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Pathways to ecstasy use in young adults: Anxiety, depression or behavioural deviance?

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Abstract

Aims

To investigate pathways to ecstasy use disorders from pre-birth to early adulthood with particular attention to the relationship between early depressive and anxiety symptoms and later ecstasy use disorders.

Design

Prospective, longitudinal, population-based study started in Brisbane, South East Queensland (Australia) in 1981. Participants were 2143 young adults, followed up from pre-birth to young adulthood.

Measurements

Ecstasy use disorders were assessed with the composite international diagnostic interview (CIDI-Auto). Maternal socio-economic position and mental health status were assessed at baseline (antenatal visit); maternal substance use was measured at the 5-year follow-up, adolescents' behaviour at the 5- and 14-year follow-up and tobacco and alcohol use were assessed at the 14-year follow-up.

Findings

Eight syndrome scales of childhood behaviour were examined. After adjustment for important confounders, delinquent and aggressive behaviour in early adolescence remained significantly associated with ecstasy use disorders in early adulthood. The associations became statistically non-significant when adolescent tobacco and alcohol use were included in the model [OR = 1.50 (95%CI = 0.75, 3.01) for delinquency and OR = 1.69 (95%CI = 0.92, 3.12) for aggression]. Formal mediation tests were statistically significant (p = 0.001 for delinquent behaviour and p = 0.05 for aggressive behaviour).

Conclusions

Our findings suggest a pathway from early deviant behaviour to ecstasy use disorders, possibly mediated through licit drug experimentation in early adolescence.

Keywords: Ecstasy (drug); Adolescent behaviours; Drug usage; Deviant behaviour; Longitudinal study

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1. Introduction

Since its introduction in the late 1980s, the use of ecstasy has increased exponentially and become one of the most commonly used recreational drugs among youth in the United States, Europe and Australia ([Degenhardt et al., 2004], [Degenhardt et al., 2005] and [United Nations Office on Drugs and Crime, 2006]). In Australia, ecstasy was the second most popular illicit drug amongst young people in 2004, with 12% of those aged 20–29 years reporting use in the last year (Australian Institute of Health and Welfare, 2005).

The psychoactive ingredient in ecstasy is MDMA (3,4-methylenedioxymethamphetamine), an indirect monoaminergic agonist that both stimulates release and inhibits reuptake of serotonin (5-HT). However, in Australia a significant proportion of drugs sold as ecstasy contain other psychoactive substances as well as or instead of MDMA; most often methylamphetamine and ketamine ([Australian Crime Commission, 2007] and [Fowler et al., in press]). Uncertainty regarding the contents of tablets sold as ecstasy complicates interpretation of the sequalae of ecstasy use, however a number of authors have pointed to seritonergic alterations to explain associations between ecstasy use and both cognition and affect ([Parrott, 2001], [Parrott et al., 2000] and [Thomasius et al., 2003]).

Indeed, although ecstasy is increasingly perceived as a relatively innocuous drug in some youth sub-cultures (Duff, 2005), there is cross-sectional evidence of an association between regular ecstasy use and long-term cognitive impairment, particularly impaired memory ([Parrott, 2001], [Parrott, 2002] and [Thomasius et al., 2003]). Ecstasy use has also been associated with alternations in mood both during and in the days immediately following use (Parrott and Lasky, 1998), however the evidence for longer-term mental health impacts of ecstasy use remains weak. Most studies of the link between ecstasy use and mental health have been cross-sectional in nature, and based on self-report by convenience samples of polydrug users ([Parrott, 2001] and [Parrott, 2002]), making interpretation of any associations difficult. Indeed, a recent cross-sectional study of current and past ecstasy users found that although ecstasy use was associated with seritonergic alterations and impaired verbal memory, self-reported psychopathology was associated with polydrug use in general, rather than ecstasy use in particular (Thomasius et al., 2003). More recently, a meta-analysis of 25 cross-sectional studies identified an association between ecstasy use and depressive symptomatology, however the authors noted that the clinical significance of the association was debatable, and that many of the studies included in the analysis had failed to adequately control for other drug use (Sumnell and Cole, 2005).

