

Primary and Secondary Hypohedonia

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Having shown taxometrically that there exists a hypohedonic schizotypal taxon in a college population, J. J. Blanchard, S. W. Gangestad, S. A. Brown, and W. P. Horan (2000) suggested that P. E. Meehl erred in revising his 1962 theory by postulating a normal-range individual differences variable of hedonic capacity that potentiates schizotypy into schizophrenia. The aversive drift and secondary anhedonia of Meehl's theory imply that the schizotypal taxon will generate hypohedonic taxonicity in an adult population. Psychometrically measurable hedonic disposition (as distinguished from genetic primary hedonic capacity) is "dragged along" by the schizogene, especially in the social domain. To choose between causal interpretations it could be ascertained whether the schizotypal anhedonic taxon is composed of individuals who are schizotaxic on the basis of psychophysiological, cognitive, and soft neurologic indicators.

Blanchard, Gangestad, Brown, and Horan (2000) reported a taxometric analysis of measures of social pleasure propensity that strongly evidences the existence of a taxon identified as schizotypal social anhedonia in a college population. They take this finding, which I have no reason or motive to doubt, to show that I erred in revising my original theory of schizophrenia and should revert to it. Their article is a clear and scrupulously fair treatment of my theory and its changes as well as an exemplary application of taxometric method. The authors are to be commended for their large sample size and their thorough analysis of internal statistical relations within their data, two features of sophisticated taxometrics that are insufficiently appreciated (Meehl, 1999). Given the high quality of this research, my contribution should not be viewed as a criticism (or even, in the usual sense, a reply) but as a comment on the state of the art in psychopathology and the terrible complexities we face.

Because the conclusion drawn concerns the revisions of my schizophrenia theory, I briefly review that history. In my presidential address to the American Psychological Association (Meehl, 1962), I proposed a theory of schizophrenia inspired by the writings of the deviant psychoanalyst Sandor Rado (1956, 1960; Rado & Daniels, 1956). I conjectured (with Popperian freedom) that schizophrenia is not primarily a mental disorder but a neurologic disorder of genetic origin. What is inherited as endophenotype is a neurointegrative deficit I call schizotaxia. I offered some speculations as to the single-unit neurophysiology of this integrative defect, but they are not the central core and may be disregarded in examining the theory's main postulate: that aberrated synaptic control parameters are the immediate neurophysiological basis for what Bleuler considered the core psychological trait, his associative loosening. Schizotaxia is analogous to other recognized special defects of central nervous system (CNS) integration (e.g., dyslexia, tone deafness, prosopagnosia; see Meehl, 1990, p. 14), except that I conjecture it is ubiquitous, present at all levels from

spinal cord to frontal lobes, impairing all sub-systems but in different amounts. (I shall not discuss my efforts in 1962 and in subsequent papers to derive the main clinical features of schizotypy from that neurologic base.) I conjectured that "*on all actually existing social learning regimes*" [italics in original] persons with the schizotaxic brain acquire, by social learning processes, the schizotypal personality. I specifically denied that schizotypy as a personality make-up—with its characteristic signs, symptoms, traits, and psychodynamics—is inherited; I cannot understand why articles and textbooks have attributed that absurd belief to me.

The theory assumes that, once developed, the schizotypal personality is permanent but that it can exist like other disorders (e.g., diabetes, gout, cardiac disease) in varying degrees of clinical compensation. With Rado, I recognize the compensated schizotype, the decompensated, the disintegrated, and the deteriorated states. These categories are not disjunctive but successively inclusive. As a result of my clinical experience as a psychotherapist, I attached considerable importance to the decompensated but non-disintegrated schizotype called by Hoch and Polatin (1949) "pseudoneurotic schizophrenia" and labeled by some as "borderline state"; I prefer to call it the Hoch-Polatin syndrome. I listed four clinical features of the schizotype, in all degrees of compensation, one of which—perhaps unduly influenced by my training analyst, Bernard C. Glueck, M.D., a Rado disciple—was anhedonia. Subsequently (Meehl, 1972), I demoted anhedonia's causal role to being 1 of 13 polygenic potentiators that collectively determine, together with adverse environmental factors, whether a schizotype decompensates into clinical schizophrenia. However, I did not retract the diagnostic importance of impaired pleasure disposition as one of the clinical signs of the schizotypal personality.

