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McCann, M., Higgins, K., Perra, O., McCartan, C., & McLaughlin, A. (2014). Adolescent ecstasy use and depression: cause and effect, or two outcomes of home environment? *European Journal of Public Health*, *24*(5), 845-850. https://doi.org/10.1093/eurpub/cku062

Published in:
European Journal of Public Health

Document Version:
Peer reviewed version

Adolescent ecstasy use and depression: cause and effect, or two outcomes of home environment?

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Conflicts of interest: None declared

Background: This study assessed the association between adolescent ecstasy use and depressive symptoms in adolescence.

Methods: The Belfast Youth Development Study surveyed a cohort annually from age 11 until 16. Gender, Strengths and Difficulties Questionnaire emotional subscale, living arrangement, parental affluence, parent and peer attachment, tobacco, alcohol, cannabis, and ecstasy use were investigated as predictors of Short Mood and Feelings Questionnaire (SMFQ) outcome.

Results: Out of 5,371 respondents, 301 (5.6%) had an SMFQ greater than 15, and 1,620 (30.2) had missing data for SMFQ. Around 8% of the cohort had used ecstasy by the end of follow up. Of the non-drug users, around 2% showed symptoms of depression, compared to 6% of those who had used alcohol, 6% of cannabis users, 6% of ecstasy users and 7% of frequent ecstasy users. Without adjustment, ecstasy users showed around a fourfold increased odds of depressive symptoms compared to non-drug users (OR 0.26 95%CI 0.10, 0.68). Further adjustment for living arrangements, peer, and parental attachment attenuated the association to under a threefold increase (OR 0.37 95% CI 0.15, 0.94). There were no differences by frequency of use.

Conclusions:

Ecstasy use during adolescence may be associated with poorer mental health; however this association can be explained by the confounding social influence of family dynamics. These findings could be used to aid effective, evidence based drug policies which concentrate criminal justice and public health resources on reducing harm.

Keywords: Ecstasy, Depression, Longitudinal Study

Introduction:

After cannabis, MDMA (Ecstasy) is the most commonly used illegal drug in Europe (1). Recent ESPAD data indicate three per cent of 15-16 year old students in Europe have used ecstasy in their lifetime (four per cent among students in the UK) (2). Users report heightened mood and feelings of euphoria; these recreational effects are primarily attributed to its effect on serotonin receptors, and some theorise that continued use may be harmful, in particular for depression, a condition in which serotonergic deficits are implicated (3). Evidence for the effects of ecstasy use on serotonin, however, is ambiguous. Studies reported no effect of ecstasy on serotonin binding among males, and an apparently reversible effect among females (4). Such studies may not fully address confounding, and depressive symptoms may not be reducible down to a serotonergic defect; cognition, rumination and biased memory all play key roles in depressive symptoms (5) and all have neurological bases beyond serotonin uptake. At the population level: one study whose baseline assessment took place before ecstasy was widely available found that people in poorer mental health were more likely to start taking ecstasy (6). Another study did not replicate this (7), although both found delinquency and aggression were associated with ecstasy use. More recently a study has demonstrated poorer mental health among ecstasy users (8). With conflicting evidence for an association, and with reviews highlighting the lack of quality evidence (9), a further study of the potential effects of the drug seems warranted.

Findings from previous studies have pointed towards both forward, and reverse causal mechanisms. It is plausible that cross-sectional and longitudinal associations suggesting these two mechanisms could both be due to confounding influences – for example family instability or deprivation (10,11) - predisposing individuals to poorer mental health and a greater likelihood of using illicit drugs such as ecstasy. The effect these contextual factors may have on the association between ecstasy use and depression warrants investigation. There may be gender differences in the effects of substance use (e.g. due to differences in body mass); problems with emotional adjustment at younger ages may

make people more prone to harm from external factors such as substance use; and a supportive family environment may buffer against the negative effects of substance use. This study, having followed children throughout their teenage years is well placed to assess the importance of early exposure to drugs, and to assess the role of family and social context.

This paper aims to address the following:

Is adolescent ecstasy use associated with later symptoms of depression (age 16)?

