Abstract

Aims

To examine the consequences of alcohol consumed and symptoms of alcohol use disorder during adolescence in adulthood.

Design and Methods

A longitudinal, prospective birth cohort study, the Christchurch Health and Development Study (CHDS) was examined across a 35 year period. We estimated the associations between two measures of adolescent alcohol use (amount of alcohol consumed; symptoms of alcohol use disorder) and a series of mental health, substance use and psychosocial outcomes in adulthood, adjusting for individual, behavioural and family covariates.

Results

The pattern of results indicates symptoms of alcohol use disorder were predictive of mental health disorders in adulthood. Volume of alcohol used in adolescence predicted increased risk of substance use disorders, lower educational attainment, and higher risk of welfare dependence in adulthood.

Discussion and Conclusion

Early consumption of larger volumes of alcohol led to continuation of this pattern in adult life with resulting poorer educational achievement, increased welfare dependence and substance use disorders. Early symptoms of Alcohol Use Disorder, however, led to increased adult levels of mental health disorders. This relationship persisted across a 20-year study period and after adjustment for statistically significant covariate factors. The study shows that early patterns of alcohol use have a direct and specific impact upon adult outcomes. This understanding will be of use to clinicians, policy-makers and researchers.

Keywords: Underage drinking, Alcoholism, Mental Health, Cohort studies, Public Health

Introduction

Mental Health and Alcohol Use disorders now account for half of the leading causes of disability(1, 2). The 2006 New Zealand Mental Health Survey, Te Rau Hinengaro(3) provides robust data showing a 46.6% lifetime prevalence of mental health disorders in New Zealand, these being associated with higher levels of physical co-morbidities and increased need for healthcare. In New Zealand more than 1 in 20 adults have a lifetime history of any alcohol use disorder(4). Alcohol is the most commonly used substance of dependence, carrying a correspondingly high burden of disease. There is a high level of co-existence between substance use and mental health disorders, with both groups at risk of inequitable physical health outcome. Adding to our understanding of this complex interaction between the variables that lead to wide-ranging adverse outcomes is therefore of clear importance.

The relationship between teenage drinking patterns and adult health and psychosocial outcomes is of wide interest. This is true both clinically and for effective early intervention and healthy policy-making. There is considerable literature making clear links between early alcohol use and later problems(5). These problems are both poorer psychosocial and mental health(6) and addictions(7) outcomes; though the effects of specific alcohol use patterns on specific adult outcomes other than alcohol use are poorly described. The least bias methodology for examining such links is a general population cohorts(8). This literature makes clear multiple risk factors for early alcohol use exist including: genes, ethnicity, personality, adversity(9), social milieu(10) and parental problems(11). What remains less understood is how these factors interlink, and the subsequent pattern of adult psychosocial, addictions and mental health problems. Cross sectional studies provide an indication of association but are unable to assess impact over time. An ability to assess for multiple covariates is also a weakness, with so many factors implicated, a failure to control of major individual, psychosocial and familial factors raises the issue of confounding. Of particular note in the cohort studies examined are the problems of recall bias of alcohol use, the failure of a general population sample, or the use of samples started in the adolescent period. These factors create significant weaknesses in the literature base that have been identified as requiring assessment using a general cohort sample(12).

Worsening these problems is translation into the clinical or public health space. Understanding the clinical continuum of alcohol use disorder is therefore essential to enable translation, if complex. In the DSM-5 diagnostic taxonomy, criteria for Alcohol Use Disorder requires a pattern of compulsive and harmful alcohol use occurring over at least one year(13). Further the diagnosis is a hybrid dimensional and categorical one, although this probably reflects the biological basis of the disorder and the growth of the taxonomy over six decades(14). The diagnosis of Use Disorder is a permanent one, with the possibility of specification as being in sustained remission, making early detection and prevention important. This is particularly so as evidence suggest the diagnosis is fragile in youth, with 50% of teenagers who meet criteria for alcohol use disorder no longer doing so in early adulthood(15). As such understanding the pattern of use in early adolescence, and subsequent adult problems provides context both for the clinician and public health physician.

