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LETTER TO THE EDITORS

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Methodological problems with ecstasy and the SCL-90

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Parrott et al. (2001) present an interesting set of data gathered from four cities in two European states. In this paper, they argue that the recreational use of Ecstasy is associated with a range of psychiatric problems identified by the SCL-90. However, by their own admission, the analysis of these data is incomplete. Because the missing analyses are crucially important to the purpose of the research, and for a number of other reasons, we would argue that their conclusion regarding the effects of Ecstasy is premature and not supported by their data.

A major problem is that the scale used in their study, the SCL-90, may not have been the most appropriate for their purpose. Derogatis (1994) found that "item analysis of the original SCL-90 revealed items on the anxiety and obsessive-compulsive dimensions were flawed psychometrically...". There are no sanctioned norms in existence for the SCL-90, and it represents an unfinished and incomplete version of the SCL-90-R. Finally, the large volume of research in existence demonstrating the validity and sensitivity of the instrument has been conducted primarily with the SCL-90-R, not the SCL-90 prototype" (Derogatis 1994, p. 3). This statement severely limits confidence in these data. In addition there appears to be no psychometric data on the "positive moods and life experiences" questions that were added by Parrott et al. to the SCL-90. Adding questions to a psychometric scale negates its reliability and validity as determined on the original version. The reason why these questions were unable to discriminate between the groups could have been due to the nature of the questions rather than differences between the subject groupings.

The SCL-90 is a self report scale that assesses psychiatric symptomatology. Parrott et al. state that psychiatric symptoms are different from psychobiological symptoms/problems. As the only measure used in this study was the SCL-90, the logical conclusion is that no validated measure of psychobiological problems was used. In addition, Parrott et al. do not provide details of how problems were assessed. The SCL-90 identifies clinically relevant psychiatric symptoms by comparing scores to tables of norms. This does not appear to have been done in this study and, as Derogatis (1994) points out, no norms are available for this purpose anyway. In order for psychiatric symptoms to be considered problematic it is necessary to demonstrate that their severity is outside of the normal range and thus warrant some form of clinical intervention. Therefore, no data are presented by Parrott et al. that can be used to demonstrate any definitive association between drug use and either psychiatric or psychobiological problems.

Another problem is that it is uncertain whether the subjects were drug free when they completed the SCL-90. No attempt was made to determine when the subjects had last used any drug. It is possible that a large proportion of the drug using subjects had consumed drugs within the previous 24 h. The instructions used in the study appear to rely upon the subjects understanding that "not under the effects of drugs" included the after effects of drug use and also them being able to dissociate the acute and chronic "hangover" effects of drugs from their normal psychological state. As the time period of the assessment was the previous 4 weeks, it is highly probable that the SCL-90 was merely recording the side effects of recreational drug use and not lasting and significant psychiatric symptoms. Indeed, as the "positive" scale was not different between the groups, this suggests that the scores on the SCL-90 represent drug side effects; if they represented psychiatric problems, one might expect the "positive" and "negative" scores to correlate negatively.

It can also be noted that, in Table 2 of Parrott et al.'s paper, each group has a mean score for "MDMA side effects", but no scale from which these data could have originated is detailed in the Methods section. It seems odd that these side effects should occur if Ecstasy has not been used, and suggests that this scale has very poor psychometric properties.

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There is no mention of the statistically significant age differences between the groups. As adult psychopathology might be emerging during the age range of the groups (i.e. 19–24), this is a major confound in this study. The means suggest that it is highly probable that if the groups were redefined by age, there would be a statistically significant relationship between age and scores on the SCL-90. As only a small proportion of young people use Ecstasy, it should not be difficult to find age matched controls. The sex distribution of the groups also differed, with only 40% males in the non-drug users group but 80% males in the "heavy" Ecstasy polydrug users. Reneman and colleagues (2001) have shown that female but not male Ecstasy users show reductions in 5-HT transporter binding after having used an average of 530 tablets. In this context, the sex distribution is crucial. Both of these confounds could have been easily removed with appropriate multifactorial statistics.

Further, if 3,4-methylenedioxymethamphetamine (MDMA)-induced neurotoxicity is the causal mechanism that is being tested in this experiment, then it is necessary to demonstrate that a neurotoxic dose of MDMA has been ingested. Parrott et al. equate Ecstasy with MDMA, but there is substantial evidence that MDMA is not the only drug sold as Ecstasy. The World Health Organisation (1997) has defined Ecstasy as the generic term for a wide range of compounds and analyses of Ecstasy tablets would support this definition (Rothe et al. 1997; Arimany et al. 1998; Milroy et al. 1999; Sherlock et al. 1999; Ramsey et al. 2001). It would be unwise for any researcher to equate the observed differences between Ecstasy users and controls with the (neurotoxic) effects of MDMA alone. The UEL Drug History Questionnaire (Parrott et al. 2000) is unable to provide data which could determine whether a neurotoxic dose has been ingested, as it only provides the number of times used. The key question is the amount of drug used on those occasions; and this is clearly problematic. For example, this same group of researchers has recently published a paper where "light" users of Ecstasy were defined as having used less than 100 tablets (Fox et al. 2001). According to the criteria used in the present study by Parrott et al., these would be "heavy" users of Ecstasy. These inconsistencies do not aid the interpretation of these data and limit comparisons with other studies, even from the same group.