Schizotaxia is a neurologic disorder, with schizophrenia as a psychological complication found in a minority (I conjecture about 10%) of cases. Causally and statistically, the situation is analogous to such conditions as hyperuricemia, in which only a small subset of patients experience gout; elevated serum glucose, in which a subset of patients experience diabetic blindness; or essential hypertension, in which a subset of patients have cerebral hemorrhage. Clinicians and researchers should cultivate the (deviant) habit of

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considering the neural integrative defect as the entity's defining property, although the rare mental complications, being readily visible and socially important, are what enabled Kraepelin and Bleuler to discern it.

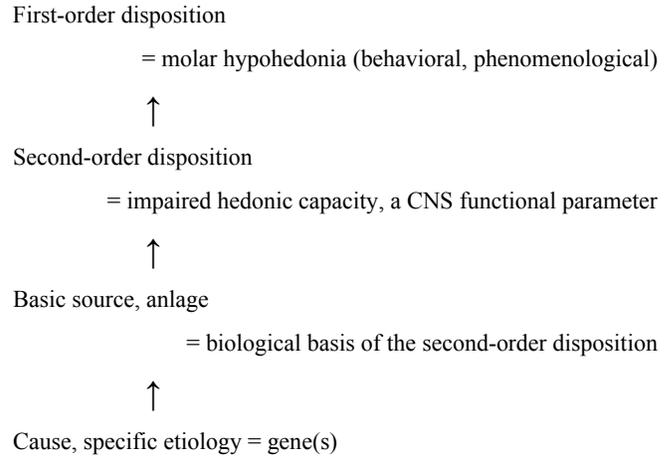
Research on the soft neurology and psychophysiology of schizophrenics and their relatives and of preschizophrenic children, psychometric indicators of schizotypal cognitive slippage and hedonic impairment, and epidemiological studies of these in college populations are conceptually integrated under the total evidence rule (Lubinski, 2000) to corroborate this conjecture (see, e.g., Blanchard et al., 2000; Chapman, Chapman, & Fowles, 1993; Clementz, 1998; Fish, Marcus, Hans, Auerbach, & Perdue, 1992; Korfine & Lenzenweger, 1995; Kwapil, 1998; Kwapil, Miller, Zinser, Chapman, & Chapman, 1997; Lenzenweger, 1999; Lenzenweger & Korfine, 1992, 1995; Lenzenweger & Loranger, 1989a, 1989b; Lowrie & Raulin, 1990; McDowell, Myles-Worsley, Coon, Byerley, & Clementz, 1999; Parnas, Cannon, Jacobsen, Schulsinger, Schulsinger, & Mednick, 1993; Raine, Lencz, & Mednick, 1995; Tyrka et al., 1995).

A couple of years later (Meehl, 1975), addressing not the problem of schizophrenia but psychopathology generally (and normal personality), I conjectured the existence of a normal-range individual differences variable of hedonic capacity, not requiring anything pathological in the usual sense, analogous to traits like Spearman's *g*, social introversion, or dominance, that show wide dispersion in the general population. I further conjectured that this normal-range trait was strongly heritable and adduced clinical, psychometric, developmental, and experimental evidence supporting the idea. This conjecture was corroborated by Dworkin and Saczynski (1984) and by Lykken and Tellegen (1996), although the latter did not use the term hedonic capacity or cite my article. I did not say, nor do I think I implied, that the existence of such a normal-range individual differences variable precludes the existence, in addition, of a hereditary pathological defect such as Rado envisaged, because I had no opinion on that. Rado, strongly influenced by Kallman (1946), postulated a major gene, two pleiotropic effects of which were anhedonia and a proprioceptive integrative defect (kinesthetic diathesis); these effects jointly constituted the neurophysiological anlage underlying schizotypal development.

Hypohedonia is an impaired disposition to experience pleasure, accompanied behaviorally by weakened effect of objective positive reinforcement in conditioning and maintaining operants and respondents. The term is preferable to "anhedonia," which suggests zero pleasure; but the latter term is harmless when denoting the hypohedonic extreme. We assess the subjective (experienced) side by interview and psychometrics and the behavioral side by the array of operant and respondent strengths and judgments of informants, nurses' notes, and the like.