In order to address these complex problems we examined The Christchurch Health and Development Study. Comprehensive data has been collected on this sample using validated tools across the domains of physical and mental health, substance use and welfare. The prospective, longitudinal nature of the study broadens the evidence base, largely eliminated recall bias and allows the demonstrations of causality rather than mere association as can occur with cross-sectional studies. Using this sample we examined the longitudinal associations between early alcohol use disorder symptoms and volume consumed per drinking episode, and later adult mental health and psychosocial outcomes. Specifically the objectives of the study were to examine two metrics of early alcohol use: volume of alcohol consumed and symptoms of alcohol use disorder and four adult outcomes: mental health status, use disorder status, educational attainment and social welfare status. These adult outcomes were chosen as proxy markers of overall mental wellness and societal integration.

Methods

In order to examine this question, we estimated the associations between two measures of adolescent alcohol outcomes (amount of alcohol consumed; symptoms of alcohol use disorder) and a series of mental health, substance use and education employment outcomes in adulthood, adjusting for a set of individual, behavioural and family factors.

Participants

The data were gathered from the Christchurch Health and Development Study (CHDS). In this study a birth cohort of 1265 children (635 males, 630 females) born in the Christchurch (New Zealand) urban region in mid-1977 has been studied at birth, 4 months, 1 year and annually to age 16 years, and again at 18, 21, 25, 30 and 35 years (16, 17). The original cohort was comprised of 97% of all individuals born in Christchurch during the study entry period. All study information was collected on the basis of signed consent from study participants and is fully confidential. All aspects of the study have been approved by the Canterbury (NZ) Ethics Committee. Sample sizes ranged from 1025 (age 18) to 962 (age 35), representing 79% to 82% of the surviving sample at each observation. The primary driver of sample loss over the course of the study has been emigration from New Zealand, with loss of contact.

Outcome measures (lifetime measures of mental health disorders, substance use disorders, educational attainment, and welfare dependence).

Data collected in adulthood (from 18 years of age) were used to create the following outcome measures:

1. *Mental health disorders (18-35 years).* Based on the CIDI at ages 21, 25, 30 and 35 years, cohort members were classified as to whether they met DSM-IV criteria for major depression and anxiety disorder over the intervals 18-21 years, 21-25 years, 25-30 years, and 30-35 years. Participants who met criteria for either disorder during any assessment period were classified as having that disorder.
2. *Substance use disorders (18-35 years).* Using information from the CIDI, cohort members were classified as meeting DSM-IV criteria for alcohol use disorder, nicotine dependence, cannabis dependence and other drug dependence over the intervals 18-21 years, 21-25 years, 25-30 years, and 30-35 years. Again, participants who met criteria for any disorder during any assessment period were classified as having that disorder.

3. *Educational attainment.* At ages 21, 25, 30 and 35 years, cohort members were asked a series of questions concerning their educational activities, enrolment in educational programmes, and any qualifications they had obtained. This information was used to create a comprehensive measure of educational qualifications obtained to age 35 years, classified according to a seven point scale ranging from no qualifications to University degree attainment.
4. *Welfare dependence.* At ages 25, 30, and 35, cohort members were asked detailed questions concerning their receipt of social welfare benefits since the previous assessment. Cohort members who reported receiving one or more benefit during an assessment period were classified as being welfare dependent during that period (21-25 years; 25-30 years; 30-35 years). For the purposes of the present measure, cohort members who reported receipt of at least one benefit in any assessment period were classified as having been welfare dependent.

Primary alcohol-related predictors (amount of alcohol consumed; alcohol problems), ages 14, 15 and 16 years

As part of the assessment at ages 14, 15 and 16 years, cohort members were asked a series of questions concerning their alcohol consumption, and any problems they may have had relating to alcohol consumption. The questions concerning problems with alcohol were based on the Diagnostic Interview Schedule for Children (DISC) (18) at ages 15 and 16 years, in order to obtain information pertaining to DSM-III-R (ages 15 and 16) (19) symptoms of alcohol abuse/alcohol dependence (alcohol use disorder). These data provide a count measure of the number of symptoms of alcohol use disorder during each 12 month period following the previous assessment. Therefore, the present analyses used two outcome measures:

1. *Amount of alcohol consumed*. For the 12 months prior to each assessment at ages 14, 15, and 16, the amount of alcohol consumed by cohort members in a "typical" drinking session was the basis for the first primary alcohol-related predictor. These data were summed across all three assessments, and then used to classify participants on a three-level measure representing the lowest 80%, 81 to 95%, and finally the top 5% on the measure.
2. *Number of problems with alcohol reported.* Again, for the 12 months prior to each assessment at ages 15 and 16 years, the number of problems stemming from alcohol consumption was the basis of the second primary alcohol-related predictor. These data were summed across the two assessments, and then used to classify participants on a three-level measure representing the lowest 80%, 81 to 95%, and finally the top 5% on the measure.

