On method, theory, and the classification of complex disorders

Robert A. Zucker

Modern statistical techniques, including a variety of multi-dimensional scaling strategies, are only beginning to be exploited in the alcohol field (McArdle, Hamagami & Hulick, 1994; Muthen, 1997). They offer a promise for greater clarity in sorting out what has increasingly been characterized as a set of disorders rather than a single entity (Babor *et al.*, 1994). Dr Peters paper (Peters, 1997) is in this tradition, and makes use of non-metric multi-dimensional scaling with a consecutive series of 277 in- and outpatients, involving approximately 40% of women and comprising 91% of the clients voluntarily seeking treatment for alcohol problems in the Amsterdam health region. Results generally are confirmatory of the dimensional integrity of the alcohol dependence syndrome (although, interestingly, increased tolerance does not load on this dimension); they also yield an antisociality dimension whose prototype cluster is heavily male at one pole, and heavily female at the antipole, and an "agedness" dimension that is marked by chronicity at one pole and what appears to be young antisocial alcoholism (characterized by the author as a "quarrelling youngsters" antipole) at the other. The author appropriately notes that the antipole of the second dimension needs better characterization, and that the third one is better described as "a composition of two opposing prototypes where age is a likely connecting factor", rather than as a genuine dimension.

The advantage of bootstrapping technique is that it allows one to capitalize on the over determination of symptoms within groupings; then by means of iteration procedures one can begin to uncover the "true" structure of the underlying universe. Aside from idiosyncratic decision making issues that can effect the achieved solution, there are other limitations that will significantly affect the end result. One of these is noted by the author, namely that the dimensional universe being estimated can only be described to the extent that the item matrix adequately samples its composition. The other is not mentioned, but also will strongly effect the nature of the dimensional space being described, namely that the sampling of individuals must adequately represent the range of groups included in the universe being characterized; in other words, the range of "alcoholisms" must be defined and sampled reasonably well. Otherwise, the obtained solution will be limited.

In attempting to make sense out of this work I note that these results reiterate the importance of the dependence syndrome as a core dimension for characterizing alcoholic variability, and that this was the first dimension derivable from the MDS analyses. I also note that antisocial alcoholism, the most common of the "alcoholisms with comorbidity" (cf. Zucker, 1994) defined one pole of the second dimension; but I am not so clear as to how to interpret the opposing anchor for the second dimension. Like Dr Peters, I believe it would be useful to describe this dimensional space better before concluding that it involves either negative affect or impulse inhibition. For example, the risky temperament literature (e.g. Wills *et al.*, 1996) indicates that both sociability and negative mood/social withdrawal are potential motivators for problem alcohol use, but the patterns of social involvement while drinking would be expected to be very different for these two temperament variations, and the likely dominant patterns of social behaviour while not drinking would also be anticipated to be different (e.g. see Jacob, 1987).

To set these results in the larger context of the "alcoholic universe", it is important to appreciate the nature of the sampling frame for both subjects and items being characterized here. The obtained sample was representative of the population at large in education and social class, but we are left uninformed about the degree to which subjects were representative of the population of alcohol-abusing and dependent individuals in the region, as well as of the time span over which subject enrolment took place (i.e. was it a few months, a year, or even several years, so that the rarer forms of the disorder might be captured?). In addition, the issue of representativeness of alcohol dependence stage as a function of sample age has not been explicitly addressed, although the early 40s mean age of these Ss, as well as their age dispersion (SD = 10) indicates that the majority were in "mid-career". In other words, early-onset/early-remitters (Zucker, 1994) as well as late-onset alcoholics (cf. Brennan & Moos, 1995) appear to be poorly represented. Interestingly, despite this

under-representation, age-related variation does show up as one of the core characterizing dimensions of the syndrome.

These results underscore a larger point, that in a way has already been made by the emergence of the age dimension as a characterizing attribute. The point is simply that alcoholic symptomatology and the life course structure of the disorder are heavily interwoven, even when the phenomena we wish to characterize appear at first blush to be clinically intractable and isolated from context (Zucker *et al.*, 1997). Thus, even when the item domain being set up as the core identifiers for the syndrome's dimensional space heavily involve content issues of dependence, drinking behaviour and drinking pattern, issues of co-morbidity and life course "location" emerge as key definers. It is interesting to see how robust these effects must be, considering that they continue to show themselves even when the item structure marks them only in a very rudimentary way. This interaction is more likely to be present with a disorder such as alcoholism, which has substantial social as well as biological complexity to its aetiology. It would be anticipated to be less so with disorders that are more heavily neurobiologically driven, such as cocaine dependence.