Even if one does accept an association between ecstasy use and impaired mental health, cross-sectional studies cannot establish the causal direction of this association. The few longitudinal studies that have assessed the nature of the relationship between common mental health disorders and ecstasy use have yielded inconsistent results ([de Win et al., 2006], [Huizink et al., 2006] and [Lieb et al., 2002b]). Two small sample size studies found no relationship ([de Win et al., 2006] and [de Win et al., 2007]), whereas two larger scale longitudinal studies found that anxiety and depression preceded ecstasy use disorders in early adulthood ([Huizink et al., 2006] and [Lieb et al., 2002b]).

A Dutch study in particular argued for a temporal pathway from impaired mental health to ecstasy use, since depressive and anxiety symptoms were assessed in the sample before MDMA appeared as a recreational drug in the Netherlands (Huizink et al., 2006). The authors also found an 81% increase in risk of ecstasy use among those reporting delinquent behaviour in childhood, raising the possibility that another pathway, common to the development of use of other drugs, may also be at play in the development of ecstasy use disorders (King et al., 2004). This may involve an indirect pathway from parental anxiety and depression (Lieb et al., 2002a), to exposure to parental use of tobacco and alcohol (Alati et al., 2005), to children's behavioural problems and early use of licit substances (King et al., 2004). These life course factors are known to predict substance use in youth (Alati et al., 2005), but their influence on regular ecstasy use in particular awaits investigation. In this study, we examine prospectively the relationship between behavioural problems

in early adolescence and ecstasy abuse in early adulthood. The study represents an important advance over existing evidence in that it uses a longitudinal design and takes into account a wide range of important confounders ranging from pre-birth to adolescence, which previous studies have not been able to account for.

2. Methods

2.1. Participants

We used data from the Mater University study of pregnancy and its outcomes (MUSP), a birth cohort study of women enrolled in the study at the Mater Misericordiae Hospital in Brisbane, Australia, between 1981 and 1983. Baseline data were collected at the first antenatal visit from 7223 consecutive women who gave birth to live singleton babies and were followed up 3–5 days, 6 months, 5, 14 and 21 years after birth. At 14 and 21 years both mothers and children were interviewed. This study uses the baseline, birth, 14- and 21-year follow-up data.

2.2. Instruments

2.2.1. Outcomes: ecstasy use disorders (CIDI-Auto)

During the 21-year follow-up, a subsample of 2551 offspring completed the Composite International Diagnostic Interview—computerised version (CIDI-Auto) (World Health Organization, 1992). Those who reported using a drug at least 'five times ever' completed the corresponding drug use section of the CIDI to assess the presence of drug-related disorders according to DSM-IV criteria. We extracted those items from the CIDI indicative of use of ecstasy.

2.2.2. Main predictors: child behaviour at age 5 and 14

Since no self-reports of child behaviour were available at the 5-year follow-up, two modified subscales of the child behaviour checklist (CBCL) were used at age 5 ([Achenbach, 1991b] and [Achenbach and Edelbrock, 1983]). The Checklist includes subscales assessing symptoms of externalising, internalising and other problem behaviours ([Achenbach, 1991b] and [Achenbach and Edelbrock, 1983]). We used a modified form of the internalising and externalising scales, which at age 5 were completed by the mother only.

At the 14-year follow-up, child behaviour was assessed using the eight syndrome subscales of the youth self-report (YSR) version of the CBCL (Achenbach, 1991a). These include the withdrawn, somatic complaints and anxious/depressed symptoms subscales (typically referred to as 'internalising' symptoms), the social, thought and attention problems subscales, and the delinquent and aggressive behaviour subscales ('externalising' symptoms). Achenbach identified scores in the top 10% as more likely to reflect symptoms of child psychopathology ([Achenbach, 1991a] and [Achenbach, 1991b]). Accordingly, scale scores were dichotomised with those in the top 10% considered to exhibit clinically significant internalising or externalising symptoms. The use of the YSR in the MUSP study has been described extensively in previous papers, which also report on the good validity and internal consistency of the scales (Alati et al., 2004).

2.2.3. Confounders and mediators

Maternal socio-economic position (SEP) was obtained at baseline and included maternal age (13–19; 20–34, 35 years or more) and education (did not complete secondary school, completed secondary school, completed further education). Sex of the child was obtained from obstetric data at birth.

