TYPE A - TYPE B CLUSTERING OF ALCOHOLICS - A PRELIMINARY REPORT FROM AN INDIAN HOSPITAL

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ABSTRACT

To generate homogeneous clusters of alcoholics and to check the empirical stability of the clusters. Patients from consecutive admissions were assessed by face to face interview on 7 or 8 post abstinence day in a cross-sectional design. 73 male inpatients satisfying DSM-IV criteria of alcohol dependence syndrome without significant physical or cognitive deficits formed the sample. Apart from demographic variables, twelve other parameters were assessed with appropriate instruments over a span of last six months. Cluster analysis was followed by ANOVA on the twelve variables between the clusters. Stability of the clusters was checked by a three step statistical technique. Two clusters with 61 subjects and 12 subjects were accepted. ANOVA showed significant difference on nine out of twelve variables. Throughout the three steps of the check mechanism eight variables were found to consistently discriminate the two clusters. On a small sample of hospitalized alcoholics using twelve parameters we could obtain a preliminary evidence that subtypes simulating Type A - Type B could occur in a different sociocultural setting. Further studies on a bigger sample with data on treatment response are indicated.

Key words : Alcoholics, clusters, type A, type B

Alcoholism is long known to be a heterogeneous disorder. There have been numerous attempts (Babor and Lauerman 1986) to subtype alcoholism in recognition of the complex heterogeneity of this disorder. However, the issue that has ever riddled such efforts is the multiplicity of the defining characteristics of alcoholism (Babor and Meyer.1986).

Recently Babor et al. (1992) proposed empirical clusters of alcoholism (Type A and B) and could also demonstrate their clinical validity especially with regards to short term course and outcome. This is a comprehensive and an atheoretical approach, in the sense, that the authors used multiple variables of severity and vulnerability of alcoholism and in the process have avoided emphasis on a single or multiple but fewer domains. This way, the approach seems to stand out of most of the works of typology of alcoholism (Knight,1938; Jellinek, 1960; Schuckit,1985; Morey and Skinner.1986; Cloninger,1987; Zucker,1987). The robustness of such an approach was further demonstrated on a bigger sample of alcoholics by Schuckit et al. (1995).

It is obvious that having valid subtypes could have far reaching implications about the understanding of a putative vulnerability, treatment response and the course and outcome of this disorder. Nevertheless, what remains to be known about this typological model is its universality or cross- cultural validity. In other words, do such clusters exist in populations, like ours, that differ widely from their western counterparts with respect to a number of such defining variables, for example, socioeconomic
and sociocultural factors (Bales, 1946), per capita alcohol consumption (Royal College of Psychiatrists, 1986), polymorphism of alcohol dehydrogenase (ADH) and aldehyde dehydrogenase (ALDH) genes (Yoshida, 1991) and susceptibility to alcohol related health damage (Lumeng and Crabb, 1994)? The present study was carried out with this rationale in mind. Our objectives were only to determine if such clustering (A & B) could be faithfully generated in a sample of our population at all.

MATERIALS AND METHODS

The study was carried out in the Department of Psychiatry, Kasturba Medical College, Manipal. The sample (N=73) was selected from the male patients (16-60 years) referred from other medical departments as well as from other hospitals for inpatient treatment of alcoholism.

Patients satisfying DSM-IV (APA, 1994) criteria of alcohol dependence syndrome were recruited in the study. Those with cognitive deficits (as defined by a score <24 on Mini Mental State Examination) and disabling physical disorders were excluded. Written informed consent was obtained from every patient included in the study. The patients were further assessed on the following parameters (over a span of last six months) on post abstinence seven to ten days.

