Taxometric method, invented by Paul Meehl (1965) and developed by him and a number of colleagues, is a family of procedure for subjecting parts of a certain class of theories to risky tests. The theories in question postulate the existence of a latent category, the \textit{taxon}, and its \textit{complement class}. A taxon is negatively defined as a “non-arbitrary latent category.” It does not appear to be possible to positively and precisely define “taxon” (Meehl, 1999, p. 197). In fact, this appears to be because “taxon” is an open concept (Pap, 1953), for one reason because the list of its exemplars is indefinitely extensible. Nevertheless, examples of clearly taxonic categories are easy to find in biology, medicine, and even sociology: biological species; biological sex (at least for species where sex is determined chromosomally); Huntington disease, phenylketonuria and other completely penetrant, single-gene disorders; and being a member of certain discrete, often isolated or especially tightly-knit, socially defined groups (e.g., Hutterites, Shakers, Trotskyists, native speakers of Basque).

A complement class, on the other hand, may or may not be particularly cohesive, discrete, or isolated. While smallpox sufferers constitute a taxon, the class of individuals who do not have smallpox is not. On the other hand, the classes of male and female persons both constitute taxa, which (excluding rare phenomena, i.e., hermaphrodites and chromosomal aneuploidies) exclusively and exhaustively partition the human race.

The reader should note particularly that “taxon” is a term referring to certain causal structures, which are the reason why the class in question is phenotypically discrete, or the social group is isolated. It is not merely a way of referring to statistical properties of distributions of manifest variables, in respect of the latent class, as is done with finite mixture
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models (McLachlan & Basford, 1988). Examples of causal structures that give rise to a taxon include:

1. a normal genetic variant that leads to a natural category (e.g., biological sex, with only about 0.18% of individuals having a non-XX, non-XY chromosomal makeup—estimated by combining frequencies from Bock, 1993; Linden, et al., 1988; Ness, undated; Nielsen, et al., 2003, with data on the ratio of male to female live births in the U.S. from National Center for Health Statistics, 2002);

2. an abnormal genetic allele which, in single or double dose, is approximately sufficient to cause a certain disorder (e.g., the point mutation at the phenylalanine hydroxylase locus that causes phenylketonuria);

3. an abnormal genetic allele with a rather sharp threshold-type effect (e.g., excess trinucleotide repeats at the huntingtin locus, where normals have 7 to 29 repeats, while individuals with Huntington disease [HD] show 37 or more; Masuda, et al., 1995; individuals with intermediate counts of 36–40 have incomplete penetrance (Johns Hopkins University, no date);

4. a highly virulent infectious agent which is necessary but may not be sufficient for causing a certain disorder (e.g., smallpox in unvaccinated individuals);

5. a causal agent involving genetic as well as environmental influences, which acts in a threshold-type fashion (as in Gottesman and Shields’s 1967 polygenic threshold theory of schizophrenia); or

6. a set of strongly interacting, autocatalytic, jointly approximately sufficient causes that lead to a certain social outcome (e.g., a belief that one’s political or religious views are quite correct, coupled with a belief that it is essential not to mix with people whose views differ materially from one’s own—these beliefs reinforce one another in mutual positive feedback, until a cult is formed).
These form a decreasingly strong series of causal structures which might reasonably be called forms of “specific etiology” (Meehl, 1977). These causal structures are ordinarily part of the taxon-postulating theory that is to be tested.

Because the term “taxon,” as proposed here, means “non-arbitrary latent category” plus “suitable postulated causal structure,” direct demonstration that the causal structure involves a non-arbitrary category (e.g., identifying the gene for Huntington disease, demonstrating that the tuberculosis bacillus obeys Koch’s postulates) is always the most satisfactory way to establish that a given postulated categorical structure really obeys Plato’s Socrates’s principle: “That of dividing things again by classes, where the natural joints are, and not trying to break any part, after the manner of a bad carver” (Plato, Phaedrus 265e).

Taxometrics is a general approach, and a set of specific procedures, for answering theoretical questions about the existence of taxa. As such, the questions are put so as to admit of yes-or-no answers. On the other hand, there is a sense in which “taxonicity is not taxonic” (P.E. Meehl, personal communication, January 12, 2001). It would help if we could resolve this apparent paradox.

A potential resolution, in my view, lies in distinguishing between the “taxonicity” (natural yes-or-no structure) of the taxonicity question when the answer is in the negative, and its lack of taxonicity when the answer is in the affirmative. There are causal structures which are in no wise particularly close to a specific etiology (gene or germ), a sharp threshold effect, or a very strong autocatalytic interaction effect. Absent such causal structures, there is no taxonicity. In significance testing, no reasonable person seems to put other than infinitesimal subjective prior probability on the possibility that two group means are exactly equal; so group differences are a matter of degree and not yes-or-no. By contrast, in taxometrics reasonable people put sizeable prior probability on the possibility that the causal structure is not one that we have in mind when we speak of a taxon.