Is ecstasy use associated with depression after accounting for other personal, economic and family characteristics?

Do personal, economic, and family characteristics modify the association between ecstasy use and later symptoms of depression?

Methods:

Participants

The Belfast Youth Development Study (BYDS) began in the academic year of 2000. Schools from a metropolitan area and two townlands with rural catchment areas were invited to participate; 82% of schools agreed. The study surveyed children in their first year of secondary education (11/12 years). 3,834 pupils from 39 schools (87% of eligible pupils) participated in the first wave of the study. Annual visits were made, surveying the same cohort over five years, the fifth wave was completed in Spring/Summer 2005. Four schools were recruited in the second wave of the study, and through the first five waves over 74% of pupils from schools in the study provided data. Personal information was collected on the consent page of the questionnaire, and pupils were provided with an anonymous identification number to write on the questionnaire, once data was linked to the correct ID across waves, the personal information sheet was detached to protect respondent confidentiality.

There was no local ethics board from which to seek approval in 2000; however, the BYDS was granted a favourable opinion from the School of Sociology, Social Policy and Social Work Research Ethics Committee.

There were 5,371 respondents who completed at least one survey in the first five waves (2000 to 2005). At the time of the 2001 Census, over 99% of the Northern Ireland population stated their ethnicity as white, hence ethnicity was not analysed. Age was not analysed as all cohort members were from the same school year (mean age as of 1st September 2000 (nominal start date) - 11.71; s.d. (0.31). Respondents were asked about who they lived with; categorised as; biological parents, reconstituted family (biological parent & partner / step/ foster parent), single parent family, not with parents (most often living with older siblings), and no response. Those consistently providing no response were coded as missing data. Living arrangements in the fifth wave of the study was used; data from previous waves were used for those not responding in wave five.

Other analytical variables are described in this section with further detail in the online appendix. Mental health in waves one and four was assessed using the Strengths and Difficulties Questionnaire (SDQ) (12). Depressive symptoms were assessed in wave five using the Short Mood and Feelings Questionnaire (SMFQ) (13), this variable was the outcome measure for analysis. Both scales were dichotomised to create variables for 'caseness' (see appendix). Principal components analysis was used to create an affluence measure (14) based on household indicators, such as free school meal eligibility. Mean affluence score across each wave was used in analysis, broken into quintiles. The Inventory of Parent and Peer Attachment was used to measure quality of relationship with parents and peers (15), by endorsing questions such as "my parents/friends respect my feelings" and "I trust my parents/friends". This scale has good psychometric properties, and previous research has shown social attachment to be associated with depressive symptoms (16). The IPPA was administered in waves one, three and four. Analyses used standardised mean scores from all available waves.

Substance use variables:

In each wave, respondents were asked about their substance use (tobacco, alcohol, cannabis, ecstasy). Participants responded to items on lifetime use: 'have you ever tried smoking (even a puff)', 'have you ever tried alcohol, even if it was just a sip", 'have you ever tried cannabis, even it was just once', 'have you ever tried ecstasy even if it was just once?' and frequency of use: 'thinking about (name of the substance) which of these statements best describes you?' (only once, between 2 and 5 times, monthly, weekly, more than once a week, daily or having previously used them but not anymore). For this analysis, questions for alcohol, cannabis and ecstasy were combined to create a drug use variable across the wave. Virtually all drinkers reported trying tobacco hence tobacco and alcohol users appear as a single group. Virtually none of the respondents reporting ecstasy use had abstained from alcohol and cannabis. This meant that it was not possible to assess the independent effect of ecstasy on SMFQ controlling for use / non-use of alcohol or cannabis; instead, a variable representing ever reported use of; 'no drugs', 'alcohol only', 'alcohol and cannabis', or 'alcohol, cannabis and ecstasy' was entered into the models. In the first two waves, any report of substance use was considered a positive report. People reporting using ecstasy in earlier waves who subsequently reported only using once were reclassified into alcohol/cannabis/no drugs groups as appropriate. To assess if there was a dose-response relationship between ecstasy and depressive symptoms, the ecstasy user category was split depending on frequency of use; people reporting using ecstasy monthly or more frequently were considered 'frequent users'. An alternative categorisation based on infrequent/experimental use compared to standard/frequent use appears in the online appendix. Where respondents reported using ecstasy but not cannabis, alcohol or tobacco, they were retained in the 'alcohol, cannabis & ecstasy' group. Similarly, cannabis smoking non-drinkers remained in the 'alcohol & cannabis' group; this occurred for only a handful of respondents. Drug use in waves one to four were combined to produce the drug use variable, so as

to give drug use temporal precedence when regressed on wave five depressive symptoms. As the literature mentions transient depression after ecstasy use, wave five ecstasy use was also examined.