We used the Delusions-Symptoms-States Inventory (DSSI) (Bedford and Foulds, 1978) to assess maternal anxiety and depression at baseline. The DSSI was developed by clinicians and validated against a clinical sample (Bedford and Foulds, 1977). It contains two 7-item subscales measuring depression and anxiety, which have been found to correlate strongly with other scales of depression including the Beck Depression Inventory (Najman et al., 2000). Bedford and Foulds and others found that a cut-off of 4 or more symptoms produced the optimum combination of false positives and false negatives, based on Bedford and Foulds' validation studies ([Bedford and Foulds, 1977] and [Rubino et al., 1997]). Consistent with the scale authors, symptoms of depression and anxiety were defined in this study as reporting 4 or more of the 7 symptoms in the DSSI depression and anxiety subscales.

Maternal alcohol and tobacco consumption were assessed at the 5-year follow-up. We obtained information on how often mothers drank (from never to daily) and how much they drank on those occasions (from 0 to 7+ glasses). These data were used to categorise the women into three groups (abstainers/occasional drinkers; <1 drink a week; 1+ drinks a few times a week). Mothers also recalled how many cigarettes they had smoked over the 7 days prior to survey. Smoking status was categorised into non-smoker, 1–19 cigarettes per day and 20 or more cigarettes per day. We used adolescents' self-reports of quantity of alcohol consumption at any drinking occasion (recoded into 'never drank/<1 full glass', '1 or 2 glasses', '3+ glasses') to assess alcohol use at age 14. Adolescents were also asked to recall tobacco use in the previous week and data were recoded into 'never smoked', '1–9 cigarettes' or '10+ cigarettes' per day.

3. Statistical analysis

We explored univariate associations between behavioural problems, potential confounders and ecstasy use disorders using chi square tests and unadjusted logistic regression. We then fitted a series of multivariate logistic regression models with predictors of adolescent delinquent and aggressive behaviour which were found to be significantly associated with ecstasy use disorders. We progressively adjusted for child's age and gender, maternal SEP, maternal mental health, alcohol and tobacco use, and child's own alcohol and tobacco use at age 14. This analysis was conducted on 2143 participants for whom complete data were available. Results are shown for males and females together, after a likelihood ratio test of the interaction term with sex found no statistical evidence for a difference in effect between males and females.

In order to test mediation effects we followed Baron and Kenny's model (Baron and Kenny, 1986) to explore relationships with possible mediating factors. We examined the relationship between our main significant predictors (delinquent and aggressive behaviours) and the presumed mediators (child smoking and alcohol use at 14 years) and formally assessed whether or not mediation was occurring using Sobel and Goodman mediation tests (MacKinnon et al., 2002). We then conducted a series of sensitivity analyses to assess whether our results were driven by having selected variables at a specific follow-up phase. We included maternal anxiety and depression, tobacco and alcohol consumption at other time periods (e.g., birth, 6 months, 5 years). We also repeated the analysis after excluding 8 people who reported an ecstasy use disorder at an age that could be concurrent with the time the 14-year follow-up took place and those who reported having ever experimented with cannabis or ecstasy at the 14-year follow-up. Finally, we computed probability weights using a logistic regression model, with the outcome being complete data or not, to account for those lost to follow-up from the 7223 original cohort members. We included inverse probability (of having missing outcome data) weights (Hogan et al., 2004) determined from the regression coefficients from this model.

4. Results

Almost 5% (*n* = 127) of participants met the criteria for an ecstasy use disorder at age 21. Table 1 reports the univariate relationship between the eight subscales of the YSR and ecstasy use disorders at age 21. The unadjusted odds ratio for the association between attention problems at age 14 and ecstasy use disorders at

age 21 was 1.64 (95%CI 0.95, 2.83), which was not statistically significant at $p < 0.05$. Adolescents who reported delinquent and aggressive behaviours at 14 years of age were at increased risk of ecstasy use disorders, with the effect being stronger for delinquent behaviour (OR = 2.71 (95%CI = 1.61, 4.54)) than for aggressive behaviour (OR = 1.85 (95%CI = 1.07, 3.19)).