1. Sociodemographic data i.e. age, education, occupation, income, religion and marital status were obtained using a semistructured interview schedule.
2. Familial alcoholism was assessed with Family History - Research Diagnostic Criteria (FH-RDC) (Andreasen et al., 1977). A ratio of the number of the first degree relatives who fulfilled the criteria of alcoholism to the total number of first degree relatives was computed in order to indicate the degree of familial alcoholism.
3. Onset of problem drinking was measured by averaging the reported age at onset of four milestone events in the patient’s drinking career: age at first regular drinking, age when began getting drunk (or binge drinking) regularly, age at which heaviest drinking began and age at first reported diagnosis of alcoholism.
4. Dependence severity was determined by Severity of Alcohol Dependence Questionnaire (SADQ) (Stockwell et al., 1979).
5. Amount of alcohol consumed per day was calculated according to the method of quantity frequency index (QFI). If the patient consumed more than one brand of alcohol in the last six months then the QFI of each brand was calculated and added up to get the total QFI.
6. Frequency of other psychosocial substance use was assessed by a self-report questionnaire developed from Schedule of Clinical Assessment in Neuropsychiatry (SCAN) (WHO, 1992). Each item was rated on a five-point scale. The item scores were added up to provide an estimate of the total frequency of other substance use.
7. Physical consequences of alcohol consumption were obtained through a self-report questionnaire with 64 items. A score was calculated by adding up the number of the positive responses.
8. Social consequences of drinking were assessed using Quantitative Inventory of Alcohol Disorders (QIAD) (Stinnett and Schechter, 1982).
9. Cumulative alcohol related symptoms over the course of a subject’s drinking career were estimated by means of the total score obtained from Michigan Alcoholism Screening Test (MAST) (Selzer, 1971). MAST has been used in this way to measure life time severity of alcoholism in previous studies done in this area (Babor et al., 1992; Schuckit et al., 1995).
10. Years of heavy drinking in the subject’s drinking career including periods of abstinence was calculated by subtracting age at onset of problem drinking from the present age.
11. Depressive symptom count (life time severity) was measured with Current and Past Psychopathology Scale (CAPPSS) (Spitzer and Endicott, 1968).
12. Anxiety symptom severity (lifetime) was also measured with CAPPSS.
13. Antisocial personality symptom count was assessed with a twenty six-item questionnaire.
A hierarchical cluster analysis was used to generate clusters. The computer initially selected those cluster centers that were furthest apart using average linkage (between groups) for cluster membership of cases and then used the agglomeration method. The squared Euclidean measure was used to check indices of similarity. Of the original 73 patients we did cluster analysis to generate 2, 3 and 4 groups. It was observed that the 3 and the 4 groups did not match those that exist in the literature. The two cluster groups were accepted and analysis of variance (ANOVA) was performed on the two clusters. The statistical exercise was carried out according to the instructions and guidance of the consultant statistician of the institute.

We used a check mechanism initially developed by Lorr (1966) in order to test the stability of these clusters. This procedure consisted of three steps:

1. The original seventy three patients were randomly divided into two groups (A & B) of equal number (N=36) (one was dropped out at random to make the groups equal). Then each group was subjected to cluster analysis like the parent group. ANOVA was performed on the two clusters generated in each group.

2. A small number of patients (N=4) was deleted in random order from each of the two groups (A & B) of step 1. Each of the new smaller groups (A' & B') was subjected to cluster analysis. ANOVA was done on the two clusters obtained from each group A' & B'.

3. Finally the groups A & B were tested with ten variables (two variables were removed) by cluster analysis followed by ANOVA.

RESULTS

Twelve patients (16%) belonged to cluster 1 while sixty-three (84%) were included in cluster 2. The mean and SD of the twelve variables and the ANOVA are shown in Table 1. Significant differences were observed in nine out of twelve variables between the two clusters. The results of the check procedure of Lorr consisting of three steps are shown respectively in Table 2, 3 and 4.

In step 1 (Table 2), eight variables could be found significantly different between group A clusters whereas nine variables could distinguish group B clusters.

In step 2 (Table 3) the clusters of both group A' and B' (each consisting of 32 subjects) were discriminated significantly by eight variables. Finally in step 3 (Table 4) eight parameters were different between the clusters of group A whereas those in group B could be discriminated on nine variables. Thus significant differences were seen on at least

| Variable                                | Cluster 1 (N=12) | Cluster 2 (N=61) | F Value | P Value |
|-----------------------------------------|-----------------|-----------------|---------|---------|
| 1 Family alcoholism                     | 0.22 ± 0.08     | 0.14 ± 0.15     | 1.57    | 0.001*  |
| 2 Onset of problem drinking             | 27.88 ± 6.44    | 33.74 ± 7.39    | 1.55    | 0.042*  |
| 3 Daily alcohol consumption             | 1354 ± 687.57   | 317.12 ± 220.54 | 39.54   | 0.000*  |
| 4 Dependence severity                   | 66.5 ± 23.97    | 27.82 ± 13      | 14.55   | 0.02*   |
| 5 Other psychoactive substance use      | 2.5 ± 0.47      | 1.44 ± 0.79     | 2.89    | 0.048*  |
| 6 Physical problems                     | 28 ± 9.85       | 14.73 ± 8.02    | 4.67    | 0.036*  |
| 7 Social consequences                   | 8.5 ± 2.93      | 3.62 ± 2.56     | 5.74    | 0.02*   |
| 8 Lifetime severity                     | 30.5 ± 13.99    | 16.47 ± 8.60    | 10.07   | 0.003*  |
| 9 Years of heavy drinking               | 4.97 ± 3.24     | 4.97 ± 3.31     | 0.61    | 0.439   |
| 10 Depressive symptom count             | 8 ± 5.49        | 3.88 ± 3.94     | 2.13    | 0.164   |
| 11 Anti-social personalities symptom count | 11.5 ± 3.34    | 2.32 ± 2.34     | 23.12   | 0.000*  |
| 12 Anxiety symptom count                | 1.5 ± 1.14      | 2.56 ± 2.23     | 0.46    | 0.500   |