Primary hypohedonia is the heritable component of pleasure impairment, a basic reduced capacity, polygenically determined but possibly involving a major locus in addition. The conjectured basic source is a deviation in the microanatomy or neurochemistry of the limbic system, where I originally spoke of Olds (+) and Olds (-) centers (Meehl, 1962). In philosophers' terminology, primary hedonic incapacity is a second-order disposition to acquire the first-order disposition of hypohedonia (Broad, 1933/1949). We do not presently possess a direct "operational" indicator of this concept, which is defined only implicitly by the postulated theo-

retical network and whose existence we can only infer from sparse genetic data. Thus, we have



Learning that some had inferred a repudiation of Rado's pathological concept, in a subsequent article contributed to a Michael Reese symposium on anhedonic states, I clarified this point by emphasizing that the two ideas are not incompatible (Meehl, 1987, p. 50). Many behavioral and physiological variables show a sizable dispersion among "normal" individuals but also manifest extreme values induced by some specific pathology. It is easy to list examples; in medical genetics this statistical situation is the norm rather than the exception. A strong polygenic contribution to the distribution of Spearman's *g* is perfectly consistent with the existence of more than 160 Mendelizing mental deficiencies contributing pathological cases at the low end of the normal distribution of intelligence. Normal individuals show individual differences in pitch perception on music appreciation tests, but there are also individuals who are genetically tone deaf. There are normal-range individual differences in color naming and color discrimination, but some individuals are red-green color-blind because of a specific mutation on the X chromosome. There is a wide range of individual differences in average serum glucose level, but some of us are diabetic. Examples in psychology and medicine are counted not in the scores but in hundreds or even thousands (cf. McKusick, 1998). It would have been absurd to claim that, if there was a normal-range individual differences variable in hedonic capacity that had a high heritability, there could not also be an extreme (pathological) disposition because of a major gene producing Rado's anhedonia. Given this clarification (Meehl 1987), I cannot explain why I modified "normal-range individual differences factor" by the parenthetical adjective "nontaxonic" in Meehl (1990, p. 24) other than by admitting to a careless locution intended to highlight the dimensional character, with wide range, of the hedonic capacity concept itself rather than denial of a possible anhedonia taxon besides.

The four-article sequence (Meehl 1962, 1975, 1987, 1990) appears to involve three successive "undoing" tergiversations as to Rado's anhedonia, which I surely did not intend. Whether the confusion reflects muddy thinking or careless writing is of only biographical interest, but I surely cannot fault readers for the manner in which they interpret it. Throughout the sequence, what

I intended was only one modification of Rado, the addition of a normal-range individual differences factor to his theory, leaving open the (empirical) question whether clinical anhedonia was only the hypohedonic end of that dimension or a latent taxon besides. At least Meehl (1990) made clear the crucial distinction between latent (primary) hedonic capacity as a (genetic) cause and manifest anhedonia (clinically observed pleasure impairment) as an effect, the latter being subject to aversive drift in the schizotaxic individual. The important theoretical clarification here is that almost all manifest indicators, psychological or physiological, that exhibit taxonicity are causally produced (and hence statistically loaded) with intermediate-level latent quantitative factors, which are themselves taxonically distributed. This was unfortunately not set out in my earlier writings on taxometric method (but see Meehl, 1999; Waller & Meehl, 1998).

The interpretation question arising from Blanchard et al. (2000) is whether their clear showing of a social anhedonic schizotypal taxon refutes the conjecture that Rado's anhedonia is merely a deviation at the low end of the hedonic capacity dimension rather than a pathological disposition such as we would attribute to a major gene. Putting it the other way around, could a taxonic behavioral anhedonia arise in the absence of a taxonic hedonic deficit genetically produced by a major locus? Would it be possible for phenomenological hypohedonia to be statistically taxonic (anhedonia), even if the genetic primary hypohedonia were not? My answer is yes, because the phenomenological (psychometric) anhedonia could get its statistical taxonicity from the taxonicity of the schizotype. That this is not a piece of "ad hoc-ery" to preserve my amended theory (on one interpretation of what I said) is shown by the emphasis in my original and subsequent articles on the crucial role played by aversive drift (first explained and derived in Meehl, 1962, further explained in Meehl, 1990; I do not here address whether my derivation of aversive drift from the schizotaxic slippage will hold up, inasmuch as, so far as I am aware, nobody, whether favorable or unfavorable to my theory, has ever discussed it.)