Table 1: Univariate association of behaviour symptoms in childhood (child behaviour checklist) and adolescence (youth self-report) and life time diagnosis of ecstasy use disorders in early adulthood (*n* = 2433)

	MDMA (ecstasy) use disorders		
	No	Yes	Unadjusted OR
At age 5			
Internalising (CBCL)	2190	110	
Normal	95.1	4.9	1
10% range	96.4	3.6	0.72 (0.36, 1.45)
	$\chi^2 = 0.84$, $p = 0.36$		
Externalising (CBCL)	2192	110	
Normal	95.2	4.8	1
10% range	95.4	4.6	0.94 (0.50, 1.79)
	$\chi^2 = 0.03$, $p = 0.87$		
At age 14			
Withdrawn	2310	123	
Normal	94.9	5.1	1
10% range	95.3	4.7	0.92 (0.50, 1.69)
	$\chi^2 = .08$, $p = .78$		
Somatic complaints	2310	123	
Normal	95.1	4.9	1
10% range	93.6	6.4	1.32 (0.76, 2.30)
	$\chi^2 = .96$, $p = .32$		
Anxious depressed	2310	123	
Normal	94.9	5.1	1
10% range	95.3	4.7	0.91 (0.48, 1.72)
	$\chi^2 = .07$, $p = .78$		
Social problems	2310	123	
Normal	94.8	5.2	1
10% range	96.1	3.9	0.75 (0.36, 1.55)
	$\chi^2 = .06$, $p = .43$		
Thought problems	2310	123	
Normal	95.1	4.9	1
10% range	93.0	7.0	1.46 (0.85, 2.52)
	$\chi^2 = 1.91$, $p = .17$		
Attention problems	2310	123	
Normal	95.2	4.8	1
10% range	92.3	7.7	1.64 (0.95, 2.83)
	$\chi^2 = 3.22$, $p = .08$		
Delinquent behaviour	2310	123	
Normal	95.4	4.6	1
10% range	88.5	11.5	2.71 (1.61, 4.54)
	$\chi^2 = 15.39$, $p < .001$		
Aggressive behaviour	2310	123	
Normal	95.2	4.8	1
10% range	91.5	8.5	1.85 (1.07, 3.19)
	$\chi^2 = 4.96$, $p < .05$		

Table 2 reports univariate associations between life course potential confounders and lifetime diagnoses of ecstasy use disorders at age 21. Youth meeting criteria for ecstasy use disorders at age 21 were more likely to be male, born of mothers who drank and smoked when the child was 14 years of age and had themselves used alcohol and tobacco by the age of 14. Children who smoked 10 + cigarettes and had consumed 3 + glasses of alcohol at age 14 were at more than threefold greater risk of having an ecstasy disorder by age 21 [OR = 3.23 (95%CI = 1.78, 5.87) for tobacco use and OR = 3.56 (95%CI = 2.12, 5.98) for alcohol use].

Table 2: Univariate associations of potential confounding factors with life time diagnosis of ecstasy abuse/dependence in early adulthood

	Total (*n*)	MDMA (ecstasy) disorders		
		No	Yes	Unadjusted OR
Mother's age	2551	2424	127	
35 years plus		93.7	6.3	1
20–34 years		95.2	4.8	0.75 (0.36, 1.58)
13–19 years		94.4	5.5	0.88 (0.37, 2.05)
		$\chi^2 = 0.85$, $p = .66$		
Maternal education	2533	2407	126	
Post high		95.2	4.7	1
Complete high		94.7	5.3	1.12 (0.70, 1.78)
Incomplete high		96.0	3.9	0.82 (0.43, 1.57)
		$\chi^2 = 1.30$, $p = .52$		
Maternal mental health and substance use				
Maternal depression in pregnancy	2516	2392	124	
Not depressed		95.1	4.8	1
Depressed		93.4	6.5	1.36 (0.58, 3.18)
		$\chi^2 = 0.52$, $p = .47$		
Anxiety in pregnancy	2512	2388	124	
Non-anxious		95.0	5.0	1
Anxious		95.6	4.3	0.86 (0.46, 1.63)
		$\chi^2 = 0.21$, $p = .65$		
Maternal alcohol use at 5 years	2286	2178	108	
Abstainers/occasional drinkers		95.2	4.7	1
<1 drink a week		95.3	4.6	0.97 (0.51, 1.55)
1+ at few times a week		95.5	4.4	0.93 (0.42, 2.04)
		$\chi^2 = 0.04$, $p = .98$		
Maternal tobacco use at 5 years	22862	21786	108	
Nil		95.8	4.1	1
1–19 a day		94.2	5.7	1.40 (0.87, 2.26)
20+		94.2	5.7	1.41 (0.85, 2.34)
		$\chi^2 = 2.96$, $p = .23$		
Child's individual characteristics				
Child's gender	2551	95.0	4.9	
Male		93.9	6.0	1
Female		96.0	3.9	0.63 (0.44, 0.90)
		$\chi^2 = 6.38$, $p < .01$		
Child's alcohol use at age 14	2425	2302	123	
Never/<1 glass		96.0	3.9	1
1 or two glasses		90.3	9.6	2.58 (1.56, 4.25)
3 or more		87.1	12.9	3.56 (2.12, 5.98)
		$\chi^2 = 34.26$, $p < .001$		
Child's smoking previous week at 14	2424	2301	123	
Never smoked		95.5	4.5	1
1–9 cigarettes		92.0	7.9	1.82 (0.95, 3.49)
10+ cigarettes		86.7	13.2	3.23 (1.78, 5.87)
		$\chi^2 = 18.39$, $p < .001$		