These findings also remind us that content-free and theory-free strategies for characterization can only be a beginning. Ultimately, we need to have a theory of the disorder that we can confirm or disconfirm, that can guide us in both sampling of subjects and in item content selection. Lacking the theory, we may omit key identifier nodes (e.g. those who never seek treatment), or rare subtypes, that may be clinically important but require special efforts to access and sample (e.g. antisocial alcoholics among women) (cf. Hill & Smith, 1991). Dr Peters clearly is aware of these limitations, but they bear repeating.

ROBERT A. ZUCKER
University of Michigan,
Departments of Psychiatry and Psychology and
University of Michigan Alcohol Research Center,
400 East Eisenhower Parkway
Building 2, Suite A, Ann Arbor,
Michigan 48108-3318, USA.

BABOR, T. F., HESSELBROCK, V., MEYER, R. E. & SHOEMAKER, W. (Eds) (1994) Types of alcoholics: evidence from clinical, experimental and genetic research, *Annals of the New York Academy of Sciences*, 708.

BRENNAN, P. L. & MOOS, R. H. (1995) Life context, coping responses, and adaptive outcomes: a stress and coping perspective on late-life problem drinking, in: BERESFORD, T. & GOMBERG, E. (Eds) *Alcohol and Aging*, pp. 230–248. (New York, Oxford University Press).

HILL, S. Y. & SMITH, T. R. (1991) Evidence for genetic mediation of acoholism in women, *Journal of Substance Abuse*, 3, 159–174.

JACOB, T. (1987) Alcoholism: a family interaction perspective, in: RIVERS, P. C. (Ed.) *Alcohol and Addictive Behaviors Nebraska Symposium on Motivation 1986, Vol. 34,* pp. 159–206, (Lincoln, NE, University of Nebraska Press).

MCARDLE, J., HAMAGAMI, F. & HULICK, P. (1994) Latent variable path models in alcohol use research, in: ZUCKER, R. A., HOWARD, J. & BOYD, G. M. (Eds) *The Development of Alcohol Problems: exploring the biopsychosocial matrix of risk*, NIAAA Research Monograph No. 26, pp. 341–386 (Rockville, MD, US Department of Health and Human Services).

MUTHEN, B. O. (1997) Latent variable modeling of longitudinal and multilevel data, in: RAFERTY, A. E. (Ed.) *Sociological Methodology*, pp. 453–480 (Washington, DC, Blackwell).

PETERS, D. (1997) A natural classification of alcoholics by means of statistical grouping methods, *Addiction*, 92, 1649–1661.

WILLS, T. A., MCNAMARA, G., VACCARO, D. & HIRKY, A. E. (1996) Escalated substance abuse: a longitudinal grouping analysis from early to middle adolescence, *Journal of Abnormal Psychology*, 105, 166–180.

ZUCKER, R. A. (1994) Pathways to alcohol problems and alcoholism: a developmental account of the evidence for multiple alcoholisms and for contextual contributions to risk, in: ZUCKER, R. A., HOWARD, J. & BOYD, G. M. (Eds) *The Development of Alcohol Problems: exploring the biopsychosocial matrix of risk*, NIAAA Research Monograph No. 26, pp. 255–289 (Rockville, MD, US Department of Health and Human Services).

ZUCKER R. A., DAVIES, W. H., KINCAID, S. B., FITZGERALD, H. E., REIDER, E. E. & BINGHAM, C. R. (1997) Conceptualizing and scaling the developmental structure of behavior disorder: the Lifetime Alcohol Problems Score as an example, *Development and Psychopathology*, 9, 453–471.

When cluster analysis fails: method and theory in the search for alcoholic subtypes

Thomas F. Babor

The search for alcoholic subtypes has had a long and varied history, with little to guide its progress but clinical intuition during the pre-Jellinek years leading up to the modern era of alcohol studies.[1] With the development of multivariate statistical techniques and improvements in clinical assessment technology, typology research has