into psychiatry as a diagnostic tool by the American clinician Shagass (1954), who used EEG changes as his criterion for the threshold of sedation. Subsequently, in the 1950s and early 1960s there was a flurry of research on the technique, both by Shagass himself and by others exploring alternative criteria for determining the sedation threshold. Among them, at Netley, a psychiatrist colleague and I introduced a simple behavioural criterion (Claridge & Herrington, 1960).

Summarising the part of the work conducted at Netley on neurosis, two facts stood out. The first was that there was very good support for the predicted difference between the two criterion groups of anxiety based (dysthymic) patients and hysterico-psychopaths. (As noted above, there were few, if any, differences on objective laboratory measures among the subgroups of patients making up that diagnostically broad category.) The effects were especially evident for tests like auditory vigilance and sedation threshold, where dysthymics proved to have a significantly better vigilance performance and greater tolerance of the depressant drug (higher sedation threshold) than hysterico-psychopaths. The drug finding, incidentally, was scarcely a novel discovery since it had already been demonstrated by Shagass on a substantial sample of patients (Shagass & Jones, 1958). Still, it pleased Eysenck that his causal theory had been vindicated!

The other main finding from the study did not fit in with Eysenck's dysthymia–hysteria story. Since according to theory the neurotic criterion groups were merely abnormal counterparts of introversion-extraversion, comparing them should have nothing to do with N; being independent of I–E the latter logically should have had no influence, even in clinical populations. This proved manifestly not true in our research, where factor analysis of the psychophysiological data demonstrated what we identified as two distinct components of 'arousal', and led us to conclude that at the causal level *both* I–E *and* N contributed to dysthymia–hysteria, as an interaction between the two dimensions. At an individual test level, this was dramatically illustrated in some highly replicable findings on drug tolerance differences to be found among non-clinical subjects, assessed for I–E and N. The results came from studies examining both nitrous oxide tolerance (Rodnight & Gooch, 1963) and ones using the