This is why the practice, common in finite mixture models, of testing for one component
(i.e., no taxon) versus two components (taxon versus complement class) is not always a pointless exercise. Waller (personal communication, January 13, 2004) points out that there there are at least two distinct, one-component models in finite mixture analysis. One such model postulates that manifest variables, which serve as indicators of latent category membership, are independent. Another allows the variables to be associated—via conditional probabilities in latent class analysis, or covariances in continuous-variable mixture models. Using the former as the benchmark for comparing a two-component model, favors endorsing the two-component model any time the manifest variables are not independent. For example, a common (dimensional) factor that accounts perfectly well for lack of independence between manifest dichotomous variables (e.g., test items) can be misread as a taxon, in such a comparison.

In contradistinction to situations where there is in nature no taxon, suppose there really is a natural category (i.e., the causal set-up is taxonic). We are discussing a situation where such a set-up exists, whether we know it or not, i.e., whether our evidence for this is convincing at present or not. Then the strength of this taxon will be a matter of degree. That is, the taxon and complement class will differ from each other by a certain amount on the first of several manifest indicator variables (i.e., the causes which lead some people to be taxon members, while others become complement class members, influence the first variable to some degree that is between trifling and overwhelming). The same will be true for the second variable one is examining in connection with one’s taxonic theory. And so on. In this sense, something’s being measurably a taxon is a matter of degree—with respect to certain manifest variables.

Waller (personal communication, March 23, 2004) has pointed out that this last point risks being misunderstood. In particular, it appears to conflate the way things really are (ontology) with how good the evidence is, or what we know about how things are (epistemology). It is crucial not to conflate these. By saying that taxonicity is measurable with respect to a set of variables, I do not mean to suggest that “cat” and “dog” are taxonic
with respect to one indicator list (e.g., a set of facts about genes, and ancestry, and some facts about anatomy) and non-taxonic with respect to another list (e.g., furriness, presence of sharp teeth, having four legs, and eating meat). Instead, I am pointing out the following: even with the most discriminating possible list of indicators (the variables that in nature, but perhaps partly or entirely unknown to us, maximize the accuracy of multivariate discrimination between taxon and complement class), some taxa are “stronger” than others. To compare how distinct two taxa are from their respective complement classes, we would like to be omniscient so we would know how best to constitute the indicator list for each discrimination. We would then consider each taxon-versus-complement distinction to be, ontologically speaking, as distinct as it would be if measured on the best possible variables. In comparing two such taxon-complement class pairs, we would then look at how well separated the first taxon is from its complement class, compared to how well separated is the second taxon from its complement. Surely not all such comparisons would yield equal distances. In that sense, some taxa would be more taxonic than others. Some would be nearly nontaxonic (e.g., a subspecies differing little even from its closest relatives, with very few genetic, and perhaps no morphological, differences), while others would be enormous (e.g., certain archaeobacteria living near deep sea vents that reduce hydrogen sulfide, rather than using sunlight as a direct or indirect energy source).

This scheme for thinking about how taxonic a taxon is depends not on what we know now, or even what we might know next year, but instead on what we would come to know after an indefinite period of appropriate and fruitful research. Therefore, the scheme is not one that confuses epistemology and ontology.

There is a different, and weaker, sense in which something’s being a taxon is itself not taxonic. Suppose membership in a certain taxon is very, very rare in a population. Suppose that the taxon’s causes nevertheless strongly affect observable variables of interest. Such a rare taxon will not account for as great a part of individual differences, in that population, as would an otherwise similar taxon that was more common. One could grade taxa
on a continuum, from rare ones with scant explanatory power to more prevalent ones with, ceteris paribus, very high power. This is obviously related to the epidemiologist’s concept of “population attributable risk.” The appeal of this sense, in which taxonicity is not taxonic, will be greater in applying taxon concepts in public health, than in testing theories about the categorical nature of diseases.

However convincing it might be to demonstrate the yes-or-no specific etiology of membership in a category, at a certain point in the growth of scientific knowledge, one may not be able to perform a strong, direct test of one’s causal theory. Nevertheless, one may be in possession of measurements on certain manifest variables on a set of individuals. One may have reason to hypothesize that this set contains non-negligible numbers of taxon members, and of complement class members. Taxometric procedures provide ways of testing whether the data tend to corroborate or refute the existence of the conjectured latent taxon.