A striking and baffling manifestation of aversive drift is the occurrence of intensely negative affective states not clinically identifiable as variants of the commonly recognized aversive emotions. Although I lack quantitative evidence and cannot cite supportive clinical literature—surely there must be reports of it?—my psychotherapeutic experience suffices to convince me. Once a clinician has noticed it and learns to probe for it, the phenomenon is unmistakable, showing up in a sizable minority of pseudoneurotic patients and in the majority of disintegrated schizotypes. I have treated bright, introspective, and psychologically sophisticated individuals with Hoch-Polatin syndrome who complained of an acutely unpleasant mental state but steadfastly refused to accept my proffered labels (e.g., "anxiety," "shame," "guilt," "grief" [object loss], "depression"). I am persuaded that this is not a semantic or defensive matter; rather it reveals the existence of a special kind of negative affective state that I, a nonschizotype, cannot empathize with because I have never experienced anything close to it in phenomenal space. I wish I (lazy!) had been systematically recording exemplary verbal expressions of it over the years, but here are some I can recall: "My whole mind just hurts" (this from the woman—a psychology student—in whom I first noted the symptom some 45 years ago); "It's a bad pressure in the head" (query: a headache?); "No, in my mind, a stress"; "There's a kind of friction in the brain when it works"; "I am mentally stretched on a rack"; "It's this perpetual damned strain, strain, strain"; "There's a sort of tension going on inside, a pulling and

hauling"; "My thoughts are an intense irritation, like a sore." I conjecture the phenomenon to be pathognomonic of schizophrenia, deserving to be listed along with such signs as Bleuler's associative loosening, schizophasia, thought deprivation, bizarre somatic delusions, and extreme perceptual aberrations as nearly sure indicators of the disease.

Here is the main point: In writings subsequent to the 1962 presentation, I have distinguished between (a) the innate hedonic capacity arising in the endophenotype from either microstructural or neurotransmitter aberration in the limbic system and (b) the verbally reportable (or clinically ratable) pleasure level of the adult acculturated organism. This latter I referred to as secondary anhedonia (Meehl, 1972). In the schizotaxic brain, the aversive drift, which I consider to be universal among schizotypes, results in an unfavorable statistical bias in cathexes, perceptions, expectancies, and appetitive/avoidant instrumental operants. Negative affective tone seems over time to become stronger and statistically preponderant over positive. Persons, activities, places, tasks, and such, all acquire an unpleasant or painful affective accompaniment, as do images, expectancies, and memories. Meanwhile, the class of overt adient instrumental behaviors, especially but not wholly in the social domain, are weakened or suppressed by the negative inner signal or state, and an increasing proportion of operants are maintained in strength by aversive rather than appetitive control. Warding off anxiety and other aversive states occupies a larger part of the behavioral economy than gratification seeking. This is also true of neurotics, but in them less severe, less generalized, and arising on a different neurophysiological basis. Even a voluntarily undertaken new hobby gradually becomes boring, then irksome, and then even anxious or shameful (e.g., "I haven't practiced my cello lately. I hate the sight of it."). Any theory of schizophrenia that does not deal with this ubiquitous and damaging aversive drift—the effects of which led Harry Stack Sullivan to characterize the schizophrene as one who has "given up on life"—is deficient.

Early on, I raised the irksome question of how the psychologist could go about separating primary (genetic) hedonic capacity from the phenomenological impairment of pleasure observed in the adult, but I had little helpful to say about that. Unfortunately, I still do not have an answer. Neither the ethics nor the technology permits us to "do it right," which would involve permanently implanted telemetric microelectrodes giving readings of the small child's limbic system function while undergoing various life experiences. Whether sufficiently reliable observation data in very young children under standardized conditions could serve as a proxy for this invasive procedure is a problem for the developmental psychopathologists to answer. One can think of noninvasive procedures, such as magnetic resonance imaging, which might be capable of doing something of the sort, but I lack competence to discuss this.