No evidence is provided for why it is "unlikely that individuals with pre-existing phobic anxiety would decide to become heavy stimulant/MDMA polydrug users attending raves or large dance clubs" (Parrott et al. 2001, p. 81). Social phobia is the most common phobia in the general population. Ecstasy users report that it enhances social interaction and reduces anxiety, aggression, impulsivity, and obsessive-compulsive behaviour (Siegel 1986; Peroutka et al. 1988; Liester et al. 1992; Solowij et al. 1992; Bean et al. 1997; Davison and Parrott 1997). In addition MDMA administered to volunteers in the laboratory reduces anxiety (Liechti and Vollenweider 2000). Contrary to the view expressed by Parrott et al. (2001), this suggests that Ecstasy use may be a form of self-medication of pre-existing psychological problems.

If the observed psychiatric profiles were the result of MDMA-induced serotonergic neurotoxicity, then there are also theoretical problems. Serotonergic dysfunction is found in alcohol dependent patients, cocaine users, cannabis users, marathon runners, depressed patients, and victims of child abuse (e.g. Yatham and Steiner 1993; Buydens-Branchey et al. 1997, 1999; Rinne et al. 2000; Becker et al. 2001; Haney et al. 2001). It is interesting that there were no differences in the depression scores on the SCL-90. This is at odds with other studies in this area (Curran and Travill 1997; Gerra et al. 1998, 2000; MacInnes et al. 2001; Verkes et al. 2001). Psychiatric problems are also observed in users of other drugs of abuse that are not serotonergic neurotoxins which cannot be explained if Ecstasy use is the sole causal mechanism for these results. A much more comprehensive hypothesis is therefore warranted.

On a point of information, Parrott et al. suggest that organisations responsible for drug education packages tend to explain the benefits of drug use without detailing the associated harm. However, this does not fit with recent developments within the public health education field. The major harm reduction organisations (e.g. HIT and Lifeline in the UK and Dancesafe in the USA) actually base their entire philosophy upon communicating the harm associated with drug use and educating users on how to reduce it. Without a doubt, this approach has reduced the number of deaths associated with Ecstasy and other drug use. Even advocates of Ecstasy have acknowledged the harm associated with its use (Saunders 1997).

Perhaps most important, however, is the fact that, notwithstanding the above considerations, Parrott et al. actually found no effects that can be attributed to Ecstasy use. The term "Ecstasy polydrug users" employed by Parrott et al. is problematic in this context. Parrott et al. acknowledge that polydrug use is the norm in this population and the number and amount of drugs used increases simultaneously. Consequently, in order to single out Ecstasy as a contributor to negative symptoms, it is necessary to establish unique effects of Ecstasy that can be differentiated from the effects of other drugs. Parrott et al. did not examine this in their statistical analysis, thus instead of performing planned comparisons or post hoc tests between their three groups of polydrug users (who varied in terms of Ecstasy use), they conducted an overall trend analysis, which included non-polydrug users, that did not examine the specific effects of Ecstasy. As shown in Table 2 of Parrott et al.'s paper, the means for the three polydrug using groups on the SCL-90 are actually broadly equivalent despite estimated variations in Ecstasy use. In fact, despite an overall effect for drug use overall, the mean total negative scores of polydrug users actually *decline* with increasing use of Ecstasy (they are 9.57, 9.59, and 9.55, for no, "light" and "heavy" Ecstasy use, respectively). Thus, Parrott et al. do not actually demonstrate any effect of Ecstasy at all.

This fits with other data; for instance, if increasing use of Ecstasy over the past 10 years has caused drug-related psychiatric problems then there should be a huge increase in young people presenting as patients. Even with 2 million Ecstasy users in the UK there is currently no credible evidence that this is happening.

In conclusion, we would argue that the study reported by Parrott et al. used psychometrically flawed measures, failed to address obvious confounds, and only partially analysed the data. However, notwithstanding these criticisms, the data as presented fail to demonstrate an association between ecstasy use and psychiatric problems. It would therefore be unwise to use these results as the evidence base for a public health initiative.

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