

“Hedonic Capacity” Ten Years Later: Some Clarifications

PAUL E. MEEHL

Perhaps due to infelicitous formulations plus some bad editorial changes, and readers' familiarity with the emphasis on Rado's theory of schizotypy in earlier writings of mine, the intent of my "Hedonic Capacity: Some Conjectures" chapter has been variously misunderstood. I take this opportunity to offer some clarifying remarks, together with an expansion of a methodological point that was presented, but not defended, in a single sentence of the original paper.

The main point of the paper was the conjecture that while classical psychodynamics is largely correct in holding that pleasure is impaired in neurotic persons by the interference of anxiety as a state variable, as well as chronically by the anxiety signal putting the various defense mechanisms into operation, thereby inhibiting both the pleasure experience and the instrumental behaviors that tend statistically to bring it about (all of which I subsumed under the term "impedance theory"), we should consider another situation in which the causal relation between anxiety and hypohedonia is in precisely the opposite direction, namely, that a *primary (genetic) defect* in hedonic capacity results in an inadequate buffering or softening of aversive experiences. If this were true in some subset of our population, the exaggeration—both in degree and pervasiveness over life domains—of predominately aversive control which is so characteristic of the neurotic personality would be, in such cases, due to their basic deficiency in hedonic capacity.

It seemed to me that in addition to being of considerable theoretical importance in our general picture of the mind, such a possibility -which I believe is supported by one's clinical experience, once the clinician's

mind is open to considering it as an option, rather than routinely asking what is being defended against or how is anxiety impeding pleasure—is of clinical importance. Pursuit in the therapeutic sessions of how the neurotic warding off is at work will be feckless if what is actually involved is an insufficient pleasure factor adequate to countervail anxiety. In addition to wasting time, and pushing interpretations that mobilize “realistic resistance,” such pursuit has an opportunity cost, because hypohedonic clients need to learn this psychological fact as found differentially in life sectors, and to develop techniques for living within a “scarcity economy of pleasure,” as Rado put it. (Hypohedonia leading to insufficient anxiety-softening might also be true for the other emergency affect, rage, and its attenuated forms of chronic anger, resentment, rejection of close relations and the like.) I was simply inviting clinician readers to open their minds to this interesting possibility, and to consult their clinical experience as to whether it seems to fit at least some cases.

I took it for granted, of course, that many different factors can keep people from having pleasure experiences, including an external situation which is thin on delivery of reinforcements and opportunities for need gratification. But I suggested that just as an external reinforcement schedule, defined by a patient’s “social Skinner Box,” may provide him with very little pleasure, an equivalent low-reward schedule *as subjectively experienced* could be produced by a biological deficiency.

I tried to emphasize, but apparently not clearly enough, that I thought of this reduced hedonic capacity not as a *pathological taxon* but as a *normal range individual differences variable*. I conceive the “pleasure parameter” as a disposition existing in all degrees from very low to very high, presumably polygenically determined, and in this respect quite analogous to other normal range non-pathological individual differences variables such as the general intelligence factor, the various components of mechanical ability, anxiety proneness, rage readiness, energy level, social introversion, and the numerous other temperamental factors, known and unknown, on which human beings differ one from another. My choice of the term “hedonic” (rather than Rado’s “anhedonia”) was meant to highlight the conception of a positive attribute, a power or disposition, manifesting itself in degrees. So the “deficiency” is merely being at the low end of this dimension. “Capacity” has the same flavor (as contrasted with “defect,” “impairment,” or the like). Also “capacity” connotes, in traditional trait theory and psychometrics, a second order disposition having a heritable component.

Due to my references to Rado's ideas and my earlier writings on schizophrenia, some correspondents have taken it that I consider anhedonia a more or less schizospecific trait, but the article in question does not say so, and I do not believe this. The relationship of hedonic impairment to schizoidia is in my opinion one of a polygenic modifier (I prefer for certain reasons to call it a potentiator instead of a modifier), influencing the probability of developing a clinical schizophrenia in a person who is genetically schizotypal. While I'm inclined to agree with Rado that this particular potentiator is a relatively important one, it is by no means confined to schizophrenia, being found also in persons with a kind of chronic, low grade noncyclothymic depression, and, as Rado said in his later work, also quite frequent in the obsessional character.

The article attempted to pull together data from a variety of fact domains (e.g., animal learning, neurophysiology, factor analysis of clinical ratings) and I made no pretense to a rigorous showing of their equivalence; hence my title "Conjectures."

I have received correspondence about the article's expression of pessimism concerning the difficulty of separating primary hypohedonia from secondary pleasure impairment, and here I should explain the philosophy of science in which I operate. Suppose one is a strict operationist about concepts, and a strict verificationist about statements or theories (or those statement collections we call theories), and combines this with the social scientist's usual reliance upon statistical significance tests as a way of confirming theories. On those methodological views, it seems natural to say that if Meehl claims there is something called "primary hypohedonia," and something else that we find in the adult acculturated specimen, after his complex learning history, called "secondary anhedonia," such a theory is irresponsible if the theorist does not tell us, *right now*, how to tell them apart. But since I am neither an operationist nor a verificationist (positions having been held by no philosopher of science since roughly 50 years ago!) and I have grave doubts about the way in which significance tests are used in psychology to prove substantive theories, this complaint does not distress me. The fact of the matter is that many theories at a given stage of scientific knowledge are not strongly testable at the time they are proposed, and the history of the other sciences provides numerous examples. Of course a theorist can take advantage of this unfortunate fact of the unequal development of theories, required auxiliary theories, and instrumentation, to propound conjectures irresponsibly, but I hope I am not doing that. I made a couple of suggestions about ways to study primary pleasure deficit in

very young children, but not being a child clinician or developmental psychologist I did not feel competent to discuss that in any detail. It is simply a fact that the human mind is complicated, and that it has a certain developmental and structural “layer” character, so that until more is known about the function of those brain centers involved in positive reinforcement (and, on the subjective side, in the pleasure experience) than we now know, and better psychometric or experimental devices are available, we cannot reliably distinguish primary from secondary hypohedonia. When you deal with a 25-year-old schizoid personality who experiences very little pleasure, it is difficult to design an experiment or build a psychometric instrument that will tell you whether he doesn’t experience pleasure mostly because he’s too angry and afraid to do so, or that he doesn’t experience pleasure mainly because he has too few hedonic polygenes, that being one reason that he has so much anger and anxiety, these negative affects not being adequately softened by countervailing pleasure tone.

Conceiving hedonic capacity as a *dimensional higher-order disposition of polygenic origin* (analogous to Spearman’s *g*) does not, of course, preclude the further possibility that some “pathological” cases occur on the basis of a developmental anomaly or a major genetic locus. As an analogy, I point out that the main heritable component of the general intelligence factor over “normal range individual differences in IQ” is polygenic is quite compatible with the existence of the Mendelizing mental deficiencies.

Finally, with regard to my reference to Olds (+) reinforcement centers, I thought it was interesting that there are inherited differences among rats in this respect. Being strongly hereditarian in my views about personality, I take it as obvious that any time a major individual differences variable is identifiable either clinically or psychometrically in human beings, some considerable causal component underlying that variation is genetic in origin. Minnesota colleagues have said I shouldn’t have so readily identified the pleasure experience with the behavioral fact of reinforcement (defined as an event that produces an increase in operant strength); I of course agree with them that no such immediate automatic identification of the two is permissible. But it is inconceivable to me that, although conceptually distinct, they should not be intimately related.