It is clear what the theory would envisage, on the conjecture that there is no Radovian major locus anhedonic effect but only individual differences in the hedonic capacity continuum. We must think in terms of thousands (yes, I mean thousands) of distinct learning curves—where what is being acquired is cathexes, perceptions, derived motives and affects, respondent and operant conditionings—in which the several acquisition function parameters are biased in the aversive direction because of the aversive drift generated by the defect in the schizotaxic brain. Compared with a nonschizotype, a schizotype who was on a roughly similar

objective social reinforcement schedule from significant others during the developmental period (and from others as an adult) will be, in a cross-sectional study, statistically shifted in the hypo-hedonic direction. Psychometric measures of pleasure experience, whether subtle or obvious, will behave taxonically because the whole collection of acquisition function parameters has been shifted aversively by membership in the schizotypal taxon. In my revised schizophrenia theory, I treated polygenic hypo-hedonia as a potentiator of the schizogene's effect; but Fisherian interaction being symmetrical, the schizogene potentiates low-end polygenic hedonic capacity as well. The greater impairment of pleasure in the social domain is explained by the stochastic, autocatalytic, and feedback character of social reinforcement schedules (Meehl, 1990, p. 18).

Do Blanchard et al. (2000) believe that their finding of a hypo-hedonic taxon refutes the conjecture of a normal-range individual differences variable? Their data (in agreement with other such studies) clearly reveal a sizable variance in the taxon's complement class. They estimate the mean taxon-complement difference to be approximately 1.4 standard deviations, close to the expected absolute difference between two randomly chosen members of the same latent class (1.33 *SD* by the usual formula, if Gaussian). Thus, the average differences within and between groups are about equal. Rado aside, the fundamental conjecture of my 1975 article on hedonic capacity is supported by their findings. When they claim that their "findings are inconsistent with Meehl's (1989, 1990) revised proposal that hedonic capacity is a nontaxonic individual differences variable" (Blanchard et al., 2000, p. 92), they are both perpetuating the slip that I made and making an analogous one themselves. Better, they should say their findings corroborate manifest anhedonia being taxonic and are neutral with respect to primary hedonic capacity's possible taxonicity.

Their research strategy indicates the existence of a schizotypal social anhedonic taxon, with a low base rate, in a general college population. It does not tend to refute, but rather corroborates, the conjecture of a normal-range individual differences variable. I note that they label it "schizotypal anhedonia," not simply "anhedonia," suggesting that their interpretation is similar or identical to mine. If it were taken to prove that a primary anhedonia is independently taxonic, as distinguished from a secondary taxonic hypo-hedonia found in schizotypes, that inference would be unwarranted. However, I repeat that I have no reason or motive to deny such a possibility. Rado and I may both have been right about primary pleasure deficit. His major gene is, however, schizospecific (not only anhedonic), because its double pleiotropic effects are anhedonia and proprioceptive diathesis. His "action self" is formed by pleasure—"the cement of the action self"—associated with the kinesthetic experience of action. The gene-produced twofold deficiency is the anlage of schizotypy as a personality organization. My theory readily permits this interpretation but does not assert it.

Lacking utopian brain science procedures on infants and small children, there is an indirect but fairly powerful approach to distinguishing between a primary anhedonic taxon and a secondary aversive drift anhedonic taxon produced by the schizotypal taxon. Suppose the statistical taxonicity (which is all my method can attest, no statistics ever being self-interpreting) occurs in the general college population because there is a dominant schizogene (or a polygenic system with threshold) that creates aversive drift in schizotypes. Shifting our attention from the schizotypal personality

syndrome to the underlying schizotaxic brain, we predict that many of the individuals identified psychometrically by their extreme pleasure deficit should also have a schizotaxic brain. Even with current techniques, we can identify schizotaxic persons—quite apart from their personality organization or clinical psychopathology—by taxometric analysis of psychophysiological, basic cognitive, and soft neurologic signs. The taxometrically based sorting of individuals into the schizotypal anhedonic taxon is fallible, as is the sorting of some individuals as schizotaxic. Relying on multiple indicators, the taxometrically inferred valid and false-positive rates should enable us to predict the cell frequencies of a fourfold table relating anhedonia to schizotaxia. Correctness of that numerical prediction within tolerance would strongly support the schizotypal interpretation of the data. Poor agreement would disconfirm it. Second, deleting cases with high Bayes's rule probability of schizotaxia, we ascertain whether the residual group still displays hedonic taxonicity. Other internal checks would readily suggest themselves. In short, if the pleasure deficit is being "dragged along" by the schizoid taxon rather than being taxonic in its own right, we expect most of the severely anhedonic individuals to be schizotaxic. In all such analysis, one must be careful not to confuse the accuracy of individual diagnoses with statistical predictions from the model. As is often true in psychopathology, it is possible to devise satisfactorily strong tests of a theory that do not depend on high confidence in individual patient sortings.