Statistical Analysis:

Logistic regression models with an SMFQ greater than 15 as the outcome were performed using Stata 13, and MPlus 7 to account for missing data using Full information maximum likelihood estimation. FIML more accurately estimates the distribution of variables than complete case analysis, and provides unbiased estimates where the reasons for missing data are stochastic (MCAR), or dependent on covariates included in the analysis (MAR) (17). As Table 1 shows, there are several predictors of missingness (including early mental health) hence the MAR assumption may be tenable. Multilevel logistic models were used to assess variation in depressive symptoms across schools. After controlling for gender, there was no evidence of variation in rates of depressive symptoms between schools (LR test vs. single level model $p = 0.1$) hence single level regression models are presented, with standard errors adjusted for clustering within schools.

Interaction terms were used to assess if gender, SDQ group, affluence and attachment modified the association of drug use history with SMFQ score; and also whether living arrangement modified the effect of parental attachment on depressive symptoms. Sensitivity analyses excluding respondents with pre-existing emotional health problems showed no change compared to those for the full sample and thus are not reported.

Results:

There were 5,371 respondents from 44 schools in the cohort. There were 301 respondents (6%) who had a score greater than 15 on the SMFQ, indicating symptoms of depression. Of the 2,755 females in the sample, 8% had symptoms of depression in wave five, compared to around 3% for the 2,615 males. The 371 respondents with emotional problems when younger were much more likely to score

highly on the SMFQ (16%) compared to those with no problems (5%) or with missing data (3%). The rates of depressive symptoms by living arrangement were: with a reconstituted family 9%; siblings/other non-parents 2%; single parent 6%; birth parents 5%. There was no association between affluence and depressive symptoms. Parental attachment was a clear risk factor; the most securely attached quintile group had around a 3% risk of depressive symptoms, increasing to 6% for the fourth quintile, and 12% for the least securely attached. Those with missing information for the measure showed a comparable rate of symptoms as those scoring mid-range (4%). Poor attachment to peers was also associated with elevated symptoms, with rates of 5% for the best attached compared against 7% for the least securely attached (see Table 1).

[Insert Table 1 around here]

Table 1 shows the distribution of cohort characteristics by SMFQ caseness and missingness. Around 7% (368) of the cohort stated they were abstinent from all substances and 2% of this group showed depressive symptoms by wave five. Around half of the respondents (2,541) had used alcohol; 6% of whom showed symptoms of depression. A further 37% (1,965) had used alcohol and cannabis; 6% of whom displayed depressive symptoms. About 5% (278) had used ecstasy; 7% of this group showed symptoms of depression, and of the 140 frequent ecstasy users, 7% had elevated symptoms. Abstainers' likelihood of depressive symptoms was a quarter of that for ecstasy users (OR 0.26 95% CI 0.10, 0.68). Respondents who used alcohol (OR 0.67 95% CI 0.40, 1.12) and cannabis (OR 0.65 95% CI 0.40, 1.07) did not report appreciably lower levels of depressive symptoms compared to ecstasy users. Frequent ecstasy users had a similar odds of depressive symptoms compared to less frequent users (OR 1.03 95% CI 0.42, 2.50). The online appendix presents alternative frequency groupings.

After adjusting for gender, there was little change in the odds for depressive symptoms comparing the abstinent and ecstasy user groups (see Table 2). Controlling for SDQ, affluence and living arrangements led to a 4% reduction in odds; while further adjustment for peer and parental attachment led to a 17% reduction. The corresponding reductions for the alcohol group were 8% and 53%, while for cannabis and frequent ecstasy there was no attenuation in estimates. After including these confounders, there was no evidence of variation in odds of depressive symptoms among the substance using groups, while the abstinent had better outcomes.