Covariate factors

21covariate factors were included in the analyses, on the basis that they were: a) used in a prior analysis of the age of first alcohol use in the CHDS cohort (20); or b) have been found to be related to substance use outcomes in other studies of the CHDS cohort (21-26). These fell into three broad domains including: socio-economic/demographic, personality/behavioural and family/parental. There are described in detail in the online supplement.

These included:

Statistical analyses

The first set of analyses (shown in Table 3, below) modelled the unadjusted associations between the two primary alcohol-related predictors and the adult outcomes using logistic regression (for

dichotomous outcomes) and negative binomial regression (for the measure of level of education). Both predictors were entered into the model simultaneously. These models were of the form:

$$f(Yi) = B0 + \sum BijXij + ei \quad \text{(EQ1)}$$

where Y was either the log odds (for dichotomous measures) or the log rate (for the educational achievement measure) for person i; B0 was the intercept term; ∑BijXij represented the value for the predictor for person i; and ei was a random error term.

In the second step of the analyses, the models shown above were extended to include the set of covariate factors drawn from those factors found to be statistically significantly correlated ($p < .05$) with the two primary alcohol-related predictors noted above. These models were of the form:

$$f(Yi) = B0 + \sum BijXij + \sum BijXij + ei \quad \text{(EQ2)}$$

where ∑BijXij represented the set of covariate factors for person i. Covariate factors were entered into the model using methods of forward and backward variable substitution in order to obtain stable and parsimonious models. All models were fitted using SAS 9.4 (27).

CHDS research has been approved by the Southern Health and Disability Ethics Committee (New Zealand).

Results

Bivariate associations between adolescent alcohol-related predictors and lifetime mental health, substance use, education and welfare dependence outcomes (ages 18 to 35 years)

Table 1 shows the cohort divided into three groups across two measures. The first is a measure of the amount of alcohol consumed during the 12 months prior to ages 14, 15, and 16 years, classified as the lowest 80%, 81 to 95%, and finally the top 5% on the measure. The second is a measure of symptoms of alcohol use disorder at ages 15 and 16 years, again classified as the lowest 80%, 81 to 95%, and finally the top 5% on the measure. The Table shows the percentage of each group meeting criteria for each of several mental health and substance use disorders over the period 18 to 35 years, as well as the mean score on the measure of educational attainment, and the percentage of each group who had been welfare dependent at some point during the period 21 to 35 years. Linear tests of association were derived from oneway ANOVA. The Table shows that:

1. Increasing levels of both alcohol consumption and symptoms of alcohol use disorder were associated with significantly ($p < .05$) higher rates of both depression and anxiety.
2. Alcohol consumption and symptoms alcohol use disorder were also associated with significantly ($p < .0001$) higher risks of alcohol use disorder, nicotine dependence, cannabis dependence, and other illicit drug dependence from age 18 to age 35.
3. Both alcohol consumption and level of alcohol symptoms were associated with significantly ($p < .001$) lower levels of educational attainment to age 35, and significantly ($p < .05$) greater risk of welfare dependence.

Table 1. Associations between alcohol-related predictors in adolescence (ages 14-16) and lifetime mental health, substance use, education and welfare dependence outcomes (from ages 18 to 35 years)