* Statistically significant.
### Table II - Check Procedure: Step 1

| Variable                      | Group A (N=36) | Group B (N=36) |
|-------------------------------|----------------|----------------|
|                               | Cluster 1A (N=8) | Cluster 2A (N=28) | P value | Cluster 1B (N=4) | Cluster 2B (N=32) | P value |
| Familial alcoholism           | 0.31±0.17       | 0.41±0.14       | 0.001*  | 0.22±0.08       | 0.14±0.15       | 0.011*  |
| Onset of problem drinking     | 23.72±4.28      | 32.25±6.29      | 0.001*  | 27.88±8.44      | 33.74±7.93      | 0.042*  |
| Daily alcohol consumption     | 888±211.57      | 259.89±199.38   | 0.001*  | 135±68.75       | 317±220.54      | 0.001*  |
| Dependence severity           | 51.75±5.15      | 33.07±18.29     | 0.001*  | 66.50±23.90     | 27.8±13.00      | 0.021*  |
| Other psychoactive substance use | 3.38±1.59      | 1.82±1.31       | 0.001*  | 2.50±0.47       | 1.44±0.79       | 0.048*  |
| Physical problems             | 25.25±7.10      | 16.20±8.81      | 0.002*  | 28.00±9.85      | 14.73±8.02      | 0.038*  |
| Social consequences           | 8.25±2.68       | 4.07±2.88       | 0.000*  | 8.50±2.93       | 3.62±2.56       | 0.022*  |
| Lifetime severity             | 21.50±4.99      | 19.82±11.92     | 0.005*  | 30.50±13.99     | 16.47±8.60      | 0.001*  |
| Years of heavy drinking       | 4.36±2.55       | 5.14±4.19       | 0.001*  | 4.97±3.24       | 4.97±3.31       | 0.439   |
| Depressive symptom count      | 6.88±4.37       | 6.29±2.69       | 0.001*  | 8.00±5.49       | 3.88±3.94       | 0.154   |
| Anti-social personalities      | 13.12±5.02      | 3.29±2.29       | 0.001*  | 11.50±3.34      | 2.32±2.34       | 0.001*  |
| Anxiety symptom count         | 5.39±5.61       | 2.82±2.71       | 0.060   | 1.50±1.14       | 2.56±2.23       | 0.500   |

* Statistically significant.

### Table III - Check Procedure Step 2

| Variables                      | Group A' (N=32) | Group B' (N=32) |
|-------------------------------|----------------|----------------|
|                               | Cluster 1A' (N=8) | Cluster 2A' (N=24) | P value | Cluster 1B' (N=4) | Cluster 2B' (N=29) | P value |
| Familial alcoholism           | 0.31±0.17       | 0.41±0.15       | 0.001*  | 0.22±0.08       | 0.14±0.15       | 0.011*  |
| Onset of problem drinking     | 23.72±4.28      | 32.25±6.29      | 0.001*  | 27.88±8.44      | 33.74±7.93      | 0.042*  |
| Daily alcohol consumption     | 888±211.57      | 259.89±199.38   | 0.001*  | 135±68.75       | 317±220.54      | 0.001*  |
| Dependence severity           | 51.75±5.15      | 33.07±18.29     | 0.001*  | 66.50±23.90     | 27.8±13.00      | 0.021*  |
| Other psychoactive substance use | 3.38±1.59      | 1.82±1.31       | 0.001*  | 2.50±0.47       | 1.44±0.79       | 0.048*  |
| Physical problems             | 25.25±7.10      | 16.20±8.81      | 0.002*  | 28.00±9.85      | 14.73±8.02      | 0.038*  |
| Social consequences           | 8.25±2.68       | 4.07±2.88       | 0.000*  | 8.50±2.93       | 3.62±2.56       | 0.022*  |
| Lifetime severity             | 21.50±4.99      | 19.82±11.92     | 0.005*  | 30.50±13.99     | 16.47±8.60      | 0.001*  |
| Years of heavy drinking       | 4.36±2.55       | 5.14±4.19       | 0.001*  | 4.97±3.24       | 4.97±3.31       | 0.439   |
| Depressive symptom count      | 6.88±4.37       | 6.29±2.69       | 0.001*  | 8.00±5.49       | 3.88±3.94       | 0.154   |
| Anti-social personalities      | 13.12±5.02      | 3.29±2.29       | 0.001*  | 11.50±3.34      | 2.32±2.34       | 0.001*  |
| Anxiety symptom count         | 5.39±5.61       | 2.82±2.71       | 0.060   | 1.50±1.14       | 2.56±2.23       | 0.500   |

* Statistically significant.