[Insert table 2 around here]

Interaction terms in unadjusted models were used to assess if there were differential associations with substance use for different groupings of covariates. There was a weak indication that the association between drug use and depressive symptoms varied comparing males and females ($p=0.054$), no difference for those with and without prior emotional problems ($p=0.94$), the less and more affluent ($p=0.27$), nor the more or less securely attached to parents ($p=0.59$). There was inconclusive evidence for an interaction between living arrangements and ecstasy use ($p=0.10$). Models showing the drug category coefficients stratified by gender appear in the online appendix. Ecstasy use is more strongly associated with poor mental health among males than it is among females. The confounding effect of parental attachment (to attenuate the risk) is the same for both groups.

Discussion:

Ecstasy use from age 11 to 15 is associated with a higher rate of depressive symptoms at age 16; the results suggest that this may primarily be accounted for by problems with parental attachment, and variations in living arrangement. Poor parental attachment, being female, and emotional problems when younger increase the risk of having depressive symptoms by age 16. There was no evidence of variation in rates of depressive symptoms across schools, or with varying levels of affluence. While this study looked only at people from age 11 to 16, depressive symptoms when younger are highly predictive of future mental health (18).

There was no evidence of a dose-response relationship, in that higher levels of ecstasy use was not associated with higher rates of depressive symptoms. One would expect to find higher rates of depressive symptoms among those using more frequently if ecstasy was a cause of depression. There was a cross-sectional association between ecstasy use and depressive symptoms for those who began using ecstasy in the last wave of the study; this didn't attenuate after controlling for confounders. This could be due to the transient effect of ecstasy on mood; or more plausibly, changes in social circumstances between wave four and five that led to initiating drug use (see appendix); or some other mechanism. Using longitudinal data accounts for unobserved change within individuals more fully; this may explain the different findings from the main analysis.

As expected, parental attachment was associated with depressive symptoms (16). Young people emotionally distant from their parents may have poorer psychosocial resources to help them cope with life stressors. Briére et al did not find as marked a reduction in risk of depressive symptoms after controlling for family characteristics (8). There are a number of possible reasons; firstly, Briére et al used measures of conflict, communication and parental rules (containing three, six and seven items respectively). By comparison, the IPPA encapsulates communication, alienation and trust (28 items). Controlling for quality of relationship measured by internalised, rather than behavioural aspects of the parental bond, may have more fully accounted for depressogenic family circumstances. Secondly; Briére et al adjusted models for alcohol and cannabis use as confounding

variables, rather than presenting the risks conferred by incremental levels of drug use as presented here. As all ecstasy users in our study had previously used alcohol and cannabis, it was not appropriate to model this data using Briére's method. The extent to which this affects comparison of the models is unclear. Thirdly; Briére et al used assessed family level variables at ages 10 to 11, i.e. likely before initiating ecstasy, whereas the present study averaged across ages 11 to 14, before or after initiation. The present study may be conditioning a mediating pathway rather than a precedent confounder; the lack of attenuation in the estimate for new ecstasy users in wave five (see appendix) would not support this explanation. Finally; young people from Quebec may have a more elevated risk due to higher dosage, or stronger ecstasy. The lack of dose-response relationship in the current study is also not congruent with this explanation.

The attenuation in the raised risk for depressive symptoms with ecstasy after controlling for parental attachment and living arrangements suggests that a sizeable component of poorer outcomes for substance using adolescents may be factors within the home affecting both substance use and mental health. Improving family cohesiveness may have benefits for the mental health of young people, and potentially reduce substance use.

Limitations

This study could not differentiate between exposure to ecstasy, cannabis, or alcohol, because all ecstasy users also reported using other substances. This makes it impossible to infer a direct effect size as could be found by an RCT study (some of which have taken place (19)), nor is it possible to assess the effect of ecstasy controlling for effect of cannabis or alcohol exposure, as virtually all ecstasy users were also exposed to alcohol and cannabis. Similarly, this study did not assess the effect that other illicit substances besides cannabis and alcohol may have on mental health.