Outcome	Amount of alcohol consumed (ages 14-16)				Alcohol use disorder symptoms (ages 15-16)			
	1 (1-80%)	2 (81-95%)	3 (96-100%)	p	1 (1-80%)	2 (81-95%)	3 (96-100%)	p
Mental Health								
% major depression (ages 18-35)	45.3	54.0	55.2	<.05	43.2	63.4	74.4	<.0001
% anxiety disorder (ages 18-35)	38.0	32.7	51.7	<.05	36.2	41.8	59.0	<.01
Substance Use								
% alcohol use disorder (ages 18-35)	41.5	61.3	70.7	<.0001	42.3	59.0	79.5	<.0001
% nicotine dependence (ages 18-35)	27.4	55.3	70.7	<.0001	29.5	52.5	76.9	<.0001
% cannabis dependence (ages 18-35)	10.3	21.3	36.2	<.0001	10.5	26.9	28.2	<.0001
% other illicit substance dependence (ages 18-35)	4.1	8.7	25.9	<.0001	4.5	12.7	18.0	<.0001
Education/Welfare dependence								
Mean (SD) education level (to age 35)	4.58 (2.22)	3.71 (2.32)	2.81 (2.23)	<.0001	4.47 (2.24)	3.88 (2.46)	3.15 (2.20)	<.001
% welfare benefit receipt (ages 21-35)	38.3	52.0	56.9	<.0001	40.0	47.0	54.1	<.05

Bivariate associations between covariate factors and alcohol-related predictors (ages 14-16)

As noted in Methods, it could be argued that the associations between the alcohol-related predictors and life course outcomes shown in Table 1 could be due to the influence of a series of individual, family and behaviour factors to which cohort members were exposed during childhood and early adolescence. In order to examine this issue, a series of potential covariate factors were extracted from the CHDS database, and the bivariate associations between each predictor and the two alcohol-related predictors were estimated using Pearson product-moment correlations. The results of these analyses are shown in Table 2, which displays the correlation coefficient for each association, and tests of significance. The Table shows that, almost without exception, there were statistically significant correlations between alcohol-related predictors in adolescence and a range of sociodemographic, family functioning and individual factors in childhood and early adolescence. This pattern of correlations clearly shows that those reporting higher levels of alcohol consumption and a greater number of symptoms of alcohol use disorder in adolescence were exposed to higher levels of adversity and risk in childhood and early adolescence. An exception to this pattern was for family living standards and SES, both of which showed positive correlations with higher levels of alcohol consumption and alcohol symptoms. This pattern may reflect a socioeconomic gradient in which young people from more affluent families were able to obtain and consume alcohol more easily.

Table 2. Correlation coefficients for associations between covariates and alcohol-related predictors in adolescence.

Covariate	Amount of alcohol consumed (ages 14-16)	Alcohol use disorder symptoms (ages 15-16)
Sociodemographic Factors		
Maternal age	-.13***	-.08*
Maternal education level	-.11**	-.09**
Paternal education level	-.08*	-.05
Family living standards (ages 0-10) †	-.09**	-.07*
SES at birth†	-.13***	-.07*
Average family income rank (0-10 years)	-.13***	-.07*
Maori ethnicity (at birth)	.14***	.12***
Family Functioning		
Parental alcohol problems	.09**	.12***
Parental criminal offending	.12**	.11**
Parental illicit drug use	.10**	.11**
Family adversity score	.22***	.17***
Parental IPV	.14***	.16***
Parental attachment (age 15)	-.24***	-.28***

Changes of parents to age 15	.17***	.12***
Parental weekly alcohol consumption (age 11)	.01	-.02
Parent attitudes toward children's drinking (age 15)	.07*	.05
Parent attitudes toward alcohol (age 15)	.12***	.10**
Individual Factors		
Conduct problems (ages 7-9)	.18***	.20***
Attention problems (ages 7-9)	.14***	.16***
Gender (female)	-.10**	-.01
Neuroticism (age 14)	.03	.15***
Novelty seeking (age 16)	.26***	.30***
Childhood sexual abuse	.09**	.12***
Childhood physical abuse	.08*	.07*

* p<.05
** p<.01
*** p<.001

† NB: These measures were scored such that lower scores = higher adversity

Multivariate associations between alcohol-related predictors in adolescence (age 14 to 16 years) and lifetime mental health, substance use, education and welfare dependence outcomes (ages 18 to 35 years), after adjustment for covariate factors.

In the next step of the analyses, the pair of alcohol-related predictors were entered into a series of logistic regression models, in which lifetime mental health and substance use disorders and welfare dependence were modelled as a function of both adolescent alcohol consumption and symptoms of alcohol use disorder. Furthermore, a negative binomial model was fitted that modelled educational attainment as a function of both adolescent alcohol consumption and symptoms of alcohol use disorder.