Table 3 shows progressive multivariate regression models of the relationship between adolescent delinquent and aggressive behaviour and lifetime ecstasy use disorders at age 21. Those who reported aggressive behaviour in adolescence had almost twice the odds of developing ecstasy use disorders by age 21, whereas those who reported delinquent behaviour were more than twice as likely to meet DSM-IV criteria for an ecstasy use disorder by age 21. Inclusion of child's own smoking and drinking in the model attenuated the association towards the null.

Table 3: Multivariate associations of externalising behaviour and tobacco and alcohol use in adolescence and life time use of ecstasy in early adulthood (complete case analysis *n* = 2143) (OR 95%CI)

Externalising behaviour at child age 14 (YSR)	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Delinquent behaviour						
Normal	1	1	1	1	1	1
10% range	2.62 (1.47, 4.67)	2.56 (1.44, 4.56)	2.58 (1.44, 4.61)	2.55 (1.42, 4.57)	2.00 (1.01, 3.93)	1.50 (0.75, 3.01)
Aggressive behaviour						
Normal	1	1	1	1	1	1
10% range	2.25 (1.27, 4.00)	2.25 (1.27, 4.00)	2.28 (1.28, 4.07)	2.19 (1.22, 3.93)	1.84 (1.00, 3.40)	1.69 (0.92, 3.12)

Model 1: unadjusted; Model 2: sex and age adjusted; Model 3: previous + maternal socio-economic position and mental health in pregnancy; Model 4: previous + maternal substance use at 5 years; Model 5: previous + child's tobacco use at age 14; Model 6: previous + child's alcohol use at age 14.

Adolescent smoking and drinking were both associated with delinquent ($p = 0.001$) and aggressive behaviour ($p = 0.02$). We fitted a series of regression models to examine the relationships between adolescent tobacco and alcohol use and young adults' ecstasy disorders and found that tobacco and alcohol use were associated with young adults' ecstasy use ($p = 0.001$). When we conducted the Sobel–Goodman tests to formally test for mediation effects we found that the attenuation of the association between behaviour and subsequent ecstasy use was statistically significant ($p = 0.001$ for delinquent behaviour and $p = 0.05$ for aggressive behaviour) suggesting that experimentation with licit drugs in early adolescence may mediate the relationship between externalising behaviour and ecstasy use disorders. Sensitivity analyses (a) using maternal mental health, tobacco and alcohol consumption at other time periods, and (b) excluding cases who met criteria for an ecstasy use disorder at a time concurrent with measurement of behaviour, or who reported use of illicit drugs at 14 years, did not substantively alter the results; nor did the weighted analysis. Those lost to follow-up were more likely to be male ($p = 0.001$), and more likely to be born of mothers who were less educated ($p = 0.01$), single ($p = 0.04$), smoking tobacco ($p = 0.03$) and anxious ($p = 0.03$) and depressed ($p = 0.002$) at baseline. In the fully adjusted weighted analysis, the odds ratio for the association of behaviour with ecstasy use disorders was 1.60 (95%CI = 0.77, 3.34) for delinquent behaviour and 1.80 (95%CI = 0.93, 3.48) for aggression. The odds ratio for the association between anxious/depressed behaviour and ecstasy use disorders was 1.00 (95%CI = 0.50, 2.01) in the fully-adjusted, weighted analysis.