This study was based on a longitudinal panel design, with repeated surveying of the cohort on multiple occasions. Using this method, we were not able to determine time of onset of depressive symptoms, and thus analyse the incident rate of onset of depressive symptoms. A further limitation pertains to the design of the study: different questionnaires were used to assess emotional problems at age 15 (the Emotional Problems sub-scale of the SDQ) and age 16 (the SMFQ). Although both measures are supposed to tap into analogous constructs, different instruments may have produced some variation in caseness.

While this paper focused on ecstasy use, the ecstasy market is not regulated and pills sold as ecstasy in this context may vary in purity and content; even regular ecstasy users do not take steps to determine the content and purity of pills consumed (20). This issue is particularly concerning given recent deaths which have been attributed to other psychoactive substances being sold as ecstasy (21). Ecstasy users may have therefore been using substances that varied somewhat in their composition and this limits the certainty with which associations between reported use of ecstasy and outcomes can be attributed to ecstasy.

For this study, as with others (22) one of the primary limitations is relatively low numbers and lack of statistical power to detect effects. A corollary of this is that the effect sizes of ecstasy on cognitive functioning and/or mental health - should they exist at all - must be minimal to begin with. There were few frequent ecstasy users in the sample for analysis of a dose-response relationship. The small size of the group would reduce the precision of the estimate and lead to wider confidence intervals. In the absence of statistical testing, the lack of trend looking at the raw proportions, or unadjusted associations after accounting for missing data do not suggest there is a large effect to detect.

The findings of this study could be of use to those responsible for making choices affecting young people, mental health, criminal justice and families, who wish to take an evidence-informed approach to their policy decisions.

Acknowledgements:

Most importantly, many thanks to the young people who participated in the study and who made the research possible. Thanks to Michael Rosato for comments on an early version of the paper, and to the reviewers for their suggested improvements. The Belfast Youth Development Study was funded by the Health and Social Care Research and Development Division of the Northern Ireland Public Health Agency.

Key points

Recreational ecstasy use is relatively common, despite the fact it is an illegal substance. It has been suggested that its use could cause depressive symptoms; the evidence testing this hypothesis has produced conflicting and ambiguous findings.

This study used information for a cohort of school children including information on their drug use, living arrangements, affluence and family relationships, through five years of secondary level education, and assessed how these factors were associated with poor mental health.

People using ecstasy during their teenage years are more likely to have symptoms of depression by age 16; but this association attenuates after controlling for parental attachment problems. Ecstasy use itself does not appear to be a major risk factor for poor mental health.

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Table 1: Distribution of personal, socioeconomic and drug using characteristics by SMFQ score, and odds ratios for depressive symptoms

	Respondents (% of total)	**SMFQ>15 (% within group)**	**Unadjusted Odds Ratio SMFQ>15 (95% CI)**	**SMFQ Missing (% within group)**	**Unadjusted Odds Ratio Missing SMFQ (95% CI)**
Total	5,371	301 (5.6)	~~~	1,620 (30.2)	~~~