In the final step of the analyses, these models were extended to include the set of covariate factors displayed in Table 2 (above). The results of both of these modelling steps are shown in Table 3, which shows the parameter estimates and standard errors for the associations between alcohol-related predictors and each outcome, both in the unadjusted (both alcohol-related predictors only) and adjusted models (both alcohol-related predictors and the set of statistically significant covariate factors). This modelling strategy allowed us to directly compare the relative magnitude and significance of the effect of both predictors (alcohol consumption; alcohol disorder symptoms) in unadjusted and adjusted models. The Table shows:

1. For mental health outcomes (major depression and anxiety disorder), both the unadjusted and adjusted models show that the amount of alcohol consumed during the period 14 to 16 years was no longer a statistically significant predictor of lifetime mental health disorder. On the other hand, three of the four models (the adjusted model predicting anxiety disorder) found a statistically significant ($p < .05$) association between alcohol use disorder symptoms and mental health disorders in adulthood.

2. For substance use disorder outcomes (alcohol, nicotine, cannabis, and other illicit drugs) the opposite pattern was found, in which the amount of alcohol consumed in adolescence showed robust and statistically significant ($p < .05$) associations with each outcome in both unadjusted and adjusted models. However, while symptoms of alcohol use disorder were robust and statistically significant ($p < .05$) predictors for alcohol use disorder and nicotine dependence, they were not significantly associated with cannabis dependence in the adjusted model, or with other illicit drug dependence in either model.
3. As with substance use disorder outcomes, the amount of alcohol consumed in adolescence was significantly ($p < .05$) associated with educational attainment and welfare dependence in both unadjusted and adjusted models. However, symptoms of alcohol use disorder in adolescence were not significantly associated with educational achievement or welfare dependence in either model.

Across the eight models, statistically significant ($p < .05$) covariate factors included: maternal age; maternal and paternal education level; socioeconomic status at birth; family living standards; parental illicit drug use; family adversity; parental intimate partner violence; gender; childhood conduct and attention problems; novelty seeking; neuroticism; childhood sexual abuse; and childhood physical abuse.

This pattern of results suggests that while early indications (symptoms) of alcohol use disorder were more predictive of greater risk of mental health disorder in adulthood than the amount of alcohol consumed, this pattern reversed for increased risk of substance use disorders, lower educational attainment, and higher risk of welfare dependence.

Table 3. Parameter estimates for the associations between alcohol-related predictors in adolescence (ages 14-16) and lifetime mental health, substance use, education and welfare dependence outcomes (from ages 18 to 35 years), before and after adjustment for covariate factors

		Amount of alcohol consumed (ages 14-16)			Alcohol use disorder symptoms (ages 15-16)		
Outcome		B	SE	p	B	SE	p
Mental Health							
Depression (ages 21-35)	Unadjusted	-.07	.13	>.60	.79	.16	<.0001
	Adjusted	-.06	.14	>.60	.67	.17	<.0001
Anxiety disorder (ages 21-35)	Unadjusted	-.06	.13	>.60	.40	.15	<.01
	Adjusted	-.11	.15	>.40	.17	.16	>.30
Substance Use							
Alcohol use disorder (ages 21-35)	Unadjusted	.51	.14	<.001	.50	.16	<.01
	Adjusted	.40	.15	<.05	.42	.16	<.05
Nicotine dependence (ages 21-35)	Unadjusted	.79	.14	<.0001	.62	.16	<.0001
	Adjusted	.56	.15	<.001	.46	.17	<.01
Cannabis dependence (ages 21-35)	Unadjusted	.62	.16	<.0001	.45	.18	<.05

	Adjusted	.53	.18	<.01	.11	.21	>.60
Other illicit substance dependence (ages 21-35)	Unadjusted	.82	.21	<.0001	.38	.24	>.10
	Adjusted	.63	.22	<.01	.08	.26	>.70
Education/Economic							
Education level (to age 35)	Unadjusted	-.21	.04	<.0001	-.05	.04	>.20
	Adjusted	-.12	.04	<.01	.00	.04	>.90
Welfare benefit receipt (ages 21-35)	Unadjusted	.43	.13	<.01	.05	.15	>.70
	Adjusted	.36	.15	<.05	-.17	.17	>.38

Nb: Unadjusted models contain both alcohol-related predictors.