The problem presented by this and similar studies using my coherent cut kinetics method is easier to state than to resolve. It arises because the method, in each of its variant procedures, starts by conjecturing the existence of overlapping latent distributions for each candidate taxon indicator. Statistical search procedures and consistency tests are derived as theorems from that core conjecture. Any causal factor, historical or current, that can yield two populations whose distributions on the candidate indicators are to some extent separated, even if largely overlapping, will yield a taxonic result. Unfortunately, a causal influence of little theoretical interest is capable of doing this; for instance, we might have a Tellegen case (so named because it was argued by my Minnesota colleague Auke Tellegen), in which some purely administrative or judicial action, such as mainstreaming a group of mentally deficient children in a posh suburban high school, will generate taxonic statistics. I have argued elsewhere (Meehl, 1992) that this is a perfectly good taxon if one's empirical domain is educational administration or jurisprudence, just as a mixing of two distinctive subpopulations of squirrels or fruit flies by some ecological happenstance yields statistically real taxonicity; but if we are psychologists or geneticists, such a mix may be of little interest to us. Again, no statistical procedure is self-interpreting (Meehl, 1995, 1999), and taxometrics in this respect is like factor analysis, cluster analysis, multidimensional scaling, or item response theory. The schizotaxia-anhedonia case is more interesting because it arises from the laws of genetics and developmental psychopathology rather than from economics, school policy, or a probate court's ruling.

The general statistical principle involved tells us that when a taxonic causal factor interacts with (potentiates, acts on) a nontaxonic factor to shift the latter's mean from the distribution of cases lacking the taxonic factor, then indicator variables for both factors will display taxonicity, given sufficient sensitivity. Simi-

larly, if we have (fallible) manifest indicators of a basically nontaxonic factor, and a taxonic factor operates on those indicators, then even if the taxonic factor is unrepresented “directly” in our fallible indicator list, the indicators of the (basically nontaxonic) factors will behave taxonically. Notice that this statistical reasoning does not depend on details as to the causal influence but is a direct mathematical consequence of the abstract, generic notion of “acts on” or “interacts with.” This complicates life for the taxometrician, but (as Bertrand Russell says) “I am not to blame for that, not having made the world.” As Blanchard et al. (2000) appreciated, their article highlights the perennial problem of causal inference from correlational data (McKim & Turner, 1997).

Relying on research evidence, clinical experience, and theoretical extrapolation, I offer the following summary conjectures and open questions:

1. There exists a sizable normal-range individual differences factor of primary hedonic capacity, probably polygenic.

2. There may also be a taxon in the hypohedonic region of this dimension, determined by a major gene; this is an open question on available evidence.

3. If Conjecture 2 is correct, the anhedonia may be a pleiotropic effect of the schizogene (Rado), a major gene mutation independent of the schizogene(s), or an indirect effect of a more general neurointegrative defect (Fish, Meehl); this is an open question.

4. Manifest impairment of the pleasure propensity (clinical anhedonia), although found in many schizotypes in varying degree of decompensation, is also found in other mental disorders, especially major depression. Whether it can be used as a strong diagnostic sign of schizotypy is unclear, but it is not pathognomonic.

5. Either Conjecture 1 or 2 could act as potentiator of schizophrenia in the schizotypy.

6. Because schizotypal aversive drift produces secondary anhedonia, the existence of the schizotypal taxon will generate taxonicity in multiple indicators of manifest hypohedonia and in such indicators when they are combined with other, nonhedonic indicators of schizotypy (e.g., psychophysiology, neurologic, perceptual, cognitive, social).

7. The statistical taxonicity of any set of schizotypal indicators makes it difficult to ascertain whether the latent distribution of any of them is inherently taxonic apart from the influence of the schizotaxic gene(s). Schizotaxia “drags along” any indicator whose quantitative value it influences, by aversive drift or otherwise.

8. One approach to the problem of Conjecture 7 would be to see how the schizotaxic taxon relates to the anhedonic one.

9. The difficulties in appraising these theoretical conjectures and teasing apart the variables’ relations exemplify our perennial problem of making causal inferences from cross-sectional statistics.

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