Substance use					
Nothing	368 (7)	8 (2.2)	**0.26 (0.10, 0.68)**	131 (35.6)	0.77 (0.54, 1.10)
Alcohol only	2,541 (47)	155 (6.1)	0.67 (0.40, 1.12)	640 (25.2)	**0.47 (0.34, 0.66)**
Alcohol & cannabis	1,965 (37)	109 (5.6)	0.65 (0.40, 1.07)	597 (30.4)	**0.61 (0.49, 0.75)**
Alcohol cannabis & ecstasy	278 (5)	19 (6.8)	Reference	116 (41.7)	Reference
Frequent ecstasy	140 (3)	10 (7.1)	1.03 (0.42, 2.50)	57 (40.7)	0.96 (0.66, 1.40)
Missing drug info.	79 (1)	0	~~~	79 (100)	**~~~**
Gender					
Male	2,615 (49)	88 (3.4)	Reference	849 (32.5)	Reference
Female	2,755(53)	213 (7.7)	**2.29 (1.73, 3.03)**	770 (28.0)	0.83 (0.57, 1.22)
Missing info.	1 (0)	0 (0)	**~~~**	1 (100)	**~~~**
SDQ Emotional Subscale					
Normal	4,590 (86)	229 (5.0)	Reference	1,232 (26.8)	Reference
Abnormal	371 (7)	61 (16.4)	**4.11 (3.04, 5.54)**	107 (28.8)	1.10 (0.89, 1.38)
Missing	410 (8)	11 (2.7)	1.27 (0.67, 2.42)	281 (68.5)	**5.94 (3.58, 9.84)**
Affluence Quintiles					
Lowest	1,020 (19)	60 (5.9)	Reference	418 (41.0)	Reference
2	1,004 (19)	58 (5.8)	0.80 (0.54, 1.17)	288 (28.7)	0.58 (0.48, 0.69)
3	1,016 (19)	59 (5.8)	0.76 (0.51, 1.12)	253 (24.9)	0.48 (0.37, 0.62)
4	947 (18)	54 (5.7)	0.71 (0.44, 1.13)	206 (21.8)	0.40 (0.27, 0.60)
Highest	980 (18)	60 (6.1)	0.74 (0.50, 1.10)	191 (19.5)	0.35 (0.20, 0.61)
Missing	404 (8)	10 (2.5)	0.69 (0.27, 1.79)	264 (65.4)	**2.72 (2.15, 3.43)**
Living arrangements					
Biological Parents	3,685 (69)	192 (5.2)	Reference	956 (26.0)	Reference
Reconstituted family	509 (9)	47 (9.2)	**2.27 (1.57, 3.28)**	188 (36.9)	**1.67 (1.36, 2.06)**

Single parent	1,065 (20)	60 (5.6)	1.30 (0.98, 1.71)	393 (36.9)	**1.67 (1.34, 2.07)**
Siblings / Other non-parents	88 (2)	2 (2.3)	0.98 (0.23, 4.19)	59 (67.1)	**5.81 (3.74, 9.01)**
Missing info.	24 (0)	0 (0)	~~~	24 (100)	~~~
Parental Attachment					
Most secure	1,002 (19)	34 (3.4)	Reference	257 (25.7)	Reference
2	1,002 (19)	31 (3.1)	0.89 (0.57, 1.40)	246 (24.6	0.94 (0.79, 1.13)
3	1,001 (18)	43 (4.3)	1.34 (0.77, 2.32)	285 (28.5)	1.15 (0.91, 1.46)
4	1,003 (19)	56 (5.6)	**1.73 (1.09, 2.75)**	272 (27.1)	1.08 (0.86, 1.35)
Least Secure	1,000 (19)	122 (12.2)	**4.50 (2.96, 6.83)**	311 (31.1)	**1.31 (1.02, 1.68)**
Missing	363 (7)	15 (4.1)	**3.17 (1.54, 6.53)**	249 (68.6)	**6.33 (4.11, 9.76)**
Peer Attachment					
Most secure	857 (16)	41 (4.8)	Reference	215 (25.1)	Reference
2	982 (18)	46 (4.7)	1.01 (0.64, 1.58)	268 (27.3)	1.12 (0.89, 1.41)
3	920 (17)	57 (6.2)	1.36 (0.88, 2.11)	249 (27.1)	1.11 (0.87, 1.41)
4	918 (17)	53 (5.8)	1.27 (0.85, 1.91)	254 (27.7)	1.14 (0.84, 1.56)
Least Secure	924 (17)	67 (7.3)	**1.75 (1.17, 2.63)**	297 (32.1)	**1.41 (1.00, 2.01)**
Missing	770 (14)	37 (4.8)	1.37 (0.89, 2.10)	337 (43.8)	**2.32 (1.40, 3.87)**

Table 2: Logistic regression showing the odds ratios for poor mental health at age 16 by drug use, mental health and social characteristics