5. Discussion

In a large birth cohort study of Australian youth, with measures similar to those used by Huizink and colleagues (Huizink et al., 2006), we did not find any evidence of a relationship between symptoms of anxiety and depression in childhood or adolescence and increased risk of ecstasy use in early adulthood. Further, we found that distal socio-economic, parental and individual factors including early signs of externalising and internalising symptoms measured at age 5 did not predict ecstasy use disorders. To the contrary, we found that the most likely pathway leading to ecstasy use disorders is similar to that identified for other recreational drugs used by youth in Australia and overseas; involving symptoms of aggressive and delinquent behaviour, possibly mediated by the use of tobacco and alcohol in early adolescence (King et al., 2004). These results held after sensitivity analyses were conducted to assess the robustness of the findings.

Our study did not support evidence of a relationship between early signs of anxiety and depression at either 5 or 14 years and increased risk of ecstasy use disorders in early adulthood. Our findings are consistent with two small scale longitudinal studies ([de Win et al., 2006] and [de Win et al., 2007]), where no association was found between common mental health disorders and later ecstasy use. There is a range of reasons why our results might differ from those of others. Perhaps the age group at which ecstasy abuse was assessed in the MUSP cohort was slightly younger that that of other longitudinal studies. It could also be argued that differences in our results may be due to the different measures used. For example, Huizink and colleagues

(2006) focused on ecstasy use rather than abuse and Lieb and colleagues (2002b) measured mental health disorders rather than behavioural symptoms.

Longitudinal evidence on a relationship between behaviour and regular ecstasy use is scant ([Huizink et al., 2006] and [Lieb et al., 2002b]). One study did not focus on delinquent behaviour (Lieb et al., 2002b) and another study found a relationship between anxious and depressive symptoms and ecstasy use, but no significant association between delinquent behaviour and ecstasy use (Huizink et al., 2006). Nevertheless, it is worth noting that a statistically non-significant increase in risk of ecstasy abuse was also evident amongst those with greater delinquent behaviour in this study (Huizink et al., 2006). More broadly, our findings are in line with existing literature suggesting that both aggressive and delinquent behaviours and substance use are behaviours indicative of a relatively disinhibited, sensation-seeking personality style ([Disney et al., 1999], [Weinberg et al., 1998] and [Zuckerman, 1993]). Consistent with this, the results from our study point to a non–specific pathway from early behavioural disinhibition, including early adolescent rule-breaking behaviour, leading to ecstasy use in early adulthood. In addition our findings suggest that this pathway is possibly mediated through early experimentation with alcohol and tobacco.

5.1. Strengths of the study

To our knowledge this is the first study with a birth cohort design examining the relationship between behaviour and ecstasy use disorders, with measures ranging from pregnancy to 21 years of age. Our study adds to the existing evidence in that we were able to account for a range of lifetime factors which could explain associations between early behaviour and ecstasy use, but which had never been accounted for in the literature before. We adjusted for maternal depression and anxiety, which have been found to be associated with children's problem behaviour and substance abuse disorders ([Alati et al., 2005] and [Lieb et al., 2002a]), and maternal alcohol and tobacco use, and found that these only mildly reduced the strength of the association between delinquent and aggressive behaviour and ecstasy use. Further, this is the first study able to take into account the effect of early use of alcohol and tobacco and the first study to suggest that early experimentation with these licit drugs may explain at least some of the relationship between externalising behaviour in adolescence and later ecstasy use.