Discussion

This study was designed to examine two metrics of early alcohol use: volume per drinking episode, and use disorder symptoms and adult outcomes for a general population sample. The analysis provides clear patterns of differing teenage drinking in the general population and their impact upon adult life. Specifically teenagers who drank heavily continued to do so as adults, with consequential lower educational outcomes, higher level of welfare dependence and higher levels of substance use disorder. This is clinically explained by the short-to-medium-term, dose-related effect of alcohol on loss of sleep, loss of study time, apathy and anhedonia resulting in lower performance.

In contrast, teenagers with symptoms of Alcohol Use Disorder developed higher levels depression as adults. This is clinically relevant given the proven correlation between alcohol use and depression, and the difficulty in treating depression in the context of continued alcohol use. Early symptoms of alcohol use disorder may reflect higher levels of intrinsic psychopathology at a stage of life when the diagnosis of AUD is recognised as fragile. If not treated early, there can be a vicious cycle where both factors exacerbate each other, resulting in the fusion of psychopathology and early disordered alcohol use into an established AUD with associated mental health disorder requiring transition to specialist treatment.

The primary strength of this study is its analysis of a large, prospective, general population cohort examined from birth to age 35. This eliminates problems of: generalisability of the research, at least to other western countries, recall bias related to alcohol use, recall bias of personality and psychopathology. Further multiple sources of information allow for collateral checking of many psychosocial covariates and their inclusion in statistical modelling. By using peer reviewed and described tools where possible, the variable assessed are relevant to a general population. Further the background literature and study design make causal link between early alcohol use and the later examined events highly plausible as there is consistency of effect both within this study and with preliminary data from other studies. Features of the study which indicate high likelihood of a

causative relationship are the longitudinal, prospective nature of a large cohort drawn from the general population and persistence of the relationship between primary variables and outcomes over a 20-year study period and after adjustment for covariate factors. As such these findings are robust, well-adjusted and fulfil the classical interpretation of the Bradford-Hill criteria(28) and the cautions he divined(29).

The primary limitation to this study is its inability to explicitly take into account genetic and prenatal factors. Specific examination of these factors may lead to increased understanding of the interactions between nature and nurture, another element of importance in understanding all psychopathology(30), of which addiction is part. Having said this, such research is in its relative infancy, with addictions specialists recognising the need for growth in the field(31). A further limitation is the largely European and western context of the study. This limits its generalisability to dissimilar populations, albeit a strength in its ability to generalise to similar areas of the world.

This analysis displays a likely causal relationship between teenage drinking patterns and adult outcomes. Teenage consumption of larger volumes of alcohol leads to continuation of this pattern in adult life with resulting poorer educational achievement, increased welfare dependence and substance use disorders. Teenage symptoms of Alcohol Use Disorder, however, lead to increased adult levels of psychopathology. These relationships are of interest to clinicians, policy-makers and researchers and could indicate targets for social policy and early intervention. Adult mental health disorders may be amenable to reduction through early intervention for youths with symptoms of alcohol use disorder. Educational outcomes and welfare dependence may usefully be targeted for improvement by reducing the volumes of alcohol consumed by adolescents – there is good evidence that this is achieved by reductions in alcohol availability. Reductions in the rate of alcohol and other substance dependence may be targeted through both these pathways. Specific early covariate factors have also been identified as having a strong and statistically significant correlation with early drinking patterns and consequent harm; this understanding may inform child welfare services and act as screening measures for clinicians.

Although solidifying what is known from an epidemiological perspective further research examining early alcohol use and later outcomes could add more enriched information. This is particularly true of the understanding of early dyads (child and parent). Understanding beyond 35 years would also be valuable, largely to examine if the understand further the stability and change in Use Disorder diagnosis. Lastly this study directly suggests specific public health interventions to be trialled, and these, would be best undertaken with specific research in mind.

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Online supplement:

Covariate factors

As described in the main paper, 21covariate factors were included in the analyses, on the basis that they were: a) used in a prior analysis of the age of first alcohol use in the CHDS cohort (1); or b) have been found to be related to substance use outcomes in other studies of the CHDS cohort (2-7).