	Odds Ratio (95% CI) Adjusted for gender	Odds Ratio (95% CI) + SDQ	Odds Ratio (95% CI) + Affluence	Odds Ratio (95% CI) + Living arrangements	Odds Ratio (95% CI) + Parental attachment	Odds (95% + P attach
Substance use						
Nothing	**0.24 (0.10, 0.63)**	**0.24 (0.09, 0.61)**	**0.24 (0.10, 0.62)**	**0.27 (0.11, 0.68)**	**0.38 (0.15, 0.99)**	**0.37 (0.1**
gs (Tobacco & alcohol)	0.60 (0.36, 1.01)	**0.59 (0.35, 0.98)**	**0.60 (0.36, 0.99)**	0.63 (0.38, 1.05)	0.83 (0.50, 1.38)	0.81 (0.4
drugs & cannabis	0.62 (0.37, 1.03)	**0.59 (0.36, 0.97)**	**0.59 (0.36, 0.97)**	0.61 (0.37, 1.00)	0.70 (0.42, 1.15)	0.61 (0.3
gs, cannabis & ecstasy	Reference	Reference	Reference	Reference	Reference	Refer
quent ecstasy use	0.98 (0.39, 2.48)	0.84 (0.32, 2.21)	0.85 (0.32, 2.21)	0.89 (0.34, 2.34)	0.77 (0.29, 2.04)	0.95 (0.3
Gender						
Male	Reference	Reference	Reference	Reference	Reference	Refer
Female	**2.33 (1.75, 3.09)**	**2.07 (1.53, 2.79)**	**2.06 (1.52, 2.77)**	**2.02 (1.48, 2.76)**	**2.09 (1.55, 2.82)**	**2.18 (1.2**
Emotional Subscale						
Normal	~~~	Reference	Reference	Reference	Reference	Refer
Abnormal	~~~	**3.52 (2.58, 4.80)**	**3.51 (2.58, 4.76)**	**3.5 (2.57, 4.76)**	**2.88 (2.09, 3.97)**	**2.68 (1.9**
Affluence						
east affluence	~~~	~~~	Reference	Reference	Reference	Refer
2	~~~	~~~	0.94 (0.63, 1.42)	0.95 (0.64, 1.40)	0.95 (0.64, 1.41)	1.12 (0.7
3	~~~	~~~	0.94 (0.63, 1.40)	0.97 (0.65, 1.43)	0.87 (0.59, 1.28)	1.09 (0.6
4	~~~	~~~	0.87 (0.53, 1.45)	0.92 (0.54, 1.59)	0.84 (0.49, 1.42)	1.00 (0.6
Most affluent	~~~	~~~	0.92 (0.64, 1.34)	1.00 (0.69, 1.46)	0.86 (0.59, 1.25)	0.98 (0.6
ng arrangements						
ological Parents	~~~	~~~	~~~	Reference	Reference	Refer
onstituted family	~~~	~~~	**~~~**	**2.04 (1.38, 3.02)**	**1.74 (1.16, 2.63)**	1.18 (0.2

Single parent	~~~	~~~	~~~	1.11 (0.79, 1.56)	1.02 (0.74, 1.42)	2.00 (0.3
/ Other non-parents	~~~	~~~	~~~	0.90 (0.20, 4.18)	0.75 (0.16, 3.56)	1.19 (0.2
ental Attachment						
Most Secure	~~~	~~~	~~~	~~~	Reference	Refer
2	~~~	~~~	~~~	~~~	0.91 (0.57, 1.45)	**0.28 (0.1**
3	~~~	~~~	~~~	~~~	1.39 (0.77, 2.52)	**0.24 (0.1**
4	~~~	~~~	~~~	~~~	**1.75 (1.09, 2.82)**	**0.35 (0.2**
Least Secure	~~~	~~~	~~~	~~~	**4.09 (2.55, 6.57)**	**0.45 (0.3**
eer attachment						
Most Secure	~~~	~~~	~~~	~~~	~~~	Refer
2	~~~	~~~	~~~	~~~	~~~	**0.54 (0.3**
3	~~~	~~~	~~~	~~~	~~~	**0.60 (0.3**
4	~~~	~~~	~~~	~~~	~~~	0.80 (0.5
Least Secure	~~~	~~~	~~~	~~~	~~~	0.78 (0.5

Standard errors adjusted for clustering at school level.

Regression for SMFQ>15 using Full Information Maximum Likelihood including missing data