5.2. Limitation of the study

Unlike Huizink and colleagues, we cannot claim that ecstasy was introduced in the Australian market after the behavioural assessment at child age 14. However, the fact that our findings held after exclusion of reports of drug use which may have been concurrent with the assessments at age 14 suggests that the temporal sequence is likely to lead from externalising/licit drug use to ecstasy use rather than vice versa. It is also important to acknowledge that reports of delinquent and aggressive behaviour and tobacco and alcohol use were obtained during the same wave of data collection, and it is therefore impossible to determine whether behavioural problems preceded licit drug use, or vice versa. Although the latter interpretation seems less plausible and would contradict established evidence of a pathway from early behavioural problems to early tobacco and alcohol use ([King et al., 2004], [Loeber et al., 1999] and [Lynskey and Fergusson, 1995]), we cannot exclude it. Future studies with the capacity to better assess this temporal sequence are needed to confirm the possible mediating effect of alcohol and tobacco use in the relationship between behaviour and ecstasy use disorders.

Similarly, we could not test for a ‘gateway’ pathway (Hall and Lynskey, 2005) leading from licit drug experimentation to cannabis initiation, and through to ecstasy disorders, since the study did not have longitudinal data on cannabis use in adolescence. Future studies with the capacity to assess the temporal sequence between the onset of tobacco, alcohol and cannabis use and ecstasy use disorders should explore interrelations amongst these substances and later ecstasy use disorders.

The main limitation of this study is loss to follow-up. This analysis was conducted on a subsample of the initial birth cohort for which data on the CIDI were available. This group represents about 35% of the 7223 offspring and such loss may introduce bias in our results. Comparisons between those lost and those still in the study suggest that we may be under-estimating both the prevalence of ecstasy disorders and the strength of the associations we found. In particular, participants who were lost to follow-up were born of mothers who were more likely to be anxious and depressed at baseline. This may have resulted in a non-significant finding for the association between anxiety and depression and later ecstasy use disorders. In order to explore this possibility and the direction of bias, we attached inverse probability weighting to subjects included in the analyses, to restore the representation of those lost to follow-up. We found no substantive differences between the weighted and non-weighted results, which suggests that attrition is unlikely to have substantively biased our findings in either direction.

Despite the fact that the present study employed a wider range of covariates than any other study previously published on this subject, we were unable to assess a number of potentially important factors. The lack of an association between ecstasy use disorders and behaviour in childhood and other early factors, and the presence of an association with behaviour and lifestyle factors measured in adolescence, would suggest that environmental and modelling influences are stronger predictors of ecstasy use disorders than genetic or biological influences. However, we did not have access to genetic measures and could not test empirically for gene-environment interactions. Other measures our study could not take into account included societal and paternal influences such as peer group modelling, and paternal mental health, behaviour and substance use. By including measures of these factors, future longitudinal studies may be able to more fully explicate the causal pathways identified here.

Finally, the average age of onset of ecstasy use in Australia is 22.8 years (Australian Institute of Health and Welfare, 2005), so in our data, collected at 21 years of age, we would have identified only a proportion of those who would use ecstasy by their mid-twenties, with a bias towards those with earlier onset, who are also likely to be those exhibiting more chronic and harmful patterns of drug use, and higher rates of antisocial behaviour (Disney et al., 1999). Because of this, if we had assessed ecstasy use at, say, 25 years of age, the association between delinquent/aggressive behaviour and ecstasy use may have been weaker or we may have identified other predictors (e.g., anxiety and depression) of ecstasy use. Further follow-up of this cohort will be needed to assess associations between behavioural problems and ecstasy use amongst those who initiate ecstasy use later in young adulthood.

5.3. Conclusions and public health implications

Our findings did not support evidence of an association between anxiety and depression in adolescence and the development of ecstasy use disorders in early adulthood. To the contrary, we identified a non-specific pathway from early deviant behaviour to ecstasy use disorders, possibly mediated through licit drug experimentation in early adolescence. To the extent that these findings can be replicated, they suggest that pathways to ecstasy use in young adulthood may differ little from pathways to use of other illicit drugs. Intervention strategies targeting impaired mental health as a key factor in the uptake of ecstasy use may therefore be missing the mark. Taken together with previous studies, these findings suggest that there may be little that is unique about pathways to ecstasy use, and that strategies to prevent use or minimise harm associated with ecstasy use can be adapted from strategies targeting other drugs. Furthermore, given the potential mediating role of licit drugs in the development of ecstasy use disorders in this sample, public health strategies aimed to reduce ecstasy use and harm in young adults should acknowledge the importance of targeting early use of tobacco and alcohol. Given the significant health harms caused directly by these licit substances, such strategies may have both immediate and longer-term benefits.

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