These included:

Measures of family socio-economic and demographic background

- *Maternal age*. Assessed at the time of the survey child's birth.
- *Family living standards (0-10 years).* At each year a global assessment of the material living standards of the family was obtained by means of an interviewer rating.
- *Maternal and paternal education.* Parental education level was assessed at the time of the survey child's birth reflecting the highest level of educational achievement attained.
- *Family socioeconomic status (SES).* Family SES was assessed at the time of the survey child's birth using the Elley-Irving (8) scale of socio-economic status for New Zealand.
- *Averaged family income (0–10 years).* At each year, estimates of the family's gross annual income were obtained from parental report and were recoded into decile categories.
- *Maori ethnicity (at birth).* Maori ethnicity was assessed at the time of the cohort member's birth.

Individual, personality and behavioural factors

- *Gender*. Recorded at birth.
- *Child conduct problems (7–9 years).* When sample members were aged 7–9 years, information on child behaviour problems was obtained from parental and teacher report using a behaviour questionnaire combining items from the Rutter et al. (9) and Conners (10) parental questionnaires. (α = .97).
- *Neuroticism (age 14).* This was assessed using a short form version of the Neuroticism scale of the Eysenck Personality Inventory (11) at age 14. (α = .80).
- *Novelty-seeking (age 16).* Novelty-seeking was assessed at age 16 using the novelty seeking items from the Tridimensional Personality Questionnaire (12), (α =.76).

Family functioning, parental behaviour and abuse exposure measures

- *Parental illicit drug use (0–11 years).* At age 11, parents were questioned regarding their history of illicit drug use. The cohort member was classified as having a parent history of illicit drug use if one of his/her parents was reported to have a history of illicit drug use.
- *Parental alcohol problems (0–15 years).* This was assessed at age 15 years via parental report. These reports were used to form a dichotomous measure of whether or not the young person's parents reported experiencing problems with alcohol.
- *Parental criminality (0–15 years).* At age 15 years, parents were questioned as to whether any parent had a history of criminal offending. The cohort member was classified as having a parent history of criminality if one of his/her parents was reported to have a history of offending.
- *Parental alcohol consumption.* At age 11, parents were asked how many alcoholic drinks they would normally consume in a week and how many they had consumed in the past

week. These measures were combined to form a measure of the parents' typical weekly alcohol consumption.

- *Parental approval of adolescent drinking.* At age 15 years, cohort members were asked to describe their parent's views about adolescent alcohol consumption on a five-point scale ranging from strongly opposed to unconcerned.
- *Parental attitudes to alcohol use*. At age 15 years, cohort members were asked to rate their parents attitudes toward alcohol use in general on six three-point scales reflecting both their parent's use of alcohol and attitudes to alcohol use.
- *Changes of parents (to age 15 years*). At each assessment from birth to 15 years, information was gathered on changes in the cohort member's family situation since the previous assessment. Using this information an overall measure of family instability was constructed up to age 15.
- *Parental attachment (age 15).* This was assessed using the parental attachment scale developed by Armsden and Greenberg (13) and administered when sample members were aged 15. The full parental attachment scale was used in this analysis and was found to have good reliability ($\alpha = 0.87$).
- *Exposure to harsh/abusive physical punishment (childhood physical abuse; 0–16 years).* At ages 18 and 21 sample members were asked to describe the extent to which their parents used physical punishment during childhood (14). This information was used to create a four-level scale reflecting the most severe form of physical punishment reported for either parent.
- *Childhood sexual abuse (0-16 years).* At ages 18 and 21 years sample members were questioned about their experience of sexual abuse during childhood (<16 years) (15). Questioning spanned an array of abusive experiences from episodes involving non-contact abuse (e.g. indecent exposure) to episodes involving attempted or completed intercourse. A four-level scale was devised reflecting the most extreme form of sexual abuse reported by the young person at either age. For the purposes of the present analyses, those cohort members who reported having been exposed to penetrative sexual abuse were classified using a dichotomous measure.
- *Parental intimate partner violence (0–16 years).* At age 18, sample members were questioned concerning their experience of parental intimate partner violence during their childhood (prior to age 17 years). The questioning was based on a series of eight items derived from the Conflict Tactics Scale (16).

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