Eight variables consistently throughout the check procedure.

**DISCUSSION**

The basic purpose of the study was to replicate Type A - Type B clustering in a sample and setting that differ markedly with respect to a number of defining characteristics from those of the studies of Babor et al. (1992) or Schuckit et al. (1995). To that extent, we were able to generate two clusters, albeit with skewed sample distribution i.e. 61 subjects in cluster 1 and 12 in cluster 2. The fact that on ANOVA nine out of twelve parameters could significantly differentiate cluster 1 from cluster 2 suggests that the clusters were fairly homogeneous. We further examined the stability of these
### TABLE IV CHECK PROCEDURE STEP 3

| Variables | Cluster 1A (N=8) | Cluster 2A (N=28) | P value | Cluster 1B (N=4) | Cluster 2B (N=32) | P value |
|-----------|-----------------|------------------|---------|-----------------|-------------------|---------|
| 1. Familial alcoholism | 0.31±0.17 | 0.14±0.15 | 0.003* | 0.22±0.08 | 0.15±0.15 | 0.007* |
| 2. Onset of problem drinking | 23.72±4.21 | 32.25±6.56 | 0.000* | 27.88±6.44 | 33.18±7.41 | 0.000* |
| 3. Daily alcohol consumption | 888±221.57 | 259.89±215.31 | 0.000* | 1564±697.57 | 316.32±220.74 | 0.000* |
| 4. Dependence severity | 51.75±9.15 | 33.07±23.61 | 0.003* | 66.5±23.90 | 27.5±12.71 | 0.000* |
| 5. Other psychoactive | 3.36±1.35 | 1.82±1.17 | 0.001* | 2.50±0.47 | 1.44±0.79 | 0.048* |
| 6. Physical problems | 25.25±7.15 | 16.11±9.40 | 0.002* | 28.00±9.85 | 14.44±7.73 | 0.027* |
| 7. Social consequences | 8.25±2.58 | 4.07±3.03 | 0.000* | 8.60±2.93 | 3.68±2.62 | 0.018* |
| 8. Lifetime severity | 21.50±4.99 | 19.82±11.95 | 0.675 | 36.50±19.99 | 16.56±5.69 | 0.003* |
| 9. Depressive symptom count | 6.68±4.37 | 6.29±6.35 | 0.745 | 8.00±5.49 | 4.26±3.22 | 0.239 |
| 10. Antisocial personalities | 13.12±4.96 | 3.29±3.31 | 0.000* | 11.50±3.34 | 3.56±2.37 | 0.000* |

*Statistically significant

clusters by a statistical check mechanism (Lorr, 1966). Firstly, we randomly divided the data to ensure that the programme produces the same number and type of clusters with each, and adding or subtracting small number of individuals or items to or from the data and repeating the analysis to make sure that the clusters obtained are not radically altered by doing so (Strauss, Bartko and Carpenter, 1973). It was found that the data showed consensual validity on eight out of the original twelve variables viz., familial alcoholism, onset of problem drinking, amount of alcohol consumed per day, dependence severity, polydrug use, medical complications, social consequences and antisocial personality. This means that eight variables most consistently could distinguish between cluster 1 and 2 throughout the three steps of that check procedure.

Thus cluster 1 in this study represents alcoholics with poor prognostic features, for example, family risk factors, earlier onset of problem drinking, higher consumption of alcohol, greater severity of dependence, more polydrug use, more alcohol related physical and social consequences and comorbid antisocial personality traits. In contrast, patients in cluster 2 had relatively better prognostic factors like lower family risk factors, later onset, less severe dependence, less frequent polydrug use, fewer physical and social problems. To a great extent, these clusters resemble Type A - Type B dichotomy of Babor et al. (1992).

We have to refrain from making broad generalization from our data due to certain methodological limitations. Firstly, our sample size was small and probably just enough to generate statistically significant clusters. Secondly, we studied less (12 instead of 17) number of variables. This might not have affected the separation of clusters since Schuckit et al. (1995) showed that clusters were robust even on five out of seventeen variables. Thirdly, the sample was recruited from hospital based population who are likely to have more severe alcoholism than those in the community. It is likely that this has resulted in the skewed distribution of the sample between the clusters. Finally, our validation of the clusters was based on mathematics and not on clinical reality.

In conclusion, we hardly make any pretense at establishing the cross cultural validity of Type A - Type B dichotomy of alcoholics. At best we share a convincing preliminary evidence that such clusters could potentially exist across socioculturally disparate populations and settings. This opens the scope for further examination of this paradigm in clinically
relevant ways